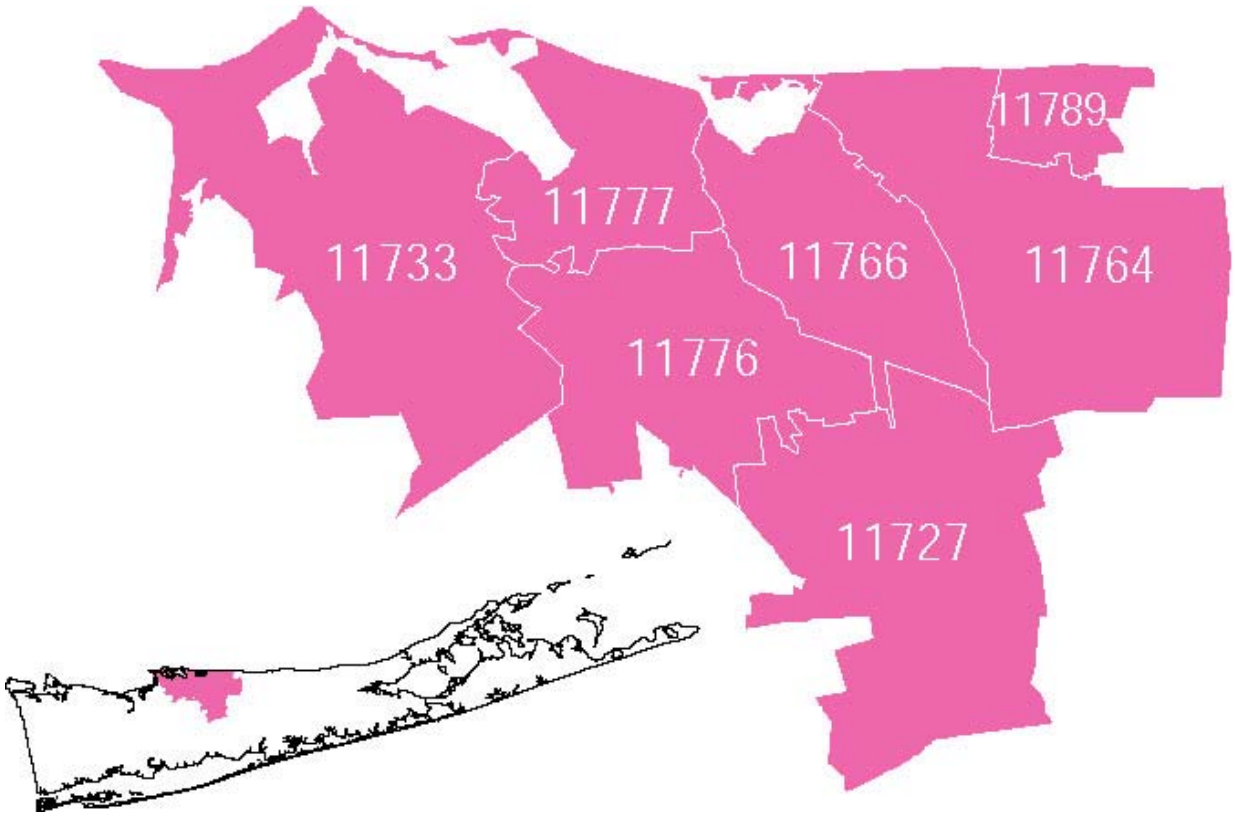
CORAM, MT. SINAI, PORT JEFFERSON STATION (CMP) FOLLOW-UP INVESTIGATION



Final Integration Report

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Department of Health
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I. Background Summary

A. Purpose of this Report

This report describes the evaluations conducted and the findings of the Coram/Mt. Sinai/Port Jefferson Station (CMP) Follow-up Investigation. The CMP investigation is designed to evaluate possible elevated environmental exposures and other unusual factors that could be related to the elevated breast cancer incidence between 1993 and 1997 observed in the CMP area, a seven ZIP-Code area in Suffolk County.

Three separate teams of researchers first worked independently then collectively to prepare this report. An epidemiologic team evaluated a variety of factors of the study population. A toxicologic team evaluated about 165 substances to characterize their likelihood of being risk factors for breast cancer in people. An environmental team evaluated a variety of environmental data on possible exposures that could have a potential relationship to the observed breast cancer incidence in the CMP area. All three teams then worked collectively to integrate their research and evaluate health risks associated with possible elevated environmental exposures in terms of their relationship to both breast cancer and other non-cancer health effects.

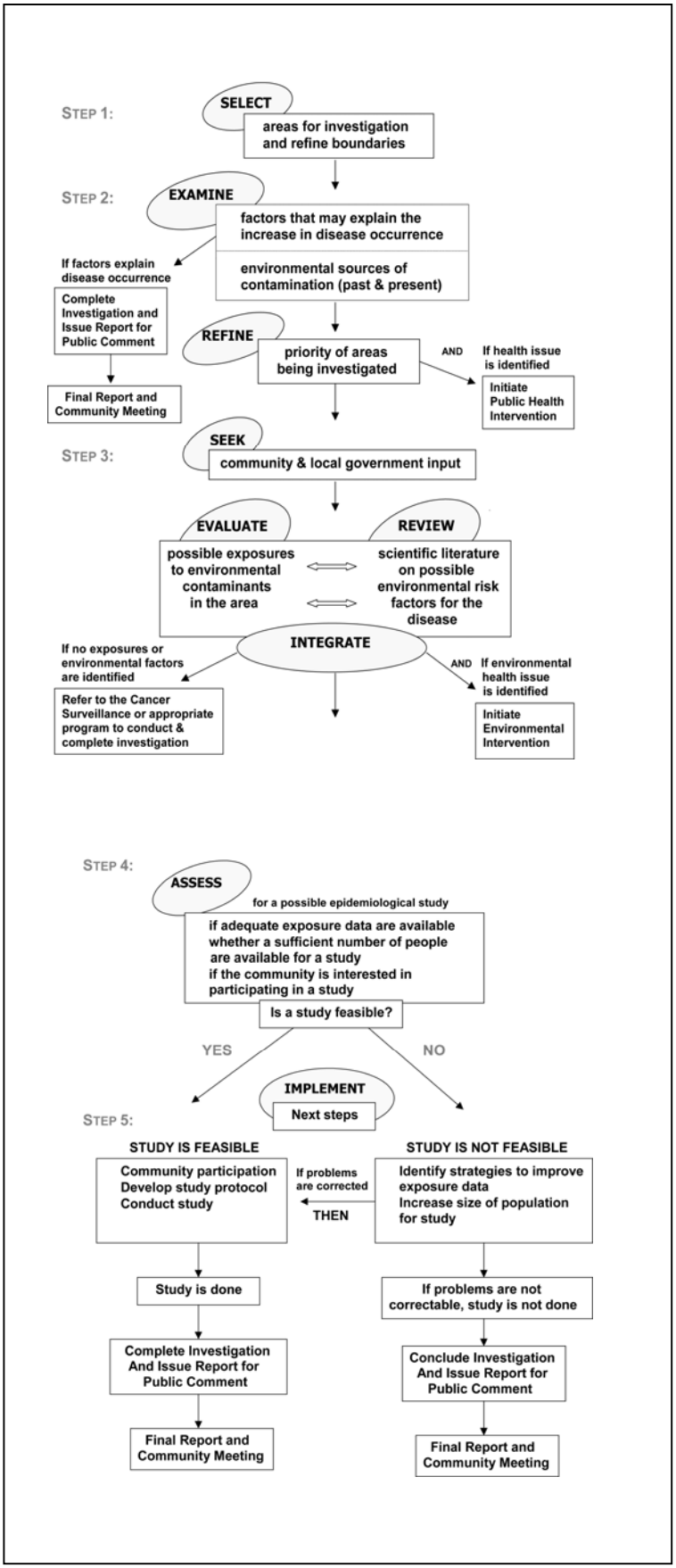
Health risk evaluations, which are described in the Integration Evaluation of this report, have been completed for compounds found to be elevated in the CMP area compared to other areas of the state. Several of these previously published in *the Working Draft Integration Report,* released in June 2004, are more detailed to illustrate the approach used to characterize health risks associated with possible environmental exposures in the CMP area.

As part of the Final Report, New York State Department of Health (NYS DOH) researchers have completed health risk characterizations and documented their findings. This report contains the complete set of conclusions about whether there were unusual factors in the CMP area that could be linked to elevated breast cancer incidence. It also makes recommendations based on these conclusions as called for as part of Step 3 of the Unusual Disease Pattern Protocol *(Figure 1)*. Finally, because the CMP Follow-up Investigation is the first cancer investigation that uses the Unusual Disease Pattern Protocol, this report recommends a re-evaluation of the use of the Protocol for future cancer investigations.

B. Background: the Coram/Mt. Sinai/Port Jefferson Station Investigation

The CMP Follow-up Investigation was conducted as part of the New York State Cancer Surveillance Improvement Initiative, also known as the Cancer Mapping Project. This investigation follows a Protocol called the Unusual Disease Pattern Protocol. This Protocol was developed to conduct investigations in New York State in ZIP Codes or other geographic areas where the incidence of a disease is significantly greater than expected. This Protocol was used for the first time in the CMP area to identify unusual environmental or other factors that may help to explain elevated breast cancer incidence diagnosed in this geographic area between 1993 and 1997.

Figure 1. Unusual Disease Pattern Protocol



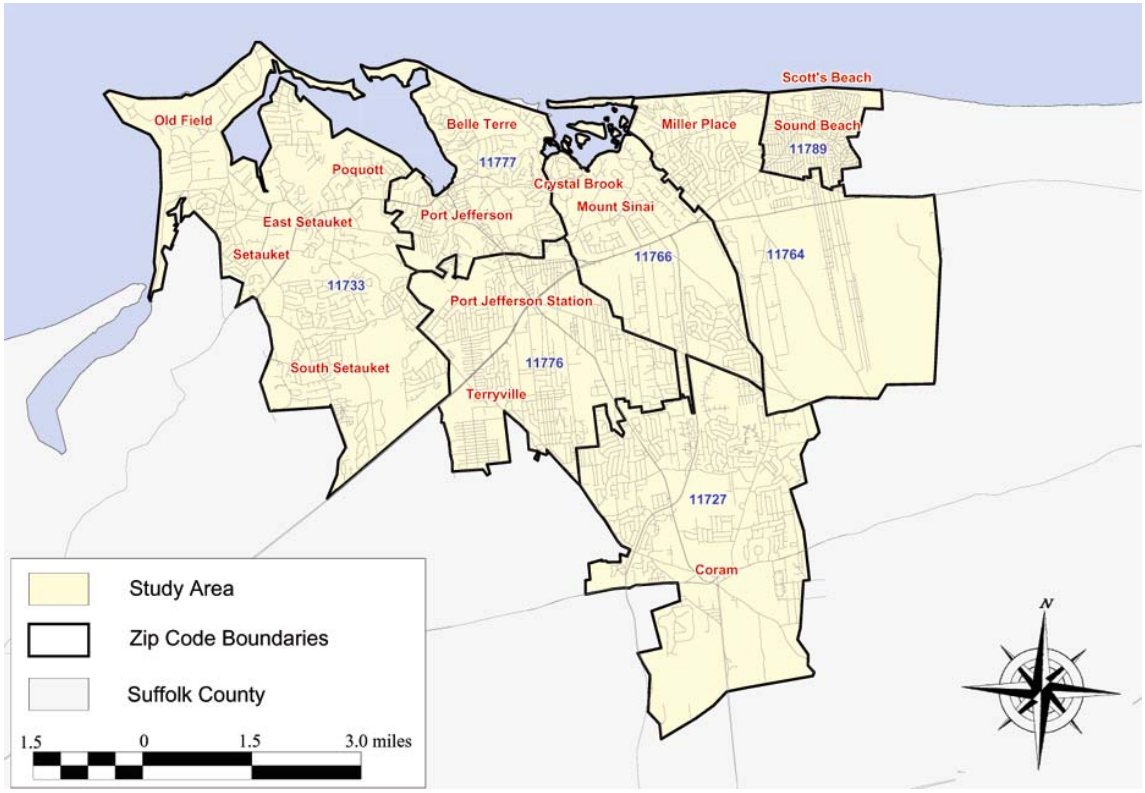
This Protocol was developed after areas of elevated cancer incidence were identified on ZIP Code level cancer maps released in 2000 and 2001. The maps reflected the newly diagnosed cases of breast, lung, colorectal and prostate cancer diagnosed over five-year periods (1993-1997 for breast, lung and colorectal cancer and 1994-1998 for prostate cancer). Using these maps, investigators identified areas of elevated cancer incidence and began prioritizing these areas for follow-up investigation.

Unusual Disease Pattern Investigations follow a five-step Protocol *(Figure 1).* Each investigation may or may not result in all five steps being completed, depending on the outcome of Integration (Step 3) of the Protocol. At the close of each investigation, a report of the findings will be issued for public comment. This is the final report for the CMP communities.

To identify unusual factors that may explain the higher incidence of breast cancer in the CMP area, NYS DOH researchers compared many environmental and demographic factors within the CMP area to New York State as a whole and to other geographic areas. NYS DOH staff also evaluated the likelihood that many environmental chemicals could be related to breast cancer incidence using the scientific literature. They then used this information to generate conclusions about the relationship between possible exposures to elevated levels of contaminants and other factors, and breast cancer incidence. NYS DOH staff also made recommendations about follow-up activities.

C. Project Location and Boundaries

Figure 2. Location of CMP Area



1. Selection of the Study Area

The CMP Follow-up Investigation is the first follow-up cancer investigation to be conducted as part of the Cancer Mapping Project. NYS DOH selected the CMP area to test the Unusual Disease Pattern Protocol because of the following:

- Of all the geographic areas identified on the ZIP Code-level breast cancer maps as having statistically elevated cancer incidence, the CMP area had one of the largest percentages of excess cases.
- Breast cancer maps were the first maps to be developed.
- The CMP area is relatively small and well-defined, making it amenable to study.

NYS DOH researchers first identified the CMP area on ZIP Code-level maps published in *Breast Cancer Incidence by ZIP Code 1993-1997* (NYS DOH, 2000). *Figure 2* shows the location of these communities in Suffolk County. *Table 1* lists these communities and associated breast cancer statistics.

Table 1. ZIP Codes in the CMP area and associated breast cancer incidence information (age adjusted, 1993-1997)

ZIP Code	Location	Number of Cancers Observed	Number of Cancers Expected	% Excess
11789	Sound Beach	30	16.8	79
11727	Coram	97	61.5	58
11766	Mt. Sinai	37	24.5	51
11776	Port Jefferson Station	85	59.8	42
11777	Port Jefferson	43	31.7	36
11733	East Setauket	64	51.6	24
11764	Miller Place	34	33.6	1
Total area		390	279.5	40

Source: NYS Cancer Registry

The ZIP Code-level maps compared the *standardized incidence ratios (SIRs)* for breast cancer (the ratio of the actual number of newly diagnosed breast cancer cases and the expected number of breast cancer cases, considering age and population size in each ZIP Code). The maps also displayed the results of a statistical technique called SaTScan, used by researchers to evaluate elevations of cancer incidence that would not be expected to appear just by chance in an individual ZIP Code or in groupings of ZIP Codes.

Using SaTScan, NYS DOH researchers identified parts of Coram (11727), Mt. Sinai (11766) and Port Jefferson Station (11776) as the highest areas of elevated cancer incidence (p-value=0.003). Because Unusual Disease Pattern Investigations involve the evaluation of environmental factors and explore possible relationships to disease incidence, researchers reviewed the NYS DOH Suffolk County breast cancer maps and the SIRs in neighboring ZIP Codes to create a contiguous area between Coram, Mt. Sinai, Port Jefferson Station and Sound Beach (11789), which also had high breast cancer incidence. In doing so, researchers included East Setauket (11733), Miller Place (11764) and Port Jefferson (11777).

2. Study Area Environmental and Demographic Setting

The CMP area is in the northern part of central Suffolk County, along the north shore of Long Island, approximately 50 miles east of New York City. The area is approximately 52 square miles. The CMP area has experienced significant growth during the past 20 years. According to data from the 1980 U.S. Census (census blocks conforming to 1999 ZIP Code definitions), the population in the CMP area was 85,823. According to the 2000 U.S. Census the population in this area increased by almost 23% to about 105,320. The population of Suffolk County as a whole grew about 10% during the same time period.

Land use in the CMP area also has changed over time, with a steady increase in residential properties and associated service businesses on formerly rural and agricultural lands. According to 1997 U.S. Census Bureau data, 2,520 businesses were located in the CMP area compared to 38,564 businesses in the rest of Suffolk County. Forty-two percent (42%) of the businesses in the CMP area provide services such as medical offices, legal

services, and beauty shops. In Suffolk County, 35% of the businesses provide services. Less than 3% of the businesses in the CMP area are involved in manufacturing, compared with more than 6% in the rest of Suffolk County.

Today, land use in the CMP area is characteristic of western Suffolk County, with the majority of land use taken up by housing and light commercial properties. Major features in the area include an oil/natural gas power plant, small quarries and a wastewater treatment plant. Three major hospitals are in the immediate area, including the medical center at the State University of New York at Stony Brook, which is immediately west of the area. Port Jefferson is a terminus for the Long Island railroad and the ferry between Bridgeport, Connecticut and Port Jefferson, New York.

The Coram, Port Jefferson Station and East Setauket ZIP Codes are the largest population centers within the CMP area, with between 20,000 and 25,000 people residing in each ZIP Code in 2000. Miller Place, Mt. Sinai, Port Jefferson and Sound Beach ZIP Codes have fewer people living there, with between 7,500 and 11,000 people in each ZIP Code in 2000.

Median household income for the entire CMP area in 1999 was approximately $72,000. Median income ranged from $56,000 in Sound Beach to $98,000 in East Setauket. About 45% of the area's employees work in management and professional positions, and approximately 30% of area residents are employed in educational, health and social services industries. Select demographic characteristics of the CMP area are discussed in greater detail in *Chapter II. Epidemiological Evaluation,* and raw demographic information may be found in *Appendix II-1* and *Appendix II-2* of this report.

D. Project Description and Methods Summary

This report describes three distinct evaluations conducted by NYS DOH staff during the CMP Follow-up Investigation and integrates this information to make conclusions about breast cancer incidence in the CMP area.

Epidemiological Evaluation. The epidemiological team conducted an evaluation to verify the excess incidence of breast cancer in the CMP area. This effort involved critically examining factors that possibly could account for the higher than expected incidence, or number of new cases of cancer diagnosed during the years 1993 through 1997. Researchers evaluated whether a particular age group, race or ethnic group was driving the excess in cancer incidence in the area. Researchers reviewed epidemiological studies in the scientific literature. They then conducted an analysis of the CMP area adjusting for demographic and socioeconomic characteristics that are known risk factors for breast cancer. Researchers also conducted an analysis of the CMP area over time to evaluate the persistence of elevated breast cancer incidence over time. Finally, they conducted a length of residence evaluation to collect information about how long women with breast cancer lived in this area.

Toxicological Evaluation. The toxicological research team reviewed the scientific literature to identify compounds that have been linked to breast cancer based on evidence from human, laboratory animal and other studies. This

review was used to develop a classification system and an associated list of possible and unlikely environmental risk factors for breast cancer.

Environmental Exposure Evaluation. The environmental exposure research team used a variety of existing environmental data sets and information collected from the community and other environmental and health agencies to evaluate a history of possible environmental exposures in the CMP area. Researchers first screened environmental exposure data by evaluating the completeness of environmental information to draw conclusions about possible environmental exposures. In cases where researchers determined there was enough information, they then compared this information for the CMP area with selected areas in New York State. When comparison area data were insufficient or unavailable, other data were used to evaluate environmental exposures in the CMP area.

Integration Evaluation. The three research teams regrouped to review the results of the epidemiological, toxicological and environmental evaluations, and to assess the risk associated with environmental exposures in terms of their relationship to both breast cancer and other non-cancer health effects. This evaluation resulted in a set of conclusions about whether there were unusual factors in this area that could be linked to elevated breast cancer incidence and recommendations for the CMP area.

E. Limitations of Analysis

Conclusions in this report are based on an overall weight of evidence assessment of the epidemiological, environmental and toxicological evaluations. As a result, they are limited by the amount and quality of information about the individual breast cancer cases, historical environmental exposure information, and information from the scientific literature that demonstrates a particular compound as a risk factor for breast cancer.

The long latency period for breast cancer also poses major barriers for developing conclusions about linkages between particular risk factors and breast cancer incidence. The latency period is the period of time between the first exposure to a factor that may have contributed to a disease and the appearance of any symptoms of that disease. Scientists estimate a latency period of between 5 and 40 years for breast cancer to occur after being exposed to a risk factor. This means that for the CMP study population (diagnosed between 1993 and 1997), researchers are interested in evaluating exposures that could have occurred for some period of time between 1953 and 1992. Unfortunately, the information to evaluate possible environmental exposures in that time frame is limited. In addition, researchers also have limited information about how long each woman diagnosed with breast cancer lived at her residence or in the CMP area prior to her diagnosis. Women are also exposed to many risk factors for breast cancer throughout their lives, and some risk factors are more important than others, depending on the age of the woman at the time of her exposure.

In spite of these limitations, researchers have used these evaluations to hypothesize about the relationship of possible environmental exposures and other factors to breast cancer incidence in these communities. As such, this

evaluation is designed to inventory factors in the CMP area that are elevated or unusual based on a consistent, critical examination of existing environmental and demographic data sets.

II. Epidemiological Evaluation

A. Initial Epidemiologic Evaluation—Confirmation of Breast Cancer Excess

As part of an earlier step in the Unusual Disease Pattern Protocol, NYS DOH staff critically examined factors that could possibly account for the higher than expected number of new breast cancer cases for the years 1993 through 1997 in the CMP area. These factors included the following:

- **Breast cancer screening.** Researchers examined the stage distribution of breast cancer cases to assess the possibility that screening in the CMP area was more widely used and led to higher disease detection in this area than in other areas of the state.
- **Population estimation.** Researchers verified population estimates for New York State and the CMP area to make certain that these were not over- or underestimated in such a way that would cause the breast cancer incidence in the CMP area to stand out as higher than the rest of the state.
- **Seasonal residents**. Researchers evaluated whether higher seasonal residence in the CMP area than in other areas of the state could have caused women who were not year-round residents to be counted as being residents of these communities when they were diagnosed with breast cancer.
- **Standards of medical care.** Researchers reviewed disease and reporting characteristics that are influenced by medical care practices to evaluate the possibility of erroneous reporting that would have lead to higher numbers of cases identified in the CMP area.

  Researchers confirmed that the breast cancer excess was not likely due to any of the above factors. In the present step of the Unusual Disease Pattern Protocol, the research team examined an additional factor that could have some bearing on the nature of the breast cancer excess:

- **Persistence over time.** Researchers examined breast cancer data in the CMP area for prior and more recent years. If the excess was only observed between 1993 and 1997 (the years of the original mapping analysis), it might indicate an unlikely occurrence that was still a product of chance, or a time-limited effect. If the excess pre-dated 1993 or persisted after 1997, it would provide evidence of a continuing effect that would be less likely to be due to chance.

1. Screening/Stage Distribution

To evaluate whether part of the increased breast cancer incidence in the CMP area could be attributed to screening, researchers examined the stage distribution of breast cancer cases in the CMP area. Cancers diagnosed *in situ*, or before invading adjacent tissues, were included in this analysis because increased levels of breast screening in an area would be expected to lead to greater detection of both *in situ* and early stage cancers. *In situ* breast cancers were not included in the mapping analysis. Since it is known that breast cancers in women

under age 50 are more likely to be diagnosed either *in situ* or at a late stage than breast cancers in women over age 50, state health researchers examined the stage distribution separately for older and younger women.

Table 2 shows the numbers and percents of breast cancers diagnosed at different stages in the CMP area as well as in the comparison areas of New York State, New York State exclusive of New York City, and Suffolk County. New York State was selected as a comparison area since the expected numbers of cases in the mapping analysis were calculated based on breast cancer incidence rates for New York State. New York State exclusive of New York City was selected as a comparison area since the demographic features of the CMP area are more similar to this large geographic area than to the state including New York City. Suffolk County was chosen as a comparison area to account for any factors influencing breast cancer diagnosis that may be present on a regional scale, such as medical care practices. The use of three comparison areas helps to provide a broader perspective on the source of any differences found.

Table 2. Breast cancer cases by age group and stage of disease at time of diagnosis, CMP and selected comparison areas, 1993-1997

Summary stage	New York State		NYS, excl. NYC		Suffolk County		CMP	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Age under 50								
In situ	2447	15.2	1564	15.9	213	15.4	22	14.9
Early	7445	45.7	4665	47.4	655	47.4	69	46.6
Late	5217	32.0	3007	30.5	455	33.0	55	37.2
Unknown	1146	7.0	613	6.2	58	4.2	2	1.4
p-value*		<0.05		<0.05		>0.05		
Age 50 and over								
In situ	5918	11.1	3807	11.5	449	11.0	40	13.1
Early	27523	51.6	17586	53.0	2083	51.2	147	48.0
Late	14368	27.0	8467	25.5	1142	28.1	91	29.7
Unknown	5485	10.3	3330	10.0	395	9.7	28	9.2
p-value*		>0.05		>0.05		>0.05		

Source: NYS Cancer Registry
**overall stage distribution compared with CMP*

The stage distribution for women under age 50 in the CMP area was significantly different ($p < 0.05$) from the stage distributions for the New York State and New York State exclusive of New York City comparison areas, but not significantly different from the overall stage distribution for breast cancers diagnosed in Suffolk County women. The proportions of cancers diagnosed at a late or unknown stage accounted for most of the differences in stage distributions. When the statistical comparisons were restricted to cancers with known stage (not shown), the proportions of *in situ*, early and late stage cancers in the CMP area were not significantly different from those in

New York State or New York State exclusive of New York City. For women age 50 and older, the overall stage distributions in the CMP area and the three comparison areas were not significantly different.

If the increased incidence in the CMP area were due to a screening effect, one would expect the proportion of cancers diagnosed at an *in situ* or early stage to be higher in the CMP area than in the comparison areas. As indicated in *Table 2,* that is clearly not the case for either younger or older women. Therefore, these data do not provide any evidence that the excess in breast cancer incidence in the CMP area is an effect of increased screening or disease detection.

2. Population Estimation

Inaccuracies in population estimation have implications for the analysis of cancer incidence. The cancer incidence rate depends on a) the number of cancers diagnosed in a certain population, and b) the size of that population. Since population counts were not available from the 2000 US Census at the time of the mapping analysis, the analysis used estimates of post-1990 Census populations. Any inaccuracies in the estimated statewide, age-specific population proportion could result in inaccuracies in the calculation of the expected numbers of cases in each ZIP Code. Any inaccuracies in the local population estimates could have caused further inaccuracies in calculating the expected numbers of cases for individual ZIP Codes because the statewide rates were applied to the population estimates for each ZIP Code to obtain the expected number of cases for that ZIP Code. If the expected numbers of cases were over- or underestimated, the resulting standardized incidence ratios would have under- or overestimated cancer excesses. Therefore, it was important that researchers reexamine the accuracy of statewide and CMP area population estimates to assure accuracy in the determination of the excess breast cancer incidence in the CMP area.

To conduct this analysis, population estimates for 2000 provided by Claritas, the commercial vendor that provided the post-1990 census ZIP-Code population estimates used to calculate expected numbers of cases, were compared with counts by sex and age group from the 2000 Census for New York State, New York State exclusive of New York City, and Suffolk County. For all ages combined, Claritas underestimated the statewide population by about 4%. The underestimate was greatest in New York City (about 8%), and only about 2% outside of New York City. The net underestimate in Suffolk County was similar to that in New York State outside of New York City.

If individual age groups are examined separately, the net underestimates statewide, as well as in New York State exclusive of New York City and Suffolk County, are accounted for largely by underestimates of persons in younger age groups (males under age 45 and females under age 40). For females ages 50 and over, who account for about 80% of breast cancer cases in New York, Claritas population estimates were about 1% higher than the Census counts statewide. Estimates for New York State outside of New York City and Suffolk County were within 0.1% of the Census counts.

To account for the effects of inaccuracies in population estimates, expected numbers of cases in each of the seven ZIP Codes individually and in the seven ZIP Codes combined were recalculated based on linear interpolation between populations from the 2000 U.S. Census and populations from the 1990 U.S. Census for the same

geographic areas. The results are shown in *Table 3.* (Minor discrepancies in numbers from *Table 2* are due to updates to the Cancer Registry database). The use of more accurate population figures resulted in some changes in the standardized incidence ratios for the individual ZIP Codes. However, in the seven ZIP Codes combined, the effect was negligible.

Table 3. Breast cancer excesses before and after recalculation using 2000 U.S. Census population data, ZIP Codes included in the CMP area, 1993-1997

ZIP Code	Location	Number of cancers observed	Post-1990 Census population estimates		1990 and 2000 Census population estimates	
			Number of cancers expected	Percent Excess	Number of cancers expected	Percent Excess
11789	Sound Beach	31	17.6	76	18.9	64
11727	Coram	102	64.8	57	69.8	46
11766	Mt. Sinai	36	25.8	40	24.0	50
11776	Port Jefferson Station	87	62.4	39	64.8	34
11777	Port Jefferson	43	33.4	29	33.9	27
11733	East Setauket	65	54.3	20	51.5	26
11764	Miller Place	34	35.4	-4	28.7	18
Total area		398	293.7	36	290.8	37

Sources: NYS Cancer Registry, post-1990 U.S. Census population estimates provided by Claritas, 1990 and 2000 population estimates derived from U.S. Census data

3. Seasonal Residents

The CMP area was not identified as an area with a large number of seasonal residents.

4. Other Factors

The percent of breast cancers that were microscopically confirmed in the CMP area and the three comparison areas is illustrated in *Table 4.*

Table 4. Microscopic confirmation of invasive breast cancer cases diagnosed 1993-1997, CMP and selected comparison areas

Confirmation	New York State		NYS, excluding NYC		Suffolk County		CMP area	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Confirmed	57171	93.4	35670	94.7	4570	95.5	376	95.9
Not confirmed	4013	6.6	1998	5.3	218	4.6	16	4.1

Source: NYS Cancer Registry

In all areas, the vast majority of breast cancer cases were confirmed by microscopic examination of tumor tissue, indicating a very low potential for the excess to be related to uncertainties in disease diagnosis. The percent with microscopic confirmation is slightly greater in the CMP area than in the state as a whole, but consistent with that generally found in Suffolk County. This may indicate a slightly higher standard of medical care in the area.

Table 5 displays the distribution of tumors in the CMP area and the comparison areas by the number of sources (e.g. hospitals, laboratories) reporting each tumor.

Table 5. Number of information sources reporting each tumor, invasive breast cancers diagnosed 1993-1997, CMP and selected comparison areas

Number of information sources reporting	New York State		NYS, excl. NYC		Suffolk County		CMP	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
1	31335	51.1	19899	52.7	2221	46.4	145	37.0
2-4	28356	46.3	17080	45.3	2431	50.7	228	58.2
5-7	1528	2.5	722	1.9	136	2.8	17	4.3
8 or more	87	0.1	38	0.1	4	0.1	2	0.5
p-value*	<0.001		<0.001		<0.001			

Data Source: NYS Cancer Registry
**overall source number distribution compared with CMP*

Table 5 shows that breast cancers in the CMP area were likely to be reported by a larger number of sources than cancers in residents of Suffolk County as a whole, New York State exclusive of New York City, and New York State as a whole. Differences between the CMP area and all the comparison areas in the overall distributions of numbers of sources reporting each tumor were statistically significant ($p < 0.001$). This finding is consistent with a greater level of interaction with the health care system in this area.

5. Stability of the Results Over Time

The temporal characteristics of the disease excess may be important when looking for possible associations with environmental and other factors. Observed and expected numbers of cases in each of the seven CMP ZIP Code areas were calculated for each year from 1990 through 2000 based on populations interpolated between the 1990 and 2000 censuses and the most recent data from the Cancer Registry. (Slight discrepancies are due to updates to the Cancer Registry database.) For the three-year period 1990-1992, before the years included in the mapping analysis, observed numbers of cases in the seven ZIP Codes combined were 12% higher than the numbers expected, compared with 37% higher in the five years included in the mapping analysis. In the three-year period 1998-2000, following the years included in the analysis, observed numbers of cases, however, remained 35% higher than expected in the seven ZIP Codes combined.

Similar results were found for the three core ZIP Codes (11727, 11766, and 11776) of the CMP area. These three ZIP Codes combined showed a 3% excess in breast cancer cases in the period 1990-1992, a 42% excess in the years of the mapping analysis, and a 40% excess in the three years of data since. These results show that although the excess in breast cancer incidence in the CMP area did not appear in any great strength until the time of the analysis, the excess has persisted over time.

6. Conclusion

The stage distribution of breast cancers diagnosed among women in the CMP area is not consistent with an excess that would be attributable to an unusually high level of breast cancer screening in the area. There were discrepancies between the population estimates provided by Claritas that were used to calculate expected numbers of cases and the 2000 U.S. Census figures in some individual ZIP Codes. However, these included overestimates and underestimates, and the net effect of these in the seven ZIP Code areas combined was minor. There was not an appreciable number of seasonal residents in the area that may have caused the number of persons at risk to be underestimated. Data suggest a slightly higher standard of medical care and/or level of access to medical care in the area (once symptoms have appeared), but do not indicate any unusual features of cancer diagnosis or reporting that could have caused a falsely elevated number of reported cases. Although a marked elevation in breast cancer incidence in the CMP area, and especially in the core ZIP Codes of Coram, Mount Sinai and Port Jefferson Station, did not appear until the time period covered in the original mapping analysis, this elevation has persisted in the years since, making it less likely to be due to statistical fluctuation coinciding with the analysis period. The evaluation thus further supports the likelihood that the elevation in breast cancer incidence in the CMP area is not due to chance or to factors related to the analysis.

B. Examination of Disease Excess

Researchers examined available data on the women diagnosed with cancer and on the cancers themselves to identify any unusual features of the disease excess that would help to focus the investigation. As part of an earlier step in the Unusual Disease Pattern Protocol, researchers evaluated whether any age group of women was particularly affected by the excess, and whether the excess involved any unusual cell types of breast cancer. Additional evaluations considered the racial and ethnic characteristics of the women with breast cancer.

1. Age Groups Affected

Researchers compared the observed and expected numbers of cases for three age groups of women separately to determine whether any age group was particularly affected by the excess. These results are presented in *Table 6.* (Slight discrepancies are due to updates to the Cancer Registry database.) For the seven ZIP Codes combined, numbers of breast cancer cases observed were about 40% higher than the numbers expected in women under the age of 50, women age 50 to 64, and women age 65 and older.

Table 6. Breast cancer excesses by age group, ZIP Codes included in the CMP area

ZIP Code	Location	Number of cancers observed	Percent excess by age group			
			Overall	under 50	50-64	65 +
11789	Sound Beach	31	79	80	159	48
11727	Coram	99	58	40	73	98
11766	Mt. Sinai	36	51	50	14	52
11776	Port Jefferson Station	86	42	64	38	32
11777	Port Jefferson	44	36	46	51	18
11733	East Setauket	64	24	6	38	13
11764	Miller Place	33	1	-13	-25	18
Total area		393	40	41	44	40

Source: NYS Cancer Registry

Within individual ZIP Codes, the size of the excess (or deficit) varied across the different age groups, with no consistent trend. Some of this variation may be related to the variability encountered when dealing with small numbers, but even for the ZIP Codes with the largest numbers of cases (Coram and Port Jefferson Station), the excess in cases was greatest in different age groups. In ZIP Code 11727 (Coram), the excess was greatest in age groups 50-64 and 65 and over, while in ZIP Code 11776 (Port Jefferson Station), it was greatest in the under-50 age group. Results thus show that the overall excess in the seven ZIP Code areas is present in women of all ages, with no age group consistently standing out.

2. Race/Ethnicity

The racial and ethnic distributions of the women in the CMP area diagnosed with breast cancer are presented in *Table 7*. In the table, races other than white are grouped due to the small number of women in the CMP area who were identified as being of other races. Women were identified as being of Hispanic origin if the ethnicity reported to the Cancer Registry was either Spanish, Puerto Rican, Cuban, Mexican American, Central or South American or another Hispanic ethnic group, or if their place of birth was a country with a mainly Hispanic population, or if their surname matched a list of Spanish surnames.

This table shows that the proportion of women with breast cancer who were identified as white was significantly higher in the CMP area than in any of the comparison areas (p-value < 0.01). The proportion of women with breast cancer in the CMP area who were identified as being of Hispanic origin was not significantly different ($p > 0.05$) than the proportion of women with breast cancer in all of New York State or Suffolk County identified as being of Hispanic origin. The proportion of women with breast cancer in the CMP area who were of Hispanic origin was greater, however, than the proportion of women with breast cancer in New York State exclusive of New York City who were of Hispanic origin ($p < 0.001$). The racial and ethnic distribution of the breast cancer cases reflects that of the CMP population (see Appendix II-1).

Table 7. Racial identification and Hispanic ethnicity of women diagnosed with invasive breast cancer 1993-1997, CMP and selected comparison areas

	New York State		NYS, excl. NYC		Suffolk County		CMP	
	Number	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Racial identification								
White	53489	86.2	36063	94.5	4575	94.7	391	98.2
Other than white	8573	13.8	2115	5.5	257	5.3	7	1.8
p-value*		<0.0001		<0.005		<0.005		
Hispanic ethnicity								
Hispanic	3671	5.9	661	1.7	149	3.1	16	4.0
Non-Hispanic	58391	94.1	37517	98.3	4683	96.9	382	96.0
p-value*		>0.05		<0.001		>0.05		

Source: NYS Cancer Registry
**compared with CMP*

3. Histologic Types

Table 8 shows the numbers and percent of total breast cancers accounted for by cancers of specified cell types that make up 1% or more of total breast cancers statewide, and by nonspecific cell types (coded as “malignant neoplasm” or “carcinoma, not otherwise specified”). Numbers and percents are given for New York State as a whole, New York State exclusive of New York City, Suffolk County, and the seven ZIP Codes of the CMP area.

Table 8. Distribution by cell type (morphology), invasive breast cancers diagnosed 1993-1997, CMP area and selected comparison areas

	New York State		NYS, excl. NYC		Suffolk County		CMP	
Morphology	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Adenocarcinoma, NOS	1791	2.9	1064	2.8	120	2.5	14	3.6
Tubular adenocarcinoma	645	1.1	467	1.2	60	1.3	4	1.0
Mucinous adenocarcinoma	1145	1.9	731	1.9	71	1.5	2	0.5
Infiltrating ductal carcinoma	40106	65.6	25012	66.4	3273	68.4	264	67.4
Comedocarcinoma	977	1.6	664	1.8	70	1.5	4	1.0
Medullary carcinoma	756	1.2	448	1.2	44	0.9	7	1.8
Lobular carcinoma	5440	8.9	3485	9.3	430	9.0	38	9.7
Infiltrating ductal and lobular carcinoma	2625	4.3	1499	4.0	235	4.9	20	5.1
Other specified	2180	3.6	1224	3.3	146	3.1	15	3.8
Unspecified	5519	9.0	3074	8.2	339	7.1	24	6.1

Source: NYS Cancer Registry

In the CMP area, as well as all other comparison areas, the majority of breast cancers diagnosed are infiltrating ductal carcinomas. These make up 66% of total invasive breast cancers in the state as a whole, and 67% of breast cancers in the CMP area. The second most frequently occurring type of breast cancer is lobular carcinoma, accounting for 9% of cases statewide and 10% of cases in the CMP area. The third most frequently occurring type in all areas is mixed infiltrating ductal and lobular carcinomas. Most of the other specific cell types occur at slightly higher frequencies in the CMP area compared to the comparison areas, while the CMP area shows a slightly lower proportion of cases with nonspecific cell type.

The higher proportion of tumors with a specified cell type and lower proportion with a nonspecific cell type is probably another indication of a higher standard of medical care in the CMP area, as more tumors undergo pathologic examination. Since a greater proportion of tumors are given a specific histologic diagnosis, it would be expected that the proportions of all specified cell types would be slightly higher in the CMP area than in any of the comparison areas.

4. Summary

In the CMP area, the excess in breast cancer incidence is present in women of all ages, with no particular age group consistently standing out. The vast majority of women in this area diagnosed with breast cancer were white,

and only a few were of Hispanic ethnicity. There are no particular breast cancer cell types appearing in unusual numbers.

C. Breast Cancer Risk Factors

Any investigation into possible factors related to breast cancer incidence in an area needs to consider what is already known about risk factors for breast cancer. The epidemiologic literature on breast cancer is extensive. In the mid-1990s, the Collaborative Group on Hormonal Factors in Breast Cancer identified 66 epidemiologic studies from around the world that included at least 100 women with breast cancer (Collaborative Group, 1996*)*. Several more have been completed since. This chapter is therefore not intended to be comprehensive. Current understandings of established risk factors are summarized in some recent scientific and popular reviews (Hulka 2001, Kelsey 1996, Hankinson 2002, ACS 2003, Harvard 2000).

A number of risk factors for breast cancer have been identified. The most important of these are sex and age. Breast cancer risk in females is about 100 times greater than risk in males. Risk increases sharply with age until approximately age 50, and then more gradually, leveling off at about age 70. To control for these risk factors, the mapping analysis was restricted to breast cancer in females, and expected numbers of cases in each ZIP Code were calculated taking into account the age distribution of females in that ZIP Code.

Breast cancer is known to occur more frequently in white women than in African American, Asian/Pacific Islander or Native American women. Indications are that it also is less common among women of Hispanic origin than among non-Hispanic whites. Breast cancer incidence has been found to be highest in the countries of North America and Northern Europe, and lowest in the developing countries of Asia, Africa and South America.

Other risk factors relating to genetic, reproductive, nutritional and other factors are well established. Women with a family history of breast cancer are known to be at an increased risk of the disease, particularly if the cancers occurred in first-degree relatives affected at a young age. A number of mutations have now been identified at two breast cancer genes that confer an extremely high risk of breast cancer on female carriers, although most familial cases have not been associated with identified mutations. It has been estimated that a family history of the disease in first degree relatives account for 5-10% of breast cancer cases.

Women who have had a prior breast cancer are known to be at a greater risk of developing a second cancer in the other breast, or in the remaining breast tissue. Due to the counting rules in use at the time by the Cancer Registry however, second (or later) primary breast cancers were not included in the mapping analysis. (Breast cancers that were diagnosed in persons with a history of another type of cancer, for example colon cancer, were included.) Risk is also known to be greater in women with certain types of benign breast disease and in post-menopausal women with a mammographic finding of dense breasts (Byrne 1995).

The importance of reproductive factors in affecting breast cancer risk has been known for a long time. Women who have never given birth (or had a full-term pregnancy) are at a higher risk for breast cancer compared to women who have carried a pregnancy to term.

Among women who have given birth, the age of a woman at her first delivery is an important factor influencing breast cancer risk. Women who are under 20 years old when they have their first full-term pregnancy have the most reduced risk of breast cancer. Women who are between the ages of 20 and 29 when they have their first full-term pregnancy have a slightly greater risk than women under 20 years old who carry full-term. Women who are older than 30 when they have their first full-term pregnancy have a risk about equal to, or slightly greater than, women who had never given birth.

Women with more children also have a lower risk of breast cancer compared to women with fewer children. Researchers have considered that women who have their first full-term pregnancy at a young age are more likely to end up having more children than women who start childbearing late in life. Even among women who were the same age at first full-term pregnancy, however, those with more total births have a lower risk than those with fewer births. Breastfeeding is another reproductive factor that has received attention in relation to its effects on breast cancer risk. A recent article from the Collaborative Group on Hormonal Factors in Breast Cancer (2002a) shows that total duration of breastfeeding has an independent effect on reducing the risk of breast cancer over and above that of the reproductive factors discussed previously. Differences in the duration of breastfeeding may account for much of the observed difference in breast cancer rates between developing and developed nations.

Other reproductive factors that have been shown to increase the risk of breast cancer include an early age at menarche and a late age at menopause. Both of these factors imply a longer duration of exposure to endogenous estrogens. Some recent studies have also shown a higher risk of breast cancer among women with higher levels of circulating estrogens (Thomas 1997).

These reproductive factors are all associated with variations in the levels, types and timing of endogenous estrogen a woman is exposed to. It might be expected that exogenous estrogens such as those found in various hormone preparations may also play a role. In many studies, oral contraceptives (birth control pills) have been found to increase risk of breast cancer. The greatest increased risk has been observed in current users, and former users within five years of discontinuing use; little increased risk is observed ten or more years after discontinuing use. Hormone replacement therapy is used to counteract the effects of the cessation of estrogen production during menopause. Replacement therapy has been found to increase breast cancer risk to the same extent as not going through menopause.

Dietary factors are believed to play a role in breast cancer. Obesity is an established risk factor for breast cancer in post-menopausal women. International comparisons show higher rates of breast cancer in countries where dietary fat consumption is high, but dietary intervention studies have not been able to reduce breast cancer risk in women by restricting fat consumption. Several recent studies have suggested that physical activity may decrease the risk of breast cancer, possibly by leading to anovulatory cycles and hence, lower total estrogen exposure in some women. Some studies have shown the risk of breast cancer to be greater in taller women, which might be related to

high caloric intake or to a faster growth rate during childhood and adolescence (Tretli 1989, Kelsey 1996, Harvard 2000).

It has often been observed that breast cancer rates are higher in more affluent areas (see, for example, Rimpela 1987). This is usually attributed to childbearing patterns, as more affluent women are more likely to attend college and thereby delay childbearing. Certain occupations have been observed to be associated with characteristically high breast cancer rates, including teaching and health care occupations (see, for example, NYS DOH 1986, Bernstein, et al., 2002). These associations as well might be attributable, at least in part, to childbearing patterns.

Breast cancer studies in the past 20 years have produced fairly consistent results showing elevated breast cancer risk associated with heavy alcoholic beverage consumption, defined in most studies as more than three drinks per day. An important study published in 2002 (Collaborative Group 2002b) reanalyzed individual data from 53 epidemiological studies, which included 58,515 women with breast cancer and 95,067 without the disease. This meta-analysis concluded that breast cancer risk was elevated by approximately 30% for individuals consuming between three and four drinks per day and by approximately 40% for those consuming more than four drinks per day. If the observed relationship is causal, the authors estimate that about 4% of breast cancers in developed countries are attributable to alcohol.

Studies of cigarette smoking and breast cancer conducted in the 1960s and 1970s usually compared women who have ever smoked to women who have never smoked. They showed no consistent association between smoking and breast cancer risk (Collaborative Group, 2000b). However, these studies did not take into account the possible effects of exposure to secondhand tobacco smoke or *"passive smoking."* Because study questionnaires did not ask about passive smoking, women with this type of exposure were grouped together with non-smokers in these analyses. In some more recent studies, passive smoking was taken into account. These more recent studies show associations with both passive and active smoking and breast cancer risk. In a Canadian study of 2,317 breast cancer cases and 2,438 controls for whom full risk factor histories, including lifetime residential and occupational histories of exposure to passive smoking, were gathered by questionnaire, active and passive smoking were each associated with more than a doubling of premenopausal risk of breast cancer. Postmenopausal risk estimates were not as high, but were also elevated (Johnson, 2000). Five other studies have assessed passive as well as active smoking, and these studies consistently show an approximate doubling of breast cancer risk associated with passive and with active smoking (Lash, 1999; Morabia, 1996; Smith, 1994; Hirayama, 1990; Sandler, 1985).

Studies of tobacco exposures are complicated by the fact that individuals with tobacco exposures are also more likely to be consumers of alcoholic beverages. Since heavy alcohol use is associated with increased breast cancer risk, alcohol use needs to be carefully addressed in these studies. An additional complication in studying the effect of tobacco use is that active smoking is known to have an antiestrogenic effect resulting in, for example, some women having an earlier natural menopause, which would reduce their potential risk for breast cancer. Studies of tobacco exposure and breast cancer have also suggested that individual genetic variations that affect the body's metabolism of compounds from cigarette smoke alter breast cancer risk associated with tobacco exposures (Morabia, 2000; Ishibe, 1998).

It has been estimated that known risk factors account for only 30% of breast cancer cases. A more recent study, however, concluded that almost half of breast cancer cases in the United States population could be accounted for by these risk factors: later age at first birth, never having given birth, higher family income and family history of breast cancer. Inclusion of additional risk factors, including earlier age at menarche, history of benign breast disease, and alcohol consumption, would increase the proportion even further (Madigan and Ziegler, 1995).

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D. Sociodemographics of the CMP Area

As described in Section II-C, breast cancer incidence has been found to be associated with several demographic and socioeconomic factors, including race, national origin, household income, and occupation. Demographics for the CMP area were compared with New York State, New York State excluding New York City, and Suffolk County, with attention given to population characteristics associated with increased risk of breast cancer. The sources of the data were the United States Census for 1990 and 2000. It should be noted that certain items, including race and occupational and industry groupings, are not comparable from one census year to the other, although comparisons within each census year are valid. Complete tables for the CMP area as a whole, the three comparison areas, and each of the seven individual ZIP Codes within the CMP area are presented in *Appendix II-1* and *Appendix II-2*. Highlights of this analysis are described below.

Race is a known risk factor for breast cancer, with incidence for females in New York 30% higher among whites than African Americans (135.8 per 100,000 vs. 99.6 per 100,000 age-adjusted to the 2000 US population, according to the most recent New York State Cancer Registry cancer statistics available on NYS DOH's web site, January 2004). Data for 1990 show that the CMP area was predominantly white (93%), with 4% of the population African American. The percentage of whites in New York State excluding New York City and Suffolk County was about the same as the CMP area, but both New York State excluding New York City and Suffolk County had a slightly higher percentage of African Americans. The racial distribution in New York State was approximately 74% white and 16% African American. Census data for 2000 show similar results.

Another factor that has been associated with breast cancer is socioeconomic status. It has often been observed that breast cancer rates are higher among women in more affluent areas, which has been attributed to a larger concentration of women who have decided to delay childbearing to pursue higher education or employment. Data for 1990 show that there were fewer people in the CMP area living below the poverty level than in New York State

and New York State excluding New York City. The CMP area was comparable to Suffolk County. The median household income was 65% higher in the CMP area compared with New York State, and 45% higher than New York State excluding New York City. Residents of the CMP area who were 25 years of age and older were more likely to have a high school or college diploma compared with all of the other areas. Data for 2000 show similar results, although the occupational and industry groupings used were different than in 1990. The CMP area had a high percentage of professional educators and healthcare workers, relative to the comparison areas. In particular, according to the 1990 Census, 14% of employed persons in the CMP area worked in education. This compares to about 10% in the comparison areas.

Population mobility is an important factor to consider in geographically-based health studies. Data for both 1990 and 2000 show that almost 40% of people over the age of five living in the CMP area, as well as the three comparison areas, were not living in the same house five years previously. People in the CMP area were slightly more likely to have moved from other parts of the same county. There is no indication of substantial migration to the CMP area from other areas of the state.

1. Spatial Analysis Results after Adjustment for Demographic and Socioeconomic Factors

Because it is well known that breast cancer risk varies by demographic and socioeconomic characteristics, the spatial analysis was repeated with adjustment made for variables in addition to age. These additional variables included race, income, seasonality and education.

Age was categorized into 18 five-year age groups up to 85 and older. Race was categorized into three groups: white, black, and other, which includes Asian, Pacific Islander, and American Indian. Claritas ZIP Code-level estimates published in 2000 were used, with the values for 1993-1997 obtained by linear interpolation between the 1990 and 2000 population estimates. The measure used for income was median household income, which was grouped into six categories: less than $30K, $30K- less than $40K, $40K- less than $50K, $50K-less than $60K, $60K- less than $70K, and $70K and over. Claritas ZIP Code-level estimates published in 2000 were used, with the values for 1993-1997 obtained by linear interpolation between the 1990 and 2000 values. Seasonal residence was defined as the percentage of housing units occupied seasonally and came from the 1990 census, reaggregated to the 2000 Claritas ZIP Code definitions. Two measures of education were used: low educational attainment was defined as the percentage of the population over 25 years old without a high school diploma. High educational attainment was defined as the percentage of the population over 25 with a bachelor's degree or higher. Both education measures came from the 1990 census, reaggregated to the 2000 Claritas ZIP Code definitions.

A Poisson regression model was constructed and used to compute expected counts adjusted for these variables in the following way. First, the population of each of the 1,574 ZIP codes in the state was stratified into the 18 age groups and 3 race groups. Next, measures of income, seasonal residence, and education were assigned to each stratum within each ZIP Code. A single measure of income, seasonal residence, and education was used in each ZIP Code. That is, age- and race-specific measures of these variables were not used. Expected counts were

computed for each stratum and then summed to produce an expected count for each ZIP Code. The results of this analysis are presented in *Table 9.*

Table 9. Breast cancer excesses before and after adjustment for demographic and socioeconomic factors, ZIP Codes included in the CMP area, 1993-1997

ZIP Code	Name	Number of cancers observed	Adjusted for age only		Adjusted for multiple factors*	
			Number of cancers expected	Percent Excess	Number of cancers expected	Percent Excess
11789	Sound Beach	31	17.6	76	19.1	62
11727	Coram	100	63.3	58	68.9	45
11766	Mt. Sinai	36	25.0	44	28.1	28
11776	Port Jefferson Station	86	60.9	41	66.6	29
11777	Port Jefferson	43	31.9	35	36.1	19
11733	East Setauket	64	53.1	21	61.1	5
11764	Miller Place	33	33.8	-2	37.6	-12
Total area		393	285.6	38	317.5	24

*Source: NYS Cancer Registry; * Adjusted for age, race, seasonal residence, income and education using post-1990 U.S. Census population estimates provided by Claritas*

The excess of breast cancer cases in each ZIP Code was reduced by adjustment for the demographic and socioeconomic factors. The overall excess decreased from 38% to 24% (the slight discrepancies in numbers are due to updates to the Cancer Registry database). Although still elevated, this area was not selected by SaTScan as being significantly elevated. The excess of breast cancer cases in the three ZIP Codes that form the core of this area (11727, 11766, and 11776) was reduced from 49% to 36%.

2. Length of Residence

U.S. Census figures on migration apply to the entire population of an area, while women with breast cancer make up a specific subset of this population. To learn more about how long women with breast cancer lived at their addresses in the CMP area prior to being diagnosed with breast cancer, NYS DOH staff reviewed available sources of information on length of residence. One of these sources was the *Cole's Cross-Reference Directory* for Suffolk County for the years 1993, 1995 and 1998. The Cole's directories contain lists of residential and business telephone customers with published telephone numbers arranged by street address (address section), and by telephone number (reverse look-up section). The street address section includes a notation as to when each entry was first listed. Another source of information was current property records, obtained from the Suffolk County Real Property Tax Service Agency. As an additional validation of this information, each woman with breast cancer was looked up in the 1987 telephone directory for Suffolk County. This year is five years prior to 1993, the earliest year

of diagnosis of the women included in this study. The reverse look-up feature of the Coles' directories was also used to obtain information on women for whom a telephone number was available.

A set of rules was applied to assign year of first residence based on information from the various data sources. If a woman was found in more than one data source and the information from the data sources did not agree, the earlier of the years of first residence was assigned.

Based on the combination of all data sources available, it was possible to assign a year of first residence to 291 of 402 women living in the CMP area who were identified as being diagnosed with breast cancer between 1993 and 1997, or 72.4%. (Minor discrepancies in total numbers of women with breast cancer are due to updates to the Cancer Registry database.) Women to whom we were able to assign a year of first residence tended to be younger than women to whom we were unable to assign a year of first residence, with a median age of 55 compared with 59. In particular, while 13.8% of women to whom we were able to assign a year of first residence were age 75 or older, 27.0% of women to whom we were not able to assign this information were in this age group. Women for whom we were able and not able to assign a year of first residence were equally likely to be other than white (1.7% vs. 1.8%). Women for whom we were unable to obtain information on year of first residence were more likely to be recorded as Hispanic (3.6% vs. 2.1%), although numbers were small.

Length of residence at the address at breast cancer diagnosis prior to the time of diagnosis was computed as the difference between the year of first residence and the year of breast cancer diagnosis. This value ranged from 0 to 45 years, with a median of 13 years. Seventy-eight percent of women for whom we were able to find information on year of first residence lived at their address at diagnosis for 5 years or more prior to diagnosis, and 62% lived there for 10 years or more. *Table 10* summarizes results for length of residence prior to diagnosis.

Table 10. Length of residence prior to diagnosis, women in the CMP area diagnosed with breast cancer 1993-1997

Length of residence (years)	n	%
0-4	63	22
5-9	48	16
10-19	83	29
20+	97	33
Total assigned	291	100
Unknown	111	
Total women	402	

Results for length of residence are broken down by age group in *Table 11*. As can be seen, length of residence generally increased with increasing age. It should also be noted, however, that age groups 75-84 and 85 and older

have the greatest proportion of women for whom a length of residence prior to breast cancer diagnosis could not be determined.

Table 11. Length of residence prior to diagnosis by age group, women in the CMP area diagnosed with breast cancer 1993-1997

Age group	Median* (years)	5 years or more* (%)	10 years or more* (%)	Unknown (%)
25-34	2	36	27	21
35-44	5	57	31	29
45-54	14	84	63	23
55-64	19.5	87	75	30
65-74	18	75	61	21
75-84	13	91	87	42
85+	22.5	88	88	47
All ages	13	78	62	28

** among those for whom length of residence could be determined*

If all of the women for whom we were unable to find information on year of first residence were assumed to have lived at their address at the time of diagnosis for under 5 years, then 57% of the total would have lived there for 5 years or more. If all the unknowns were assumed to have lived there for five years or more, then 84% of all women would have lived there for 5 years or more.

In summary, the various data sources available allowed us to obtain information on year of first residence for 72% of the women in the CMP area diagnosed with breast cancer between 1993 and 1997. Of these, 78% had lived at their address at the time of breast cancer diagnosis for 5 years or more, and 62% had lived there for 10 years or more. This may be compared to the approximately 60% of all residents (age 5 or older) who lived in the same house five years prior to the 1990 and 2000 U.S. Censuses. This longer length of residence may be related to the fact that women with breast cancer are on average older than the general population and thus may tend to be more residentially stable.

E. Data Evaluation

A strength of the Cancer Mapping Project is the high quality of the source of the cancer data. The statewide spatial statistical analyses of ZIP Code-level breast cancer incidence used data that are uniformly and comprehensively gathered for the entire state by the New York State Cancer Registry. The Cancer Registry meets criteria for timeliness, completeness, and accuracy set by the North American Association for Central Cancer Registries at the gold (highest) level. Population data used in the analyses were the best available at the time. Since data from the 2000 Census were not available at the time of the original mapping of breast cancer incidence, estimates of post-

1990 Census populations were used to account for population changes since the 1990 Census. These data were then validated when the 2000 Census became available. Corrections to the populations resulted in some changes to the findings for individual ZIP Codes, but the overall conclusion of the analysis was not altered. Socioeconomic data from the US Census are also available only for the entire population of the area, and not for particular subgroups such as women over the age of 40 or 50, who are at greatest risk for breast cancer.

The evaluation of length of residence for women with breast cancer was based on various data sources, none of which was designed for this purpose. They each have strengths and limitations that affect both the accuracy of the information we were able to obtain, and the number of women for whom we were able to obtain it. Comparison of the information on year of first residence obtained from different data sources for the same woman has shown at least 60% agreement to within one year, and almost 80% to within five years. We also found that women for whom we were able to obtain information on year of first residence were on average younger than women with breast cancer for whom we were unable to obtain this information, and so the women for whom we were able to obtain length of residence information may not have been entirely representative of all women with breast cancer in the CMP area. For example, it is possible that many of the older women for whom we were not able to obtain information on year of first residence had recently moved to alternative living situations (e.g. senior housing, family care) and so may have been living there for a shorter time prior to their diagnosis than older women for whom we were able to obtain information. Still, it is unlikely that all women for whom we were unable to obtain information lived at their address at the time of diagnosis for under 5 years, and so it is likely that women with breast cancer tend to be longer-term residents than the general population.

The Cancer Mapping Project and CMP Breast Cancer Investigation are geographically-based screening evaluations that assess cancer incidence for groups of individuals. The demographic and socioeconomic information presented here is also for groups of individuals in the CMP follow-up area. Group-level data are useful for suggesting hypotheses for additional research. However, because the individuals within the group vary in terms of their individual risk factors for breast cancer, group-level data do not allow conclusions to be drawn about the causes of a particular individual's cancer.

The issue of migration is an important factor that limits the strength of conclusions that can be drawn from any demographic analysis of a disease with a long latency period such as cancer. Most studies that have identified factors that cause cancer in adults have noted a latency period between the first exposure to the factor that initiated the disease and the appearance of any symptoms of the disease. Latency periods observed in studies of breast cancer have ranged from 5 to 40 years, depending on which factor women were exposed to and the age of the woman at the time of exposure. Consequently, information on possible environmental exposures at the time of cancer diagnosis may not be relevant to the causation of the cancer. Furthermore, although women with breast cancer in the CMP area appear to be somewhat less mobile than the general population, an appreciable proportion of them are still recent arrivals. Information on possible past environmental exposures at the address at the time of diagnosis may therefore not be relevant to the causation of cancers in a considerable proportion of people.

F. Conclusions

The epidemiological evaluation has confirmed that the excess in breast cancer incidence in the CMP area is not likely due to features of disease detection or reporting in the area, or characteristics of the analysis such as population estimation (including underestimation of the population at risk due to seasonal residents). The breast cancer excess has persisted in the years following the original analysis. Examination of the characteristics of cases has identified no population subgroups disproportionately affected, and there is no evidence of any unusual breast cancer cell types.

Further evaluation of the sociodemographic characteristics shows that the CMP area has several characteristics associated with a higher risk of breast cancer. These include a higher percentage of people identified as white, and higher income and education levels. There is also a higher proportion of people employed in the education and health care fields, which have been associated with higher breast cancer incidence in studies that did not control for reproductive factors.

A statistical model was constructed to see how much of the excess in breast cancer incidence could be related to variations in racial composition, income, and educational levels. Socioeconomic factors are not believed to affect breast cancer risk directly, but are correlated with reproductive and lifestyle risk factors for breast cancer such as age at first childbirth and alcohol consumption, which are not as easily measured. In the statistical model, the magnitude of the breast cancer excess in the CMP area was reduced. Although breast cancer incidence in this area was still greater than expected, this area was no longer statistically significant. The particular statistical model that was applied relied on broad, group-level socioeconomic indicators as surrogate measures for individual characteristics related to breast cancer risk. If it were possible to accurately adjust for characteristics related to family history, lifestyle, and reproductive factors known to be related to breast cancer risk, the apparent elevation in the CMP area may have been further decreased. In other words, one cannot rule out the possibility that known reproductive and lifestyle risk factors associated with socioeconomic status could in fact account for most of the increased incidence in the CMP area. On the other hand, it is also possible that other, as yet unknown, risk factors that happen to be related to socioeconomic status could also be playing a role in breast cancer incidence. These as yet unknown risk factors, or interactions among risk factors, could include environmental or occupational exposures as well as behaviors or lifestyle.

Population mobility is an important factor to be considered in any evaluation of the possible role of environmental factors in a disease with a long latency, such as breast cancer. Available information from various sources indicates that women with breast cancer in the CMP area tended to be longer-term residents than the general population in this area. An appreciable number, however, were most likely still recent arrivals, indicating that information on possible past environmental exposures in the CMP area may not be relevant to the causation of breast cancer in some proportion of women.

Any conclusions drawn from this descriptive epidemiologic evaluation of the CMP area should be considered cautiously. The purpose of presenting additional information is to evaluate whether the CMP area stands out as different from the rest of New York State or Suffolk County in any way that might suggest a possible explanation for

elevated breast cancer incidence. This type of evaluation cannot assess whether or not there are causal links between particular demographic or risk factors and health outcomes.

Appendix II-1. Demographic Profile - 1990 U.S. Census CMP, Suffolk Co, ZIP Codes 11727, 11733, 11764, 11766, 11776, 11777, 11789

Appendix II-1								
Region/Study Area ZIP Code(s)	**New York State**		**New York State excluding New York City**		**Suffolk Co.**		**Total All ZIPs in CMP**	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Population:								
Males:								
00-14 years	1,838,553	21.30	1,107,625	21.35	138,598	21.44	10,503	22.53
15-24 years	1,337,058	15.49	815,166	15.71	101,974	15.77	7,233	15.52
25-34 years	1,555,714	18.02	885,075	17.06	113,821	17.60	8,223	17.64
35-44 years	1,325,377	15.35	792,185	15.27	100,904	15.61	8,338	17.89
45-54 years	905,551	10.49	552,871	10.66	75,922	11.74	5,718	12.27
55-64 years	758,912	8.79	473,702	9.13	58,995	9.12	3,447	7.39
65-74 years	571,345	6.62	359,541	6.93	36,656	5.67	1,963	4.21
75-84 years	274,167	3.18	164,023	3.16	16,086	2.49	1,017	2.18
85+ years	65,534	0.76	37,798	0.73	3,634	0.56	177	0.38
Total	8,632,211		5,187,986		646,590		46,619	
Females:								
00-14 years	1,754,488	18.72	1,049,033	19.14	131,552	19.48	10,148	21.11
15-24 years	1,310,088	13.98	776,493	14.17	96,862	14.34	6,930	14.42
25-34 years	1,591,841	16.99	883,703	16.13	113,696	16.83	8,091	16.83
35-44 years	1,404,429	14.99	815,890	14.89	107,110	15.86	8,913	18.54
45-54 years	1,000,877	10.68	587,207	10.72	80,870	11.97	6,059	12.61
55-64 years	873,825	9.33	518,536	9.46	60,535	8.96	3,204	6.67
65-74 years	768,271	8.20	457,007	8.34	45,794	6.78	2,476	5.15
75-84 years	488,977	5.22	284,215	5.19	28,688	4.25	1,778	3.70

Appendix II-1

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co.		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
85+ years	177,848	1.90	107,821	1.97	10,347	1.53	468	0.97
Total	9,370,644		5,479,905		675,454		48,067	
Race:								
White	13,398,003	74.47	9,558,167	89.60	1,192,236	90.19	87,776	92.70
Black	2,860,590	15.90	756,543	7.09	82,473	6.24	3,616	3.82
Amer Indian, Eskimo, Aleut	59,081	0.33	35,120	0.33	3,233	0.24	160	0.17
Asian	689,262	3.83	181,041	1.70	22,185	1.68	2,643	2.79
Other	983,519	5.47	137,020	1.28	21,737	1.64	491	0.52
Total	17,990,455		10,667,891		1,321,864		94,686	
Ethnicity:								
Hispanic	2,151,743	11.96	430,515	4.04	84,238	6.37	4,132	4.36
Non-Hispanic	15,838,712	88.04	10,237,376	95.96	1,237,626	93.63	90,554	95.64
Total	17,990,455		10,667,891		1,321,864		94,686	
Household Type:								
Family	14,927,099	82.97	8,969,896	84.08	1,191,780	90.16	85,766	90.58
Non-Family	2,519,634	14.01	1,320,603	12.38	100,794	7.63	7,714	8.15
Group Quarters	543,722	3.02	377,392	3.54	29,290	2.22	1,206	1.27
Total	17,990,455		10,667,891		1,321,864		94,686	
Group Quarters:								
Prison	90,341		66,833		1,396		0	
Nursing Homes	123,354		81,735		8,110		632	

Appendix II-1

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co.		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Psychiatric Centers	18,166		14,171		4,440		0	
Juvenile Home	7,158		6,018		408		106	
Other Institution	23,541		15,032		1,821		71	
Dorms	168,641		138,202		8,379		0	
Military Quarters	12,898		11,972		73		0	
Shelters	34,813		9,089		1,330		217	
Streets	8,272		285		0		0	
Other Non-Institution	56,538		34,055		3,333		180	
Total	543,722		377,392		29,290		1,206	
Type of Occupied Housing:								
Owner	3,466,277	52.21	2,657,058	69.56	340,347	80.13	24,051	78.18
Renter	3,173,045	47.79	1,162,863	30.44	84,372	19.87	6,711	21.82
Total	6,639,322		3,819,921		424,719		30,762	
Urban/Rural Populations:								
Urban	15,164,245	84.29	7,841,681	73.51	1,273,359	96.33	93,963	99.24
Rural	2,826,210	15.71	2,826,210	26.49	48,505	3.67	723	0.76
Total	17,990,455		10,667,891		1,321,864		94,686	
Education Level, Age 25+:								
No High School Diploma	2,977,604	25.19	1,430,457	20.63	152,118	17.79	7,440	12.43
High School/College Diploma	8,840,965	74.81	5,503,838	79.37	702,925	82.21	52,432	87.57
Total	11,818,569		6,934,295		855,043		59,872	

Appendix II-1

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co.		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Place of Birth:								
New York State	12,147,209	67.52	8,259,528	77.42	1,070,863	81.01	76,190	80.47
Other US State	2,369,057	13.17	1,493,463	14.00	122,905	9.30	10,274	10.85
Native Born Outside US	622,328	3.46	145,970	1.37	23,885	1.81	1,177	1.24
Foreign Born	2,851,861	15.85	768,930	7.21	104,211	7.88	7,045	7.44
Total	17,990,455		10,667,891		1,321,864		94,686	
Residence in 1985, Age 5+:								
Same House	10,385,913	62.03	6,084,609	61.32	824,557	67.09	53,710	61.18
Different House, Same County	3,557,118	21.25	2,213,981	22.31	244,945	19.93	22,093	25.17
Different House, Same State	1,458,672	8.71	979,873	9.88	108,510	8.83	6,698	7.63
Different State	727,621	4.35	487,064	4.91	34,185	2.78	3,663	4.17
Abroad	613,727	3.67	157,068	1.58	16,893	1.37	1,624	1.85
Total	16,743,051		9,922,595		1,229,090		87,788	
Poverty Status:								
Above Poverty	15,204,466	86.97	9,408,305	91.34	1,231,276	95.25	89,982	96.20
Below Poverty	2,277,295	13.03	892,302	8.66	61,389	4.75	3,557	3.80
Total	17,481,761		10,300,607		1,292,665		93,539	
Median Household Income (1989)	$32,965		$37,381		$49,128		$54,553	

Appendix II-1 continued

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co.		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Industry of Employed, Age 16+:								
Agriculture, Fishing, Forestry		1.17		1.76		1.40		0.98
Mining		0.09		0.13		0.06		0.09
Construction		5.16		5.83		6.81		5.87
Manufacturing, nondurable goods		6.26		5.68		4.78		2.70
Manufacturing, durable goods		8.40		11.05		9.77		6.73
Transportation		5.17		4.23		5.65		3.98
Communications/other public utilities		2.72		2.74		2.85		2.32
Wholesale trade		4.17		4.25		5.01		4.02
Retail trade		14.94		16.07		15.99		14.27
Finance, insurance, and real estate		9.29		7.35		8.38		7.21
Business and repair services		5.20		4.36		5.08		3.65
Personal services		2.98		2.56		2.06		1.77
Entertainment/Recreation services		1.54		1.29		1.21		1.15
Health services		10.12		9.74		9.52		11.72
Educational services		9.55		10.59		9.87		13.99
Other services		8.18		7.18		6.28		15.20
Public Adminstration		5.07		5.20		5.27		4.69
Occupation of Employed, Age 16+:								
Executive, administrative, managerial		13.29		13.14		13.44		13.04
Professional specialty		16.69		16.46		15.50		20.85

Appendix II-1 continued

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co.		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Technicians, related support		3.50		3.75		3.57		3.89
Sales		11.20		11.77		12.87		12.26
Administrative support		18.44		17.04		17.88		15.04
Service-Private household		0.52		0.37		0.30		0.20
Service-Protective service		2.50		2.34		2.89		2.55
Service-Other		11.35		10.62		9.23		15.20
Farming, Fishing, Forestry		1.12		1.67		1.29		0.82
Production, Crafts, and Repair		9.42		10.64		11.97		8.70
Machine operators, assemblers, inspectors		5.12		5.28		4.05		2.10
Transportation, material moving		3.68		3.67		3.89		2.83
Handlers, equip cleaners, laborers		3.17		3.25		3.11		2.53

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Population:														
Males:														
00-14 years	2,883	23.58	1,764	21.53	1,197	24.85	1,145	26.48	1,983	20.41	758	17.83	773	25.01
15-24 years	1,656	13.54	1,249	15.24	761	15.80	695	16.07	1,786	18.38	711	16.73	375	12.13
25-34 years	2,493	20.39	1,323	16.14	641	13.31	558	12.90	1,832	18.86	647	15.22	729	23.58
35-44 years	2,262	18.50	1,460	17.82	1,047	21.74	903	20.88	1,491	15.35	653	15.36	522	16.89
45-54 years	1,340	10.96	1,132	13.81	577	11.98	522	12.07	1,255	12.92	614	14.44	278	8.99
55-64 years	752	6.15	742	9.05	317	6.58	231	5.34	775	7.98	456	10.73	174	5.63
65-74 years	574	4.69	364	4.44	123	2.55	177	4.09	358	3.69	242	5.69	125	4.04
75-84 years	245	2.00	142	1.73	120	2.49	82	1.90	183	1.88	147	3.46	98	3.17
85+ years	21	0.17	19	0.23	34	0.71	11	0.25	52	0.54	23	0.54	17	0.55
Total	12,226		8,195		4,817		4,324		9,715		4,251		3,091	
Females:														
00-14 years	2,594	20.37	1,659	20.02	1,234	24.99	1,076	24.49	2,045	20.37	728	16.80	812	24.28
15-24 years	1,844	14.48	1,250	15.08	613	12.41	608	13.84	1,633	16.27	548	12.64	434	12.98
25-34 years	2,484	19.51	1,231	14.85	700	14.18	683	15.55	1,742	17.36	509	11.74	742	22.19
35-44 years	2,479	19.47	1,645	19.85	1,042	21.10	911	20.74	1,562	15.56	734	16.94	540	16.15
45-54 years	1,343	10.55	1,152	13.90	654	13.24	494	11.25	1,451	14.46	701	16.17	264	7.89
55-64 years	785	6.16	668	8.06	307	6.22	213	4.85	700	6.97	404	9.32	127	3.80
65-74 years	694	5.45	432	5.21	183	3.71	239	5.44	462	4.60	276	6.37	190	5.68
75-84 years	362	2.84	213	2.57	200	4.05	121	2.75	387	3.86	305	7.04	190	5.68
85+ years	149	1.17	37	0.45	5	0.10	48	1.09	55	0.55	129	2.98	45	1.35
Total	12,734		8,287		4,938		4,393		10,037		4,334		3,344	

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Race:														
White	21,502	86.15	14,778	89.66	9,628	98.70	8,436	96.78	18,976	96.07	8,128	94.68	6,328	98.34
Black	2,664	10.67	283	1.72	19	0.19	118	1.35	350	1.77	158	1.84	24	0.37
Amer Indian, Eskimo, Aleut	110	0.44	48	0.29	0	0.00	0	0.00	0	0.00	2	0.02	0	0.00
Asian	503	2.02	1,247	7.57	108	1.11	118	1.35	315	1.59	269	3.13	83	1.29
Other	181	0.73	126	0.76	0	0.00	45	0.52	111	0.56	28	0.33	0	0.00
Total	24,960		16,482		9,755		8,717		19,752		8,585		6,435	
Ethnicity:														
Hispanic	1,559	6.25	428	2.60	266	2.73	359	4.12	1,039	5.26	244	2.84	237	3.68
Non-Hispanic	23,401	93.75	16,054	97.40	9,489	97.27	8,358	95.88	18,713	94.74	8,341	97.16	6,198	96.32
Total	24,960		16,482		9,755		8,717		19,752		8,585		6,435	
Household Type:														
Family	22,104	88.56	15,162	91.99	9,252	94.84	8,300	95.22	18,395	93.13	6,900	80.37	5,653	87.85
Non-Family	2,457	9.84	1,305	7.92	463	4.75	392	4.50	1,219	6.17	1,138	13.26	740	11.50
Group Quarters	399	1.60	15	0.09	40	0.41	25	0.29	138	0.70	547	6.37	42	0.65
Total	24,960		16,482		9,755		8,717		19,752		8,585		6,435	
Group Quarters:														
Prison	0		0		0		0		0		0		0	
Nursing Homes	315		0		0		0		45		272		0	
Psychiatric Centers	0		0		0		0		0		0		0	

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Juvenile Home	0		0		0		0		0		106		0	
Other Institution	0		15		9		25		0		22		0	
Dorms	0		0		0		0		0		0		0	
Military Quarters	0		0		0		0		0		0		0	
Shelters	84		0		0		0		74		59		0	
Streets	0		0		0		0		0		0		0	
Other Non-Institution	0		0		31		0		19		88		42	
Total	399		15		40		25		138		547		42	
Type of Occupied Housing:														
Owner	6,124	71.76	4,418	83.79	2,643	88.19	2,374	90.09	4,794	79.59	2,024	68.40	1,674	71.51
Renter	2,410	28.24	855	16.21	354	11.81	261	9.91	1,229	20.41	935	31.60	667	28.49
Total	8,534		5,273		2,997		2,635		6,023		2,959		2,341	
Urban/Rural Populations:														
Urban	24,960	100.00	15,759	95.61	9,755	100.00	8,717	100.00	19,752	100.00	8,585	100.00	6,435	100.00
Rural	0	0.00	723	4.39	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00
Total	24,960		16,482		9,755		8,717		19,752		8,585		6,435	
Education Level, Age 25+:														
No High School Diploma	2,200	13.76	583	5.52	610	10.25	617	11.88	1,958	15.91	771	13.20	701	17.35
High School/College	13,783	86.24	9,977	94.48	5,340	89.75	4,576	88.12	10,347	84.09	5,069	86.80	3,340	82.65

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Diploma														
Total	15,983		10,560		5,950		5,193		12,305		5,840		4,041	
Place of Birth:														
New York State	20,334	81.47	11,865	71.99	8,264	84.72	7,478	85.79	16,239	82.21	6,560	76.41	5,450	84.69
Other US State	2,558	10.25	2,601	15.78	996	10.21	566	6.49	1,501	7.60	1,409	16.41	643	9.99
Native Born Outside US	346	1.39	207	1.26	135	1.38	130	1.49	293	1.48	48	0.56	18	0.28
Foreign Born	1,722	6.90	1,809	10.98	360	3.69	543	6.23	1,719	8.70	568	6.62	324	5.03
Total	24,960		16,482		9,755		8,717		19,752		8,585		6,435	
Residence in 1985, Age 5+:														
Same House	13,062	56.94	8,843	57.24	5,967	66.18	5,477	67.97	11,970	65.14	4,812	58.88	3,579	61.94
Different House, Same County	6,479	28.24	3,785	24.50	2,055	22.79	1,722	21.37	4,271	23.24	2,280	27.90	1,501	25.98
Different House, Same State	2,281	9.94	1,101	7.13	470	5.21	668	8.29	1,335	7.27	439	5.37	404	6.99
Different State	816	3.56	962	6.23	417	4.63	159	1.97	500	2.72	530	6.49	279	4.83
Abroad	301	1.31	759	4.91	107	1.19	32	0.40	299	1.63	111	1.36	15	0.26
Total	22,939		15,450		9,016		8,058		18,375		8,172		5,778	
Poverty Status:														
Above Poverty	23,537	95.91	15,721	95.90	9,538	97.87	8,369	96.28	19,119	97.38	7,657	94.27	6,041	94.20
Below Poverty	1,003	4.09	672	4.10	208	2.13	323	3.72	514	2.62	465	5.73	372	5.80
Total	24,540		16,393		9,746		8,692		19,633		8,122		6,413	

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Median Household Income (1989)	$48,621		$65,693		$60,000		$57,417		$52,567		$58,826		$40,673	
Industry of Employed, Age 16+:														
Agriculture, Fishing, Forestry		0.70		1.21		1.35		0.97		0.89		0.68		1.77
Mining		0.10		0.00		0.00		0.13		0.15		0.19		0.00
Construction		4.92		4.80		8.21		7.07		5.74		4.81		9.35
Manufacturing, nondurable goods		3.29		2.94		1.75		2.56		2.96		0.91		2.92
Manufacturing, durable goods		7.00		5.57		6.03		8.02		6.89		7.78		6.09
Transportation		4.57		2.24		3.85		3.73		4.59		2.79		6.18
Communications/other public utilities		2.19		2.01		2.54		2.98		2.71		2.00		1.54
Wholesale trade		4.35		3.60		4.42		5.08		4.29		2.56		2.95
Retail trade		15.35		13.19		12.29		15.67		15.77		11.97		11.90
Finance, insurance, and real estate		8.53		8.00		6.22		6.12		6.85		5.80		5.86
Business and repair services		4.35		3.03		4.13		3.96		3.61		2.66		2.80
Personal services		2.07		1.23		1.84		1.37		2.07		1.28		2.03
Entertainment/Recreation services		1.07		1.57		1.30		0.27		1.10		0.74		1.97
Health services		10.42		12.58		10.26		13.50		11.15		14.91		12.33

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Educational services		11.26		20.50		17.74		12.91		10.36		18.35		9.98
Other services		14.18		13.82		11.86		11.75		18.11		18.67		18.06
Public Administration		6.00		3.66		6.50		4.20		3.73		3.90		4.23
Occupation of Employed, Age 16+:														
Executive, administrative, managerial		13.76		15.16		11.94		14.83		11.04		14.95		7.66
Professional specialty		16.15		30.33		23.18		20.80		15.64		30.55		14.51
Technicians, related support		3.51		3.44		4.91		3.22		4.15		4.30		4.60
Sales		13.49		12.92		10.34		14.97		11.64		10.02		10.09
Administrative support		17.86		12.80		15.08		15.81		15.70		10.68		12.63
Service-Private household		0.28		0.26		0.00		0.00		0.26		0.25		0.00
Service-Protective service		3.62		1.70		3.54		2.02		1.92		0.76		4.11
Service-Other		14.18		13.82		11.86		11.75		18.11		18.67		18.06
Farming, Fishing, Forestry		0.64		0.75		1.10		0.95		0.86		0.37		1.66
Production, Crafts, and Repair		8.38		4.69		10.40		8.19		11.29		6.05		14.06
Machine operators, assemblers,		2.14		1.06		1.77		1.79		2.43		1.69		5.06

Appendix II-1 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
inspectors														
Transportation, material moving		3.49		1.65		2.46		2.54		3.71		1.30		3.43
Handlers, equip cleaners, laborers		2.50		1.41		3.43		3.13		3.24		0.41		4.14

Appendix II-2. Demographic Profile - 2000 Census CMP, Suffolk Co, ZIP Codes 11727, 11733, 11764, 11766, 11776, 11777, 11789

Appendix II-2								
Region/Study Area ZIP Code(s)	**New York State**		**New York State excluding New York City**		**Suffolk Co**		**Total All ZIPs in CMP**	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Population:								
Males:								
00-14 years	2,010,539	22.01	1,175,843	21.98	161,194	23.21	11,791	22.61
15-24 years	1,274,660	13.95	729,983	13.64	84,577	12.18	7,098	13.61
25-34 years	1,335,832	14.62	685,718	12.82	93,970	13.53	7,185	13.78
35-44 years	1,532,612	16.78	908,164	16.97	126,229	18.18	9,225	17.69
45-54 years	1,226,472	13.43	754,262	14.10	95,955	13.82	7,624	14.62
55-64 years	780,194	8.54	479,752	8.97	63,176	9.10	4,757	9.12
65-74 years	565,080	6.19	356,743	6.67	42,714	6.15	2,798	5.36
75-84 years	324,198	3.55	208,350	3.89	21,598	3.11	1,372	2.63
85+ years	84,667	0.93	51,346	0.96	5,100	0.73	305	0.58
Total	9,134,254		5,350,161		694,513		52,155	
Females:								
00-14 years	1,910,042	19.41	1,113,312	19.82	152,517	21.04	10,875	20.46
15-24 years	1,238,680	12.59	683,451	12.17	78,680	10.85	7,003	13.17
25-34 years	1,391,660	14.14	693,724	12.35	95,883	13.23	7,014	13.19
35-44 years	1,595,980	16.22	926,050	16.48	128,977	17.79	9,243	17.39
45-54 years	1,328,867	13.50	783,840	13.95	101,410	13.99	8,315	15.64
55-64 years	900,222	9.15	522,753	9.30	69,280	9.56	5,004	9.41
65-74 years	720,387	7.32	430,090	7.66	49,801	6.87	2,952	5.55
75-84 years	535,876	5.44	330,143	5.88	33,933	4.68	1,990	3.74

Appendix II-2

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
85+ years	220,489	2.24	134,655	2.40	14,375	1.98	769	1.45
Total	9,842,203		5,618,018		724,856		53,165	
Race:								
White alone	12,891,118	67.93	9,314,066	84.92	1,200,119	84.55	92,226	87.57
Black alone	2,986,242	15.74	869,863	7.93	97,215	6.85	4,158	3.95
Amer Indian, Alaskan Native alone	79,314	0.42	42,657	0.39	4,009	0.28	220	0.21
Asian alone	1,044,423	5.50	256,313	2.34	34,143	2.41	4,371	4.15
Other race alone	1,360,298	7.17	272,174	2.48	53,129	3.74	2,264	2.15
Two or more races	615,062	3.24	213,106	1.94	30,754	2.17	2,081	1.98
Total	18,976,457		10,968,179		1,419,369		105,320	
Ethnicity:								
Hispanic	2,865,016	15.10	703,486	6.41	149,422	10.53	6,741	6.40
Non-Hispanic	16,111,441	84.90	10,264,693	93.59	1,269,947	89.47	98,579	93.60
Total	18,976,457		10,968,179		1,419,369		105,320	
Household Type:								
Family	15,486,400	81.61	9,057,736	82.58	1,255,789	88.48	91,026	86.43
Non-Family	2,909,293	15.33	1,511,890	13.78	134,991	9.51	10,198	9.68
Group Quarters	580,764	3.06	398,553	3.63	28,589	2.01	4,096	3.89
Total	18,976,457		10,968,179		1,419,369		105,320	
Group Quarters:								
Correctional Institutions	108,088		86,686		1,471		0	
Nursing Homes	123,852		81,372		8,179		937	

Appendix II-2

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Other Institution	30,322		18,334		1,602		18	
Dorms	174,111		136,250		8,365		2,661	
Military Quarters	8,598		8,368		39		0	
Other Non-Institution	135,490		67,021		8,922		671	
Total	580,461		398,031		28,578		4,287	
Type of Occupied Housing:								
Owner	3,739,247	52.99	2,827,114	70.06	374,371	79.77	27,719	79.29
Renter	3,317,613	47.01	1,208,158	29.94	94,928	20.23	7,240	20.71
Total	7,056,860		4,035,272		469,299		34,959	
Urban/Rural Populations:								
Urban	16,601,126	87.48	8,592,848	78.34	1,377,156	97.03	105,320	100.00
Rural	2,375,331	12.52	2,375,331	21.66	42,213	2.97	0	0.00
Total	18,976,457		10,968,179		1,419,369		105,320	
Education Level, Age 25+:								
No High School Diploma	2,626,324	20.94	1,163,634	16.02	130,174	13.81	6,293	9.18
High School/College Diploma	9,916,212	79.06	6,101,956	83.98	812,227	86.19	62,260	90.82
Total	12,542,536		7,265,590		942,401		68,553	
Place of Birth:								
New York State	12,384,940	65.26	8,420,389	76.77	1,127,869	79.46	82,475	78.31
Other US State	2,204,323	11.62	1,405,758	12.82	110,856	7.81	10,824	10.28

Appendix II-2

Region/Study Area ZIP Code(s)	New York State		New York State excluding New York City		Suffolk Co		Total All ZIPs in CMP	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Native Born Outside US	519,061	2.74	144,931	1.32	22,119	1.56	973	0.92
Foreign Born	3,868,133	20.38	997,101	9.09	158,525	11.17	11,048	10.49
Total	18,976,457		10,968,179		1,419,369		105,320	
Residence in 1995, Age 5+:								
Same House	10,961,493	61.76	6,398,595	62.28	854,055	64.73	59,272	60.42
Different House, Same County	3,876,450	21.84	2,310,294	22.49	295,589	22.40	25,420	25.91
Different House, Same State	1,463,942	8.25	940,280	9.15	107,972	8.18	7,296	7.44
Different State	726,477	4.09	425,234	4.14	35,285	2.67	4,081	4.16
Abroad	720,748	4.06	199,105	1.94	26,476	2.01	2,034	2.07
Total	17,749,110		10,273,508		1,319,377		98,103	
Poverty Status:								
Above Poverty	15,757,697	85.41	9,572,105	90.34	1,310,375	94.03	96,526	95.00
Below Poverty	2,692,202	14.59	1,023,264	9.66	83,171	5.97	5,078	5.00
Total	18,449,899		10,595,369		1,393,546		101,604	
Median Household Income (1999)	$43,393		$49,632		$65,288		$72,204	

Appendix II-2 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent of Total**	**Number**	**Percent**
Population:														
Males:														
00-14 years	2,646	21.11	2,146	20.97	1,467	26.31	1,216	26.83	2,566	23.06	823	18.57	927	24.90
15-24 years	1,714	13.68	2,008	19.62	705	12.64	463	10.22	1,206	10.84	531	11.98	471	12.65
25-34 years	2,083	16.62	1,008	9.85	593	10.63	483	10.66	1,791	16.09	604	13.63	623	16.73
35-44 years	2,077	16.57	1,504	14.70	1,104	19.80	906	19.99	2,148	19.30	771	17.40	715	19.20
45-54 years	1,902	15.18	1,529	14.94	907	16.27	766	16.90	1,326	11.92	637	14.38	557	14.96
55-64 years	1,136	9.06	920	8.99	495	8.88	361	7.97	1,090	9.80	554	12.50	201	5.40
65-74 years	645	5.15	647	6.32	174	3.12	217	4.79	716	6.43	324	7.31	75	2.01
75-84 years	298	2.38	390	3.81	119	2.13	98	2.16	207	1.86	141	3.18	119	3.20
85+ years	32	0.26	80	0.78	12	0.22	22	0.49	78	0.70	46	1.04	35	0.94
Total	12,533		10,232		5,576		4,532		11,128		4,431		3,723	
Females:														
00-14 years	2,367	18.33	2,035	19.95	1,454	25.78	1,141	24.42	2,328	20.52	685	15.08	865	22.47
15-24 years	1,608	12.45	2,214	21.70	581	10.30	502	10.74	1,205	10.62	411	9.05	482	12.52
25-34 years	2,021	15.65	935	9.16	634	11.24	598	12.80	1,627	14.34	599	13.19	600	15.58
35-44 years	2,194	16.99	1,735	17.00	1,128	20.00	754	16.14	1,998	17.61	717	15.79	717	18.62
45-54 years	2,029	15.71	1,527	14.97	951	16.86	842	18.02	1,630	14.37	736	16.21	600	15.58
55-64 years	1,299	10.06	931	9.12	427	7.57	353	7.55	1,176	10.36	620	13.65	198	5.14
65-74 years	790	6.12	510	5.00	267	4.73	236	5.05	661	5.83	325	7.16	163	4.23
75-84 years	470	3.64	217	2.13	164	2.91	189	4.04	532	4.69	258	5.68	160	4.16
85+ years	135	1.05	99	0.97	33	0.59	58	1.24	189	1.67	190	4.18	65	1.69

Appendix II-2 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent of Total**	**Number**	**Percent**
Total	12,913		10,203		5,639		4,673		11,346		4,541		3,850	
Race:														
White alone	19,851	78.01	17,107	83.71	10,838	96.64	8,813	95.74	20,198	89.87	8,244	91.89	7,175	94.74
Black alone	2,681	10.54	609	2.98	73	0.65	106	1.15	372	1.66	181	2.02	136	1.80
Amer Indian, Alaskan Native alone	166	0.65	19	0.09	7	0.06	0	0.00	13	0.06	9	0.10	6	0.08
Asian alone	1,092	4.29	2,021	9.89	189	1.69	143	1.55	557	2.48	271	3.02	98	1.29
Other race alone	1,004	3.95	272	1.33	27	0.24	56	0.61	602	2.68	230	2.56	73	0.96
Two or more races	652	2.56	407	1.99	81	0.72	87	0.95	732	3.26	37	0.41	85	1.12
Total	25,446		20,435		11,215		9,205		22,474		8,972		7,573	
Ethnicity:														
Hispanic	2,631	10.34	910	4.45	330	2.94	275	2.99	1,839	8.18	502	5.60	254	3.35
Non-Hispanic	22,815	89.66	19,525	95.55	10,885	97.06	8,930	97.01	20,635	91.82	8,470	94.40	7,319	96.65
Total	25,446		20,435		11,215		9,205		22,474		8,972		7,573	
Household Type:														
Family	22,018	86.53	15,994	78.27	10,453	93.21	8,779	95.37	20,123	89.54	7,017	78.21	6,642	87.71
Non-Family	3,273	12.86	1,606	7.86	703	6.27	387	4.20	1,917	8.53	1,411	15.73	901	11.90
Group Quarters	155	0.61	2,835	13.87	59	0.53	39	0.42	434	1.93	544	6.06	30	0.40
Total	25,446		20,435		11,215		9,205		22,474		8,972		7,573	
Group Quarters:														
Correctional	0		0		0		0		0		0		0	

Appendix II-2 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent of Total**	**Number**	**Percent**
Institutions														
Nursing Homes	0		350		0		0		306		281		0	
Other Institution	0		0		0		0		0		18		0	
Dorms	0		2,661		0		0		0		0		0	
Military Quarters	0		0		0		0		0		0		0	
Other Non-Institution	143		27		59		46		126		240		30	
Total	143		3,038		59		46		432		539		30	
Type of Occupied Housing:														
Owner	6,186	65.83	5,077	89.79	3,258	91.08	2,701	95.34	5,958	79.95	2,543	74.57	1,996	75.72
Renter	3,211	34.17	577	10.21	319	8.92	132	4.66	1,494	20.05	867	25.43	640	24.28
Total	9,397		5,654		3,577		2,833		7,452		3,410		2,636	
Urban/Rural Populations:														
Urban	25,446	100.00	20,435	100.00	11,215	100.00	9,205	100.00	22,474	100.00	8,972	100.00	7,573	100.00
Rural	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00
Total	25,446		20,435		11,215		9,205		22,474		8,972		7,573	
Education Level, Age 25+:														
No High School Diploma	1,936	11.31	723	6.01	400	5.71	401	6.82	2,054	13.54	423	6.49	356	7.37
High	15,175	88.69	11,309	93.99	6,608	94.29	5,482	93.18	13,115	86.46	6,099	93.51	4,472	92.63

Appendix II-2 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent of Total**	**Number**	**Percent**
School/College Diploma														
Total	17,111		12,032		7,008		5,883		15,169		6,522		4,828	
Place of Birth:														
New York State	19,644	77.20	14,438	70.65	9,702	86.51	7,940	86.26	18,238	81.15	6,307	70.30	6,206	81.95
Other US State	2,412	9.48	2,821	13.80	1,001	8.93	861	9.35	1,580	7.03	1,423	15.86	726	9.59
Native Born Outside US	327	1.29	211	1.03	60	0.53	55	0.60	117	0.52	95	1.06	108	1.43
Foreign Born	3,063	12.04	2,965	14.51	452	4.03	349	3.79	2,539	11.30	1,147	12.78	533	7.04
Total	25,446		20,435		11,215		9,205		22,474		8,972		7,573	
Residence in 1995, Age 5+:														
Same House	13,195	55.83	11,210	58.23	6,545	63.36	6,079	70.65	13,092	62.97	4,847	57.11	4,304	61.45
Different House, Same County	6,999	29.61	4,045	21.01	2,780	26.91	1,795	20.86	5,542	26.66	2,328	27.43	1,931	27.57
Different House, Same State	1,903	8.05	2,262	11.75	504	4.88	500	5.81	1,208	5.81	575	6.78	344	4.91
Different State	974	4.12	1,014	5.27	397	3.84	204	2.37	591	2.84	553	6.52	348	4.97
Abroad	565	2.39	720	3.74	104	1.01	26	0.30	358	1.72	184	2.17	77	1.10
Total	23,636		19,251		10,330		8,604		20,791		8,487		7,004	
Poverty Status:														
Above Poverty	23,813	93.96	16,816	95.44	10,979	97.98	8,845	96.26	21,018	95.07	8,041	93.66	7,014	92.86

Appendix II-2 continued

Region/Study Area ZIP Code(s)	Coram ZIP Code 11727		East Setauket ZIP Code 11733		Miller Place ZIP Code 11764		Mt. Sinai ZIP Code 11766		Pt. Jefferson Station ZIP Code 11776		Pt. Jefferson ZIP Code 11777		Sound Beach ZIP Code 11789	
Characteristic	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent**	**Number**	**Percent of Total**	**Number**	**Percent**
Below Poverty	1,531	6.04	804	4.56	226	2.02	344	3.74	1,090	4.93	544	6.34	539	7.14
Total	25,344		17,620		11,205		9,189		22,108		8,585		7,553	
Median Household Income (1999)	$59,338		$98,056		$82,309		$85,156		$65,494		$69,426		$55,847	

III. Toxicological Evaluation

A. Characterization of the Scientific Literature

Breast cancer is a common disease. In industrialized countries, about one woman in eight will ultimately be diagnosed with breast cancer. The consensus among cancer researchers is that breast cancer is a "*multifactorial*" disease, meaning it has many personal lifestyle, genetic, and environmental risk factors that interact in ways not yet fully understood to increase a person's chance of developing the disease. Some of these risk factors have been identified, particularly those related to genetics, lifestyle, and reproductive history. For example, individuals who have inherited certain genes (e.g., BRCA1 or BRCA2) have a high lifetime probability of developing breast cancer. Examples of other known risk factors are heavy use of alcoholic beverages, diet, early puberty, delayed childbirth, and delayed menopause. However, all of these known risk factors account for less than half of the total cases of breast cancer, suggesting that there are other risk factors that have not yet been identified.

Some synthetic chemicals found as contaminants in air, water, soil, and some foods are suspected environmental risk factors for breast cancer, based in part on scientific evidence that they have the ability to influence the development of cancer (*carcinogenesis*) in breast tissue. Moreover, some of these chemicals have been identified at very low levels in breast tissue or in breast milk, where they can potentially act in one of three ways to increase the risk of cancer.

Some of these chemicals are converted by enzymes present in breast tissue to highly-reactive chemicals that bind to DNA and/or otherwise cause DNA damage. This damage, which is the underlying cause of all cancers, can take the form of a gene mutation (a change in the cell's genetic code). Some mutations, if not repaired, can become a permanent part of the cell's DNA and may start the cell down the road to cancer. Other environmental chemicals appear to increase the rate at which breast cells proliferate (i.e., grow, divide and multiply). In the laboratory, cells isolated from breast tissue or breast milk and exposed to high concentrations of these chemicals in cell culture proliferated more rapidly than unexposed cells. These experiments suggest that at high concentrations these kinds of chemicals might also increase the rate at which cells proliferate in the intact breast. Rapid cell proliferation (which requires rapidly replicating DNA) increases the likelihood that a mutation in the DNA will occur and become fixed in the dividing cells' DNA before it can be repaired. Stimulating cell proliferation is one of the ways that the female hormone estrogen, which is a known risk factor for breast cancer, may accelerate carcinogenesis. Some environmental chemicals mimic the ability of estrogen to stimulate cell proliferation.

Yet other environmental chemicals bind to cell structures (called receptors) and activate genes that produce enzymes that convert other chemicals in the body to highly reactive chemicals capable of damaging DNA, and perhaps causing mutations.

Thus, some environmental chemicals have the potential to initiate and/or alter the carcinogenic process in breast tissue, and may be environmental risk factors for breast cancer in the CMP area. The next section describes the

development of a Classification System for use in identifying environmental risk factors for breast cancer. This system for classifying environmental chemicals was used to evaluate the likelihood that environmental exposures (evaluated in *Chapter IV. Environmental Exposure Evaluation*) could be linked to breast cancer incidence in the CMP area, as described in *Chapter V. Integration*. C. Methods.

B. Classification system

NYS DOH researchers developed a Classification System and applied it to evaluate about 150 substances to determine the likelihood that a given agent is an environmental risk factor for human breast cancer. The substances evaluated were based in part on the following two lists of substances that caused mammary (breast) tumors in experimental animals:

- Substances found to produce mammary tumors in rats and mice by the National Toxicology Program (NTP) of the U.S. National Institutes of Health.
- Substances reported in the scientific literature to cause mammary tumors in various animal species, tabulated by the Carcinogenic Potency Database (CPDB) at the University of California at Berkeley.

Some of the substances on the NTP and CPDB lists were not evaluated by NYS DOH researchers because information about the use of these substances indicated that their presence in the environment would be extremely unlikely. For example, acronycine and phenesterin are two substances that were excluded from this evaluation because these substances were used exclusively in laboratory experiments in the 1970s to evaluate their effectiveness as anti-cancer drugs.

NYS DOH researchers also evaluated a number of substances that are frequently found in the environment, including the groundwater of New York State. Several persistent organochlorine chemicals that are known to accumulate in breast tissue and to stimulate the proliferation of breast cells in culture were also evaluated. Other chemicals evaluated included those selected from a list of environmental chemicals (predominantly pesticides) compiled by the Program on Breast Cancer and Environmental Risk Factors (BCERF) in New York State at Cornell University.

NYS DOH researchers evaluated individual environmental agents using a weight-of-evidence analysis of three types of data:

1. Human data from studies of individuals exposed to the agent (perhaps mixed with other agents) in the workplace or in the environment.
2. Animal data from laboratory experiments where animals were exposed to repeated high doses of the substance and observed for a major portion of their lives.
3. Data from studies to determine a substance's mode-of-action (i.e., the sequence of biological events and processes that cause a cell to change in a way that can result in a disease, such as breast cancer).

The analysis examined the quality and quantity of each type of data, compared data across data types for consistency, and then classified the collective evidence to determine the likelihood that a given agent is an environmental risk factor for human breast cancer.

Each of the three data types provides information that the other data types cannot, and together they provide a good scientific foundation for an assessment. Good human data can provide direct evidence that an exposure to an agent is or may be a risk factor for breast cancer. However, sufficient human data necessary to establish a cause-and-effect relationship between human exposures and increased breast cancer incidence are almost always absent for the following reasons:

- Information about human exposure to the agent is seldom well-documented and is often confounded by simultaneous exposure to other agents.
- The population studied often includes too few women to enable reliable conclusions regarding breast cancer risk to be reached.
- The exposures may have been at too low a level, or over too short a period of time.
- The follow-up time between the exposure and cancer incidence may have been too short to allow for the full expression of the lengthy process of cancer development.
- Women studied are typically of working age when their breast tissue is relatively resistant to DNA damage (in contrast to adolescent breast tissue, which is more vulnerable).
- Data about other critical factors (e.g., lifestyle factors, reproductive, and family histories) may not be available.

More often, good human data show, at best, that a specific factor is consistently associated with an increased risk of breast cancer.

Unlike people, laboratory animals live under controlled environmental conditions where uncertainty and variation can be greatly restricted. In these experimental studies, the cancer incidence of a group of animals exposed to known amounts of a substance is compared to the cancer incidence of a control group of animals that are not exposed to the substance. Because both control and exposed animals have the same genetic background and environment (except for the chemical exposure), any important difference in cancer incidence between the two groups provides evidence for a cause-and-effect relationship between exposure and cancer. Such experiments can establish that an increase in the exposure to a particular substance causes an increase in the risk of breast tumors in that animal species. However, these studies provide only indirect evidence about the potential of a substance to cause breast cancer in humans.

The validity of using animal data to evaluate human risk from environmental exposures depends on two major assumptions:

The animal species respond to the substance in a manner similar to the way humans respond.

The effects observed at the high doses used in animal experiments also occur at much lower doses associated with human environmental exposures

A substance that causes breast cancer in one animal species may not cause breast cancer in another species of animal or in people. This is because animals and humans do not always respond the same way to the same substance. In addition, uncertainties exist in determining if the substance that causes cancer at high doses will also cause cancer at low doses. If the biological events that are necessary to cause cancer occur only at high doses, then those events will not occur at lower doses and consequently will not cause cancer at lower doses. These types of uncertainties in extrapolating results from high dose animal studies to lower human environmental doses is addressed in part by examining the third type of evidence – data on mode-of-action.

Gaining additional information about the mode-of-action by which an environmental agent causes mammary tumors in a laboratory species at high doses, and determining whether the same mode-of-action applies to humans exposed at lower doses, can greatly strengthen predictions that the data obtained in animals are relevant to human breast cancer. These data provide evidence that animals and humans respond or do not respond in the same way to a chemical exposure. They can also provide evidence that effects seen at high doses also occur, but probably at a lower rate, at low doses. The following two examples illustrate the usefulness of mode-of-action evidence.

Chemical A causes mammary cancer in experimental studies in rats and is weakly linked to breast cancer in humans. Short-term studies show that enzymes found in rat and human breast tissue convert Chemical A to a highly-reactive metabolite that induces a mutation in the same gene (known to be involved in carcinogenesis) in rat and human breast cells. These data provide strong evidence of a common mode-of-action for rat and human cancers. Moreover, studies showing that mutations are induced at both high and low doses provide evidence that the cancers seen in high-dose animal studies may also occur at low doses, including those to which humans are exposed in the environment. Collectively, these mode-of-action data strengthen the hypothesis that Chemical A is a risk factor for human breast cancer, even at low environmental exposures.

Mode-of-action data can also provide evidence that exposure to a chemical is not a risk factor for human breast cancer. Chemical B causes mammary tumors in experimental studies in rats. This effect has not been seen in humans. Short-term studies in rats show that Chemical B induces hormonal changes that do not occur in humans. Additional studies show that these hormonal changes in rats stimulate cell proliferation in mammary tissue, possibly increasing the risk of mammary cancer in rats. Collectively, these data suggest that Chemical B may not induce the hormonal responses in humans that initiate or accelerate the carcinogenic process in breast tissue in rats. Thus, the mode-of-action responsible for the induction of mammary carcinogenesis by Chemical B in rats is apparently not possible in humans. These data weaken the hypothesis that Chemical B is a risk factor for human breast cancer.

To apply an objective and consistent approach to classifying agents based on their potential as risk factors for human breast cancer, NYS DOH researchers developed a classification system. This classification scheme provides the major criteria used to evaluate the weight of evidence from human, animal, and mode-of-action studies to categorize agents as *Known, Probable, Possible, Potential, Not Classifiable,* or *Not Likely to be a Risk Factor* for breast cancer. This classification scheme is modeled after similar schemes used by the International Agency for

Research on Cancer (IARC) and the Cornell BCERF. The classifications of risk factors for human breast cancer are presented in *Table 12* and outlined in *Table 13.*

Table 12. Classification system

(Adapted from IARC [http://monographs.iarc.fr/monoeval/eval.html] January 2004 and Snedecker [http://envirocancer.cornell.edu/criticalEval/CriticalEval.cfm] January 2004)

Known risk factor for human breast cancer	Typically, only substances with sufficient human evidence to establish a causal relationship between exposure and breast cancer are placed in this class.
Probable risk factor for human breast cancer	Substances are placed in this class based on various combinations of evidence from the three types of studies. These combinations include (a) Strong human evidence, but the total evidence from human, animal and mode-of-action studies is not sufficient to establish a causal relationship; (b) Limited human evidence **and** (i) sufficient animal evidence, regardless of the evidence from mode-of-action studies **OR** (ii) limited animal evidence **and** strong mode-action evidence; (c) Inadequate or nonexistent human evidence **and** sufficient animal evidence **and** strong mode-of-action evidence.
Possible risk factor for human breast cancer	Substances are placed in this class based on various combinations of evidence from the three types of studies, but the overall evidence is weaker than that for probable risk factor. These combinations include (a) Limited human evidence **and** limited or inadequate animal data **and** limited mode-of-action evidence; (b) Limited human evidence **and** nonexistent or negative animal evidence **and** strong or limited mode-of-action evidence; (c) Inadequate or nonexistent human evidence **and** sufficient animal evidence **and** inadequate, nonexistent, or negative mode-of-action evidence; (d) Inadequate or nonexistent human evidence **and** limited animal evidence **and** strong or limited mode-of-action evidence.
Potential to affect breast cancer risk	Contaminants are placed in this class when (a) Human data are inadequate or nonexistent **and** animal evidence is inadequate, nonexistent, or negative **and** mode- of-action evidence is strong or limited; (b) Human data are inadequate or non-existent, **and** animal evidence is limited **and** the mode-of-action data are inadequate, nonexistent, or negative.
Not classifiable as a risk factor for human breast cancer	Contaminants are placed in this class when data from all three types of studies are nonexistent, inadequate, conflicting, or negative (but not consistently negative).
Unlikely to be a risk factor for human breast cancer	Contaminants are placed in this class when there is consistent evidence from at least two of the three types of studies that the substance is not a risk factor for human breast cancer and there is no conflicting evidence from the third study type. A substance may not be placed in this group based on lack of data.

Definitions:

(A) Human studies

Sufficient evidence *of breast carcinogenicity in humans:* *Several studies in humans show consistently strong evidence of statistically significant and dose-related increases in incidence of breast cancer following exposure to putative agent (allowing for a reasonable latency time and for vulnerable windows of exposure). These studies, with supporting experimental data as needed, must demonstrate coherence, specificity, appropriate time and dose relationships, and biological plausibility.*

Strong evidence*: Statistical evidence in human studies comparable to that described above, but lacking in data required to establish causation.*

Limited evidence*: A positive association has been observed between exposure to the agent and breast cancer, but chance, bias or confounding factors could not be ruled out.*

Inadequate evidence*: Suggestive data was too limited to permit a statistical assessment, or conflicting conclusions from different studies precluded a weight of evidence determination, or apparently negative data did not meet the requirements stated below for negative evidence.*

No data*: No human data have been reported regarding a possible association of exposure to the given substance and breast cancer.*

Negative evidence*: Consistent lack of evidence of exposure-related breast cancer, even though relatively high-level exposure was documented during a vulnerable life interval, and adequate follow-up time was allowed.*

(B) Experimental animal studies

Sufficient evidence *of mammary carcinogenicity in animals:* *Clear evidence of exposure-related malignant or combination of benign and potentially malignant tumors in (a) two or more species of animals, (b) two or more strains or both genders of a single species, or (c) two or more independent studies in one species carried out under different protocols (e.g., lifetime bioassay and transplacental carcinogenicity). Data from (b) and (c) must be supported by systemic tumors in fatty and/or hormonally-responsive tissues in a second species.*

Limited evidence*: Clear evidence of a carcinogenic effect does not meet the requirements stated above, or involves only benign lesions that are not believed to be potentially pre-malignant.*

Inadequate evidence*: Results lacked statistical detail, or were classified by the authors of the study as* equivocal *based on lack of a statistically-significant dose-response effect. Alternatively, apparently negative data did not meet the requirements stated below for* negative evidence.

No data*: Data on the induction (or non-induction) of mammary tumors by the given substance have not been reported in any species.*

Negative evidence*: Evidence consistently indicates lack of mammary carcinogenicity of the agent in well-conducted studies in at least two species of experimental animals.*

(C) Mode of action data

Strong evidence*: The data (molecular, cellular, and/or toxico-kinetic) support a likely mode of action indicating (a) that the substance probably induces an effect that initiates or accelerates the carcinogenic process in some human tissue (at least for a sensitive sub-population)* or *(b) that the substance, or an active metabolite, is distributed to breast tissue and may be genotoxic.*

Limited evidence*: The data suggest a potential mode of action. However, the data may be inconsistent, confounded by various difficulties, or limited in quantity or quality.*

Inadequate evidence: *The available data suggest that the substance may not be capable of initiating or accelerating the carcinogenic process, but the clarity needed to be designated as negative evidence is not present.*

No data*: There are no relevant data available relating to a potential mode of carcinogenic action of the environmental agent.*

Negative evidence*: The weight of the mechanistic data indicate that the environmental agent is probably not capable of initiating or accelerating the carcinogenic process in human breast.*

(D) Examples of other potentially relevant data*, which may be used, on a case-by-case basis, to clarify issues and thus facilitate decisions in borderline cases:*

Xenobiotic-metabolizing enzymes in human breast.
Presence of target substance or metabolites in breast or milk.
Polymorphisms of xenobiotic-metabolizing and cyto-protective enzymes: possible influence on the carcinogenesis process.

Interactions of environmental agent and endogenous antioxidants and hormones.
Potential biomarkers: Protein adducts in blood; unique gene adducts and mutations.
Induction of tumors in non-mammary tissues, particularly systemic tumors in lipid-rich and/or hormonally responsive tissues.
Structure-Activity Relationships with known carcinogen.

Each categorization considered evidence from human, animal, and mode-of-action studies. However, human evidence, in general, was given more weight in the analysis than animal data, which were given more weight than the mode-of-action data. For example, human data from good studies, in principle, are sufficient to categorize an agent as a known or probable risk factor for human breast cancer. This is true even if good animal data or mode-of-action data show that under experimental conditions, the agent did not cause mammary cancer in animals or cause effects consistent with a hypothesized mode-of-action for breast cancer.

Human data receives preferential treatment because only human data can provide direct evidence about risk factors for human breast cancer.

Unfortunately, human studies are limited for many agents. As a result, the focus of the analysis shifts to animal and mode-of-action evidence, making animal data increasingly important in the evaluation as evidence from human studies becomes weaker. Similarly, mode-of-action evidence becomes more important as animal data become weaker. Examples of this shift in focus as the evaluation moves from human data to animal data and/or to mode-of-action are shown in *Table 13.*

Table 13. Examples of Weight-of-Evidence Classifications

Category	Weight-of-evidence required to classify an agent		
	Human	**Animal**	**Mode-of-Action**
Known (1)	Sufficient	Sufficient or less than Sufficient	Strong or less than Strong
Probable (2A)	Strong	Limited or less than Limited	Strong or less than Strong
	Limited	Limited	Strong
		Sufficient	Strong or less than Strong
	Inadequate or no Data	Sufficient	Strong
Possible (2B)	Limited	Limited or Inadequate	Limited
		No Data or Negative	Strong or Limited
	Inadequate or No Data	Limited	Strong or Limited
		Sufficient	Less than Limited
Potential (2C)	Inadequate or No Data	Inadequate, No Data, Negative	Strong or Limited
		Limited	Inadequate, No Data or Negative
Not Classifiable (3)	Inadequate or No Data	Negative	Inadequate or No Data
		Inadequate or No Data	Negative
		Inadequate or No Data	
Not Likely to be Risk Factor (4)	Negative	Negative	Inadequate, No Data or Negative
	Inadequate or No Data		Negative

All terms used to characterize the weight of evidence (i.e. sufficient, strong, etc.) are defined below Table 12.

NYS DOH researchers used the Breast Cancer Risk Factor Classification Scheme to classify 165 agents as risk factors for human breast cancer. They focused on agents that have been linked with human breast cancer and/or animal mammary cancer. Researchers did not attempt to evaluate and categorize substances that have been reported or suspected of causing cancer in other organs in humans and/or animals. NYS DOH researchers will evaluate additional substances based on the results of the environmental exposure evaluation in *Chapter IV.* The toxicologic evaluation and discussion of breast cancer and non-cancer health risks associated with any additional substances discovered during the environmental exposure evaluation will be covered in *Chapter V. Integration*.

C. Results

Table 14 provides the summary results by categories for the toxicological evaluations completed to date. *Table 15* provides the draft categories for all of the agents evaluated. Only one agent, ionizing radiation (i.e., gamma rays from nuclear reactions and medical x-rays), has been classified as a known environmental risk factor for human breast cancer.

Table 14. Summary results: number of agents evaluated by category according to their potential to be a risk factor for human breast cancer

Category	Agents
Known risk factor for human breast cancer	1
Probable risk factor for human breast cancer	21
Possible risk factor for human breast cancer	63
Potential to affect breast cancer risk	44
Not classifiable as a risk factor for human breast cancer	35
Unlikely to be a risk factor for human breast cancer	3

Table 15. Results of weight of evidence analysis to evaluate agents as risk factors for human breast cancer

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
1. Known Risk Factor (1)			
Gamma/x-rays	Sufficient	Sufficient	Strong
2A. Probable Risk Factor (21)			
Alcoholic Beverage Consumption	Strong	Inadequate	Strong
Benzene	Limited	Sufficient	Strong
Benzo[a]pyrene	Limited	Sufficient	Strong
1,3-Butadiene	No Data	Sufficient	Strong
Cigarette smoking (based on data from genetically-susceptible individuals exposed at an early age)			
Active	Strong	Limited	Limited
Environmental (second hand) tobacco smoke	Strong	Limited	Limited
Dibenz[a,l]pyrene	Limited	Limited	Strong
1,2-Dibromoethane (ethylene dibromide)	Inadequate	Sufficient	Strong
1,2-Dichloroethane (ethylene dichloride)	Inadequate	Sufficient	Strong
Diethylstilbestrol	Inadequate	Sufficient	Strong
Estrogen-HRT-Premarin	Strong	Limited	Limited
Estrogen-HRT-Prempro	Strong	Limited	Limited
Ethylene oxide	Limited	Limited	Strong
Glycidol	No Data	Sufficient	Strong
Ionizing Radiation			
Neutrons	Limited	Sufficient	Strong
Light (and work) at Night	Strong	Limited	Limited
PhIP (2-amino-1-methyl-6-phenyl-imidazo[4,5-b] pyridine	Inadequate	Sufficient	Strong
Polycyclic aromatic hydrocarbons (PAH) capable of forming Bay-region Diol-epoxides	Limited	Sufficient	Strong
Pyrolysis products (mixed)	Limited	Sufficient	Strong
Urethane	No Data	Sufficient	Strong
Vinyl Chloride	Inadequate	Sufficient	Strong
2B. Possible Risk Factor (61)			
Acrylamide	Inadequate	Limited	Limited
Acrylonitrile	No Data	Limited	Limited

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
4-Aminobiphenyl	Inadequate	Limited	Strong
Aryl amines	Inadequate	Limited	Strong
Benzidine	No Data	Limited	Strong
FR-1138® [2,2'-bis(bromomethyl)-1,3-propanediol]	No Data	Limited	Limited
Chlorophenoxy herbicides (w/ TCDD contamination)	Limited	No Data	Limited
Chloroprene	No Data	Limited	Limited
2,4-Diaminotoluene	No Data	Limited	Strong
1,2-Dibromo-3-chloropropane (DBCP)	Inadequate	Limited	Strong
2,3-Dibromo-1-propanol	No Data	Limited	Strong
3,3'Dichlorobenzidine	No Data	Limited	Strong
Dichloromethane (methylene chloride)	Limited	Limited	Limited
Dichlorvos (DDVP)	Inadequate	Limited	Limited
Dieldrin	Limited	Negative	Limited
3,3'-Dimethoxybenzidine	No Data	Limited	Strong
3,3'-Dimethylbenzidine	No Data	Limited	Strong
2,7-/2,5-Dinitrofluorene	No Data	Limited	Strong
1,6-/1,8-Dinitropyrene	No Data	Limited	Strong
2,4-Dinitrotoluene (DNT)	No Data	Limited	Strong
1,2-Diphenylhydrazine (hydrazo-benzene)	No Data	Limited	Limited
Emf – Extremely Low Frequency (emf-ELF)	Limited	Inadequate	Limited
Estrogen-Oral Contraceptives	Limited	Inadequate	Limited
Etridiazole (terrazole)	No Data	Limited	Limited
Folpet	No Data	Limited	Limited
Furosemide	No Data	Limited	Limited
Heterocyclic Amines	Inadequate	Limited	Strong
Hydrazobenzene (diphenyl-hydrazine)	No Data	Limited	Limited
Ionizing Radiation -			
Hydrogen-3 (tritium)	No Data	Limited	Limited
Iodine-131	Limited	Limited	Limited
Plutonium-239	Limited	Limited	Limited
Radium-226	Limited	Limited	Limited
IQ (2-amino-3-methylimidazo[4,5-f]quinoline)	Inadequate	Limited	Strong

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
Isoprene	No Data	Limited	Limited
MeIQ (2-amino-3,4-dimethyl-imidazo[4,5-f]-quinoline)	Inadequate	Limited	Strong
4,4'-Methylenebis(2-chloroaniline) (MOCA)	No Data	Limited	Strong
Methylene chloride (See dichloromethane.)			
NFTA (N-[4-(5-Nitro-2-furyl)-2-thiazolyl]acetamide)	No Data	Limited	Limited
Nitroarenes	No Data	Limited	Strong
2-Nitrofluorene	No Data	Limited	Strong
Nitrofurans	No Data	Limited	Strong
Nitrofurazone	No Data	Limited	Strong
Nitromethane	No Data	Limited	Limited
1-/4-Nitropyrene	No Data	Limited	Strong
ortho-Nitrotoluene (2-nitrotoluene)	No Data	Limited	Strong
Ochratoxin A	No Data	Limited	Limited
9-oxo-2,7-dinitrofluorene	No Data	Limited	Strong
9-oxo-2-nitrofluorene	No Data	Limited	Strong
9-oxo-2,4,7-trinitrofluorene	No Data	Limited	Strong
Phosmet	Inadequate	Limited	Limited
Polychlorinated biphenyls (PCBs)	Limited	Negative	Limited
2,3,7,8-subst polychlorinated dioxins (PCDD)	Limited	Limited	Limited
Procarbazine	Inadequate	Limited	Limited
Sulfallate	No Data	Sufficient	Limited
2,3,7,8-Tetrachlorodibenzodioxin (TCDD)	Limited	Limited	Limited
2,4- & 2,6-Toluene diisocyanate (TDI)	Inadequate	Limited	Limited
ortho-Toluidine hydrochloride	No Data	Limited	Strong
1,2,3-Trichloropropane	No Data	Limited	Limited
Trp-P-2 (3-amino-1,4-dimethyl-5H-pyrido[4,3-b] indole)	Inadequate	Limited	Strong
Viruses			
Epstein-Barr virus	Limited	No Data	Limited
Human mammary tumor virus (HMTV)	Limited	Limited	Limited

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
2C. Potential Risk Factor (44)			
Acetaldehyde	Inadequate	Inadequate	Strong
Acrylic Acid	No Data	Inadequate	Limited
Aldicarb	No Data	Inadequate	Limited
Aldrin	Inadequate	Negative	Limited
Aniline	Inadequate	Negative	Limited
Arsenic	Inadequate	Inadequate	Strong
Beryllium	Inadequate	Inadequate	Limited
Cadmium	Inadequate	Inadequate	Strong
Carbon tetrachloride	Inadequate	Inadequate	Limited
Chlordane	Inadequate	Negative	Limited
C.I. Acid Red 114	No Data	Inadequate	Limited
C.I. Basic Red 9 Monohydrochloride	No Data	Inadequate	Limited
Cytembena	No Data	Limited	No Data
DDT congeners & metabolites	Inadequate	Negative	Limited
1,1-Dichloroethane	No Data	Inadequate	Limited
1,1-Dichloroethene (vinylidene chloride)	Inadequate	Inadequate	Limited
1,2-Dichloropropane	No Data	Inadequate	Limited
1,3-Dichloropropene	Inadequate	Negative	Limited
Diesel Particulate Matter	Inadequate	Inadequate	Limited
Diethanolamine	Inadequate	Inadequate	Limited
Endosulfan	Inadequate	Inadequate	Strong
Ethylbenzene	No Data	Inadequate	Strong
Ethylene glycol monomethyl ether (2-methoxyethanol) (EGME)	Inadequate	Inadequate	Limited
Ethylene thiourea	Inadequate	Inadequate	Limited
Hexachlorobenzene	Inadequate	Inadequate	Limited
Heptachlor	Inadequate	Negative	Limited
Indium phosphide	No Data	Inadequate	Limited
Ionizing Radiation			
Cesium-137	No Data	Inadequate	Limited
Radon-222	Inadequate	Negative	Limited
Strontium-90	No Data	Inadequate	Limited
Malathion	Inadequate	Inadequate	Limited
Mancozeb	Inadequate	Inadequate	Limited
Metam Sodium	Inadequate	Inadequate	Limited

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
Methoxychlor	Inadequate	Negative	Limited
Methylene diphenyl diisocyanate (MDI) and polymeric MDI (PMDI)	Inadequate	Negative	Limited
Methyleugenol	No Data	Inadequate	Limited
Methyl isothiocyanate	No Data	Limited	No Data
Mirex	No Data	Negative	Strong
N-Nitrosodiethanolamine	Inadequate	Inadequate	Limited
2,3,7,8-subst Polychlorinated furans (PCDF)	Inadequate	Negative	Limited
Propionaldehyde	Inadequate	Inadequate	Limited
Reserpine	Inadequate	Limited	No Data
Toluene	Inadequate	Inadequate	Strong
Toxaphene	Inadequate	Inadequate	Limited
Vinylidene chloride (see 1,1-Dichloroethene)			
Xylenes	Inadequate	Inadequate	Limited
3. Not Classifiable as Risk Factors (35)			
Alachlor	Inadequate	Inadequate	Inadequate
Atrazine	Inadequate	Inadequate	Inadequate
Bromoform	Inadequate	Negative	Inadequate
Bromodichloromethane	Inadequate	Negative	Inadequate
Captan	Inadequate	Negative	Inadequate
Carbaryl (Sevin)	Inadequate	Negative	Inadequate
Chloroacetophenone	No Data	Inadequate	Inadequate
Chloroform	Inadequate	Negative	Inadequate
Chlorophenoxy herbicides (w/out TCDD contamination)	Inadequate	Inadequate	Inadequate
Chlorpyrifos	Inadequate	Negative	Inadequate
Clonitralid	No Data	Inadequate	No Data
DCPA (Dacthal)	No Data	Inadequate	Negative
Dibromochloromethane	Inadequate	Negative	Inadequate
Dicamba (3,6-Dichloro-*o*-anisic acid)	Inadequate	Inadequate	Inadequate
Dimethyl phthalate	Inadequate	Inadequate	Inadequate
Endrin	Inadequate	Inadequate	Negative
Ethylene glycol monobutyl ether (2-butoxy-ethanol) (EGBE)	Inadequate	Negative	Inadequate
Ethylene glycol monoethyl ether (2-ethoxyethanol) (EGEE)	Inadequate	Inadequate	Inadequate

Agent and Category	Human data evidence	Animal data evidence	Mode-of- action data
Horticultural Oil	Inadequate	Inadequate	Inadequate
Hydrochloric Acid	Inadequate	Inadequate	Inadequate
Hydrofluoric Acid	Inadequate	Inadequate	Inadequate
Ionizing Radiation			
Polonium & daughters	No Data	Inadequate	Inadequate
MECOPROP (MCPP) (2-[2-methyl-4-chloro-phenoxy] propionic acid	Inadequate	Inadequate	Inadequate
Methyl ethyl ketone	Inadequate	Inadequate	Negative
Metolachlor	No Data	Negative	Inadequate
Ozone	Inadequate	Negative	Inadequate
Permethrin	No Data	Negative	Inadequate
Propylene glycol mono-*t*-butyl ether (PGBE)	No Data	Negative	Inadequate
Sevin (See carbaryl)			
Tetrachloroethene (PERC)	Inadequate	Negative	Inadequate
1,2,4-Trichlorobenzene	No Data	Inadequate	Inadequate
1,1,1-Trichloroethane (Methyl chloroform)	Inadequate	Inadequate	Inadequate
Trichloroethene (trichloroethylene) (TCE)	Inadequate	Negative	Inadequate
Trihalomethanes: chloroform ($CHCl_3$), $CHBr_3$, $CHBrCl_2$, $CHBr_2Cl$	Inadequate	Negative	Inadequate
2,2,4-Trimethylpentane	No Data	No Data	Inadequate
Vinclozolin	No Data	Inadequate	Negative
4. Unlikely to be a Risk Factor for Human Breast Cancer (3)			
2,4-dichlorophenoxyacetic acid, its salts and esters	Inadequate	Negative	Negative
Hexane	No Data	Negative	Negative
Methyl tertiary-butyl ether (MTBE)	Inadequate	Negative	Negative

IV. Environmental Exposure Evaluation

A. Introduction

The environmental exposure evaluation is designed to assess evidence of possible elevated environmental exposures in the CMP area. The outcome of this evaluation is a list of contaminants that might have been present in the CMP area at higher concentrations than in other areas of the state or compared to state standards. Contaminants considered elevated are further evaluated (see *Chapter V. Integration)* to consider the likelihood that possible exposures could be related to breast cancer and other health outcomes in the CMP area.

As part of the investigation, an *Initial Environmental Inventory* (*Appendix IV-2*) was prepared documenting environmental data sets and sources of potential contaminants in the CMP area that might be used as part of the environmental exposure evaluation. This inventory was presented to members of the CMP communities at a public availability session in June 2002. At that event, area residents also reported their environmental concerns to the State Health Department. Based on the number of comments made by area residents, major community environmental concerns included the following:

- Air emissions from industries, residential heating oil and diesel emission sources;
- Pesticide use by homeowners and area businesses;
- Hazardous waste sites;
- Drinking water;
- Industrial sites such as Brookhaven National Labs, Northville Industry Corporation East Setauket Terminal and the Port Jefferson Power Station;
- Electromagnetic Fields (EMF); and
- Radon

More details about community environmental concerns and the evaluation of data corresponding to these concerns are discussed in detail in *Section IV-E Environmental Data Review and Results*. *Appendix VI-1* provides a list of community environmental concerns and where they are addressed in this final report. *Sections IV-B* and *IV-C* describe the methods used to conduct this evaluation. *Section IV-D Limitations of this Analysis* describes limitations associated with using existing data sets for characterizing historical exposures.

B. Criteria Applied to Evaluate Environmental Data

For purposes of this report, *environmental data* are defined as existing data indicating the presence of quantifiable levels of contaminants. *Exposure* is the measure of an individual's contact with contaminants in the environment. Obtaining adequate environmental data for estimating possible exposures was a limiting factor in this evaluation.

The existing environmental data sets were not developed for evaluating exposure and each lacked certain necessary data for this purpose. To address issues associated with variability of the data sets and to provide as consistent an approach as possible, NYS DOH researchers developed criteria to select environmental data meeting minimal guidelines for estimating possible environmental exposures. This chapter describes the seven criteria that were used to evaluate environmental data. The results of evaluating of data sets are summarized in *Appendix IV-1.*

1. Definitions of Criteria

1) ***Do the data provide information that links it to the CMP area and are data sufficient to allow for a full evaluation of exposure in the CMP area?***

This criterion has two components. The first component requires that the environmental data be geographically linked to the CMP area. For example, data on pesticide use statewide or across Suffolk County are less useful for this investigation than data specific to the CMP area. The second component requires that the data set include adequate representation of the geographic area and the *analyte (*the specific chemical measured) to evaluate exposure. For example, data on sampling results for private drinking water wells would be considered complete if the majority of wells in the CMP area were tested for the same chemicals during the same time frame. Researchers examined both components together to apply this criterion, and answered the question with a “yes” or “no.” Data classified as “yes” for this criterion will be given priority for the investigation process.

2) ***What proportion (or portion) of the population is potentially exposed?***

This criterion involves the assessment of the relative size of the potentially-exposed population in the CMP area. Data that provide exposure information about a large portion of the population in the CMP area were considered to provide *widespread* exposure information. Environmental data relevant to a small portion of the population in the CMP area were classified as *localized.* Data were given a *none* classification when environmental data indicated that no one in the CMP area was exposed to a contaminant. Finally, some environmental data were classified as *unknown* if they provided no information on population exposure in the CMP area. Data classified as providing widespread exposure information were given priority in this phase of the evaluation.

3) ***How direct of an exposure measure is the data?***

Some environmental data provided direct evidence of exposure to a contaminant (e.g., biomonitoring data) or direct evidence of the presence of a contaminant in the environment (e.g., sampling data). These data were classified as *direct evidence*. All other forms of data represented indirect evidence of exposure or indirect evidence of the presence of a contaminant in an environmental medium. These types of data included estimated (modeled) levels of a contaminant or data on releases of a chemical to the environment (e.g., data on industrial emissions of a chemical, data on pesticide applications). These data were classified as *indirect evidence*. Other data provided information on the production, storage or transportation of chemicals in a geographic area. These data were classified as *limited evidence* for investigation purposes. Finally, some data provided *marginal evidence* about

potential exposure or about the presence of a contaminant in the environment (e.g., data on pesticide sales, data on location of factories). Data that provide direct and indirect evidence were given higher priority in the investigation process than data of limited or marginal evidence about potential exposure or levels in the environment.

4) ***Do the data provide evidence of a completed exposure pathway?***

The Agency for Toxic Substances and Disease Registry's *Public Health Assessment Guidance Manual* defines a completed exposure pathway as consisting of the following five elements:

1) Source of contamination (source of contaminant release into the environment, or the environmental media responsible for causing contamination at a point of exposure if the original source of contamination is unknown);
2) Environmental media and transport mechanisms (environmental media include waste materials, groundwater, surface water, air, surface soil, subsurface soil, sediment, and biota);
3) Point of exposure (a location of potential or actual human contact with a contaminated medium, e.g., residence, business, residential yard, playground, campground, waterway or water body, contaminated spring or hand-drawn well, food services, etc.);
4) Route of exposure (means by which the contaminant actually enters or contacts the body, such as ingestion, inhalation, dermal contact, and dermal absorption); and
5) Receptor population (persons who are exposed or potentially exposed to the contaminants of concern at a point of exposure).

An exposure pathway is considered *complete* when all five of the above elements are present. These data were given priority for investigation. If one or more of the five elements were absent, the exposure pathway was considered *potentially complete* and was given lower priority in the investigation. Data sets that provided no evidence of a complete or potentially complete exposure pathway (*incomplete* exposure pathway) were given the lowest priority in the investigation.

5) ***Are the data temporally relevant for this investigation?***

The time between exposure to a potentially cancer-causing agent and the time at which a person develops cancer is called the *latency period*. Given the latency period for breast cancer, which is anywhere between 5 and 40 years and the CMP study population (diagnosed between 1993 and1997), researchers are interested in evaluating exposures that could have occurred some time between 1953 and 1992. Narrowing this time period would require factoring in additional information such as the time of each woman's diagnosis, the particular environmental exposure being evaluated and the age of the woman during the time of her potential exposure. For the most part, even the oldest environmental data sets evaluated did not include data prior to the 1970s. Because of data limitations and the complexity of cancer latency, exposure data that pre-dated the cancer incidence data were considered *highly relevant* and were given the highest priority for the investigation. Data that represented a time period that was concurrent or nearly concurrent with the cancer incidence data, or that were considered reasonable surrogates for highly relevant data, were classified as *moderately relevant*. Data that represented a time period that

was later than the cancer incidence data and data that were not reasonable surrogates for relevant exposures were classified as *not relevant*.

6) ***Are the environmental data evaluated or included in another, better, data set?***

Some of the environmental data were represented in multiple data sets and, in terms of the criteria described above, certain data sets were superior to others. For example, US EPA's Toxic Release Inventory (TRI) data provides data on estimated emissions of many pollutants to air. Other data sets (i.e., U.S. EPA's Cumulative Exposure Project and National-scale Air Toxics Assessment data) used TRI emissions data to estimate the levels of certain pollutants in air. The latter data sets were considered more relevant in characterizing potential exposures in the CMP area. Therefore, data sets classified as *yes* for this criterion were initially given lower priority for investigation, while those classified as *no* were given higher priority.

7) ***Other***

This last criterion provides a place to consider other relevant factors that may be unique to specific environmental information.

The outcome of the application of the above criteria to the available environmental data is summarized in *Section IV E*. Conclusions about the use of each data set evaluated include the following:

- Data may be used for further investigation, proceed with a comparison to comparison areas or background data.
- Data present limited information for integration, but may be used in other aspects of the investigation.
- Data do not provide enough information for further investigation.

C. Approach for Estimating Potentially Elevated or Unusual Exposure

DOH researchers developed an approach to estimate elevated or unusual exposures in the CMP area. The preferred method was to compare contaminant area measures for the CMP area to comparison area measures. An elevated or unusual exposure is defined when the contaminant measure for CMP was higher than the comparison area. Based on the criteria described in *Section B*. *Criteria Applied to Evaluate Environmental Data,* environmental data sets identified as providing sufficient exposure information were evaluated for elevated or unusual exposures.

Since the data sets vary in content and in terms of their ability to characterize exposure, there was some variability in methods used to determine if a particular data set provided evidence of elevated or unusual exposure in the CMP area. The general approach was to define a measure of exposure (i.e., an estimated air concentration for a specific pollutant, a measured level of a specific pollutant in water, an estimated amount of applied pesticide product) for each data set for the CMP area, and a comparable measure of exposure for one or more other geographic area(s). These other geographic areas are referred to as *comparison areas*. The selected comparison areas were (1) Suffolk County (excluding the CMP area); (2) New York State (excluding New York City); and (3) all

of New York State. Suffolk County was chosen because it is expected to be similar to CMP in terms of its environment, degree of urbanization and population density. The two New York State comparison areas were chosen to represent the average measure of exposure for a contaminant across the state. Researchers also selected these comparison areas to be consistent with the epidemiological evaluation described in *Chapter II* of this working draft report. Where possible, researchers compared measures of exposure between the CMP area and all three comparison areas.

To determine if possible exposures in the CMP area were elevated or unusual, researchers calculated an *exposure ratio* by dividing the measure of exposure for the CMP area by the comparable measure of exposure for each comparison area. Researchers made the judgement to consider an exposure ratio of greater than one (calculated using any of the comparison areas) as an indicator of an elevated or unusual exposure in the CMP area. Researchers ranked exposure ratios for each data set to prioritize chemicals for evaluating breast cancer and non-cancer health risks in *Chapter V. Integration.*

Section E. Environmental Data Review and Results provides more information about how individual data sets were evaluated to identify elevated exposures in the CMP area. This section also presents the results of each of the evaluations.

D. Limitations of this Analysis

The environmental exposure evaluation is the most complicated aspect of this investigation. Because researchers wanted to evaluate as many local environmental concerns as possible, data sets were selected to estimate possible elevated or unusual exposures based on their ability to meet the data evaluation criteria and the number of alternative data sets available. In almost every case, data that were not developed for this type of evaluation were used.

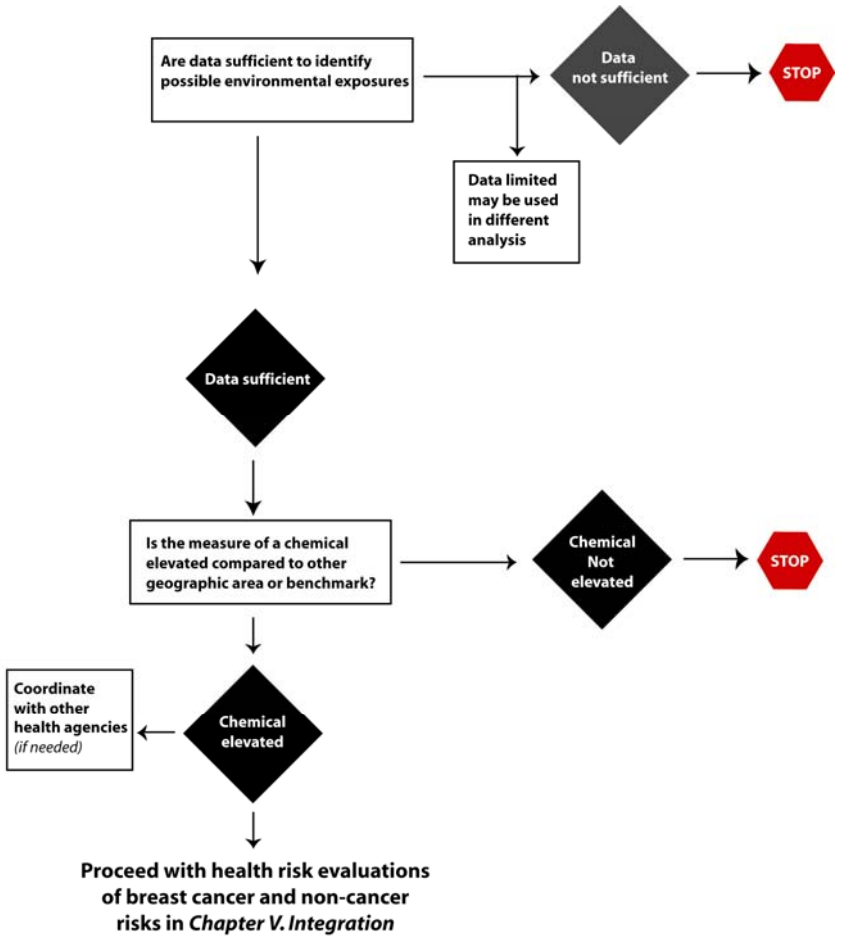
The most central limitation of this evaluation is that it uses data indicating levels of contaminants in the environment as a surrogate for human exposure. In every case, it is uncertain whether the study population had contact with the documented levels of contaminants and if that contact could have occurred during a time period that would be related to the development of breast cancer. As discussed in *Section II-D* a significant portion of the population may not have lived in the area at the time of documented exposure. In addition, breast cancer is a multi-factoral disease, and it is not well-understood how the environment may interact with known risk factors to increase a woman's risk of the disease. State Health researchers describe other uncertainties associated with the each specific analysis in more detail in *Section IVE. Environmental Data Review and Results.*

E. Environmental Data Review and Results

1. Introduction

Information in this section is presented by environmental concern. Here researchers describe the application of the criteria provided in *Section IV-B* to select the best available data sets to estimate possible exposures. Researchers also describe the application of methods outlined in *Section IV-C* to estimate elevated or unusual exposures. The process for estimating environmental exposures is shown in *Figure 3.*

Figure 3. Flow Diagram of the Results of the Environmental Exposure Evaluation



2. Radon

a. Introduction

Radon is a radioactive gas that comes from the decay of radium in the soil. Radon is a colorless, odorless, invisible gas that only can be detected through the use of proper equipment and techniques. Chronic exposure to elevated radon levels has been linked to an increased incidence of lung cancer in underground uranium miners.

Radon is continually generated from radium in rocks, soil, water and materials derived from rocks and soils, such as certain building materials. The radon concentration in a home is dependent on the type of soil on which the home is built. Cracks and openings in the building foundation provide the pathway for radon in the soil to enter a home. Other important factors that affect how much radon will be found throughout a home include the amount of ventilation and airflow patterns within a house.

Radon concentration in air is measured in units of picoCuries per Liter (pCi/L). The NYS DOH and the US EPA use 4 pCi/L as a recommended action level. When testing indicates that the radon level in the lowest primary living area of the home is above this level, the NYS DOH recommends that the homeowner take appropriate corrective action to reduce these levels.

b. Evaluation of Radon Data

The NYS DOH has data about household radon levels in New York State (http://www.health.state.ny.us/nysdoh/radon/radonmaps.htm) (1999). NYS DOH staff prepared maps for each county in New York State showing the estimated percent of homes with greater than 4 pCi/L indoor radon for the towns and cities in the county. The maps were prepared using a statewide database of more than 45,000 basement screening measurements and more than 11,000 long-term living area measurements. These measurements have been made through an ongoing detector distribution program begun by the State Health Department in 1986. Researchers evaluated the quality of this data set for characterizing historical radon exposure in the CMP area and determined this data set was adequate for comparing radon exposures in CMP with other geographic areas in New York State.

c. Radon Exposure Comparisons and Results

As part of the DOH's data set, the Department has radon measurements for 58 homes in the Town of Brookhaven, including the CMP area. Our researchers did not separate out the data for the CMP area from the remainder of the Town of Brookhaven.

Based on a statistical evaluation of those data, our researchers evaluated the likelihood that homes in Brookhaven would exceed the radon action level and compared those statistics with those for Suffolk County and New York State.

Based on this evaluation, about 0.7% of the all of the homes in Brookhaven exceeded the 4 pCi/L action level for radon in the living area and 5% of the homes exceeded the action limit for radon in the basement. On average about 1% of homes in Suffolk County exceeded the 4 pCi/L action level in the living area and 6% exceeded the action level in the basement. Statewide about 5% of the homes exceed the action level in the living area and about 18% exceeded the action level in the basement statewide.

The Department released limited data on radon levels in basements only for June of 2003. Data on radon levels in living areas are not available at this time. These data are available at the town level; 70 homes in the Town of Brookhaven were evaluated as a result of this study. Based on the data collected, about 1.4% of basements in homes in the Town of Brookhaven detected radon levels greater than or equal to 4 pCi/L. In the June 2003 data, 5.8% of homes in Suffolk County exceeded the 4 pCi/L action level for radon in basements.

Because the Town of Brookhaven had a low rate of exceeding the action level and the rates there were lower than comparison areas, radon was not considered a significant environmental exposure in the CMP area. Because radon levels can vary from home to home, the State Health Department still recommends that individual homeowners test home radon levels in every community in New York State regardless of local trend data.

3. Air Quality

a. Introduction

Air pollution was a concern frequently raised by the residents of the CMP area. People were concerned about specific sources of air pollution and the general air quality. Specifically people identified concerns about emissions from a local power plant, residential use of heating oil, diesel sources (such as trains and boats) and certain industrial facilities. To address air quality concerns, NYS DOH researchers examined a number of data sources. Because the databases were not developed for this type of investigation and varied in content, their ability to provide relevant exposure information was evaluated. Those data sources with the most useful exposure information were used to determine if air pollution levels in the CMP area were elevated. This section of the report provides a general overview of air pollutants and the evaluation of exposure information.

b. Air Pollutants and Air Pollutant Standards

An air pollutant is a substance (such as a chemical, dust, smoke or pollen) that is present in air as a solid (particulate), gas (vapor) or liquid (mist), or a combination of these. Air pollution is the presence of those substances in the air at levels (concentrations) greater than would normally be found or considered desirable. It comes from many different human sources such as cars, buses, trucks, factories, power plants and dry cleaners, as well as natural sources such as windblown dust and wildfires. Although air pollution is typically thought of as an outdoor air problem, sources also exist inside homes and places of work. Examples include tobacco smoke, home heating appliances, new carpeting and household products such as paints, cleansers, and pest-control agents.

The US EPA has been regulating certain air pollutants since the 1970 Clean Air Act. For certain pollutants, called "criteria pollutants," including carbon monoxide, nitrogen dioxide, sulfur dioxide, particulate matter, ozone and lead, the US EPA has established National Ambient Air Quality Standards (NAAQS). Primary Standards were established, designed to protect human health with an adequate margin of safety. Secondary Standards are designed to protect public welfare, including protection against decreased visibility and damage to animals, crops and buildings. Additional information about criteria pollutants is available on the US EPA's web site at http://www.epa.gov/ebtpages/airairpolcriteriaairpollutants.html (June 2005)

In 1990, the Clean Air Act was amended to include a list of "hazardous air pollutants" (also known as "air toxics") selected by Congress on the basis of potential health and/or environmental hazard. There currently are 188 listed hazardous air pollutants including chemicals such as benzene, which is found in gasoline; tetrachloroethene (PERC), which is emitted from dry cleaning facilities; methylene chloride, which is used as a solvent and paint stripper; and some metals such as cadmium, mercury, and chromium. The Clean Air Act requires US EPA to regulate emissions of hazardous air pollutants from a list of industrial sources called "source categories" (e.g., boat manufacturing, gasoline distribution, and municipal and hazardous waste combustors). Additional information about hazardous air pollutants is available on the US EPA's web site at http://www.epa.gov/ebtpages/airairpolhazardousairpollutantshaps.html (June 2005)

c. Sources of Air Quality Information

NYS DOH researchers identified a number of databases on outdoor air quality, and information about those databases was first reported in the *Initial Environmental Inventory.* NYS DOH researchers found no data on indoor air quality, other than radon, for the CMP area.

The databases originally identified in the *Inventory* include the following:

- **Air monitoring data—** US EPA's Air Quality System database that contains data of concentrations of chemicals in outdoor air
- **Emissions inventories—** US EPA's Toxic Release Inventory (TRI) and the Aeormetric Information Retrieval System (AIRS) that contain data of chemical emissions from facilities; NYS DEC's Permit-to-Construct/Certificate-to-Operate database contains permitted air emission limits for chemicals from most stationary industrial sources in New York State that were operating in September 1996
- **Modeled data—** US EPA's Cumulative Exposure Project (CEP) and National-scale Air Toxics Assessment (NATA) that contain data of estimated concentrations of chemicals in outdoor air

d. Evaluation of Air Quality Information

NYS DOH researchers applied the screening criteria discussed in *Section IV-B* to assess the usefulness of the information in the databases for evaluating possible exposures in the CMP area. The results of that assessment for

each database are detailed in *Appendix IV-1.* A brief description of each database and a summary of the evaluation are provided below.

1) ***Air Monitoring Data***

As described in the *Initial Environmental Inventory*, US EPA's Air Quality System database contains data from 10 air quality monitoring stations that operated in or near the CMP area for various times since 1965. Half of monitoring stations operated within the area, the other half operated outside of the area but near the seven ZIP Code boundaries. All five of the monitoring stations within the seven ZIP Codes ceased operating by 1984. Four of the five air monitoring stations located outside of the seven ZIP Codes also ceased operating by 1984. The remaining active monitoring station, located in Holtsville, New York, began operating in January 2000.

The air pollutants measured at these stations are sulfur dioxide, total suspended particulate matter, ozone, carbon monoxide, nitric oxide, nitrogen dioxide, total non-methane hydrocarbons (also called volatile organic compounds [VOCs] which react with other pollutants, in the presence of sunlight, to form tropospheric ozone), methane, and particulate matter less than 2.5 microns ($PM_{2.5}$) in diameter. NYS DOH researchers evaluated this database using the screening criteria, and the outcome of that evaluation is shown in *Appendix IV-1*. Overall, data on measured concentrations of chemicals in outdoor air would be considered preferable to data on chemical emissions or data on estimated chemical concentrations for characterizing human exposure to air pollutants.

The US EPA Air Quality System monitoring results had many characteristics desirable for characterizing exposures in the CMP area. Even though toxicological data do not indicate that the substances measured in this network are environmental risk factors for cancer, (see Part III. Toxicological Evaluation for a discussion on environmental risk factors for breast cancer) NYS DOH researchers evaluated these data and obtained additional information from NYS DEC to characterize air quality in the region for those pollutants.

The monitoring data for most of the pollutants do not indicate an exceedance of air quality standards. Long Island, which is considered part of the New York Metropolitan region by US EPA, has been designated as a "nonattainment area" (an area that does not meet the NAAQS) for ozone since an ozone standard was first introduced in the early 1970s. The concentrations of ozone on Long Island are comparable to levels throughout the entire New York City area, while most of the other counties in New York State do not exceed the NAAQS for ozone.

Although the concentrations of $PM_{2.5}$ in Long Island have not exceeded the NAAQS since the standards were introduced in 1997, it has been designated as non-attainment since 2004 only because it is part of the larger New York Metropolitan region that exceeds national standards. The concentrations of $PM_{2.5}$ on Long Island are below the levels measured in the New York City area. For more information about ozone and $PM_{2.5}$ monitoring, visit NYS DEC's site at http://www.dec.state.ny.us/website/dardata/airmon/index.htm. Only ozone exceeded the NAAQS and therefore was evaluated further in *Chapter V*. *Integration*.

The other monitored pollutants (total non-methane hydrocarbons and methane) do not have corresponding NAAQS or NYS DEC guideline values. For those pollutants, NYS DOH researchers sought comparable monitoring data from nearby monitors (such as monitors on Long Island) for comparisons and did not find any data.

2) ***Emissions Inventories***

NYS DOH researchers evaluated three data sets that provide information on the types and amounts of chemicals emitted from industrial facilities. These data sets are US EPA's TRI, AIRS and NYS DEC's Permit-to-Construct/Certificate-to-Operate database. TRI provides data on the amount of certain chemicals released to air (and other media) by industrial facilities. Specific types of industrial facilities that meet certain reporting criteria are required to report information on those releases to US EPA for inclusion in this database. (Examples of these reporting criteria include minimum number of employees and manufacturing, using, or releasing a certain number of pounds of a toxic chemical.) More information about TRI is found at http://www.epa.gov/tri/.

Since the development of the *Initial Environmental Inventory,* NYS DOH researchers have learned that the AIRS database is no longer maintained by US EPA, and that US EPA's National Emissions Inventory (NEI) has replaced AIRS for emissions information.

NYS DEC's Permit-to-Construct/Certificate-to-Operate (PC/CO) database provides historical information on permitted emissions for most stationary industrial sources in New York State. Prior to 1996, a Permit-to-Construct (PC) was required to be filed before a new or modified source could begin start-up operations. Thereafter, multiple-year Certificates-to-Operate (CO) were issued after inspection and approval of the source. In 1996, NYS DEC began implementation of the federal permitting program for major stationary sources called "Title V" (in reference to Title V of the Clean Air Act). This permitting program is designed to include all of the emission sources and pollution control requirements for an entire facility in a single operating permit.

NYS DOH researchers evaluated the emissions databases using the screening criteria, and the outcome of that evaluation can be found in *Appendix IV-1.* The data were considered complete for the CMP area and facility coordinates were obtained. The temporal component of the data was considered moderately relevant since the timeframe slightly predates the cancer incidence data. However, because the data are emissions information, they are limited for characterizing exposure. Because these data reflect emissions information only, the extent of the exposed population was considered unknown, and the measures of exposure were considered of moderate quality. NYS DOH researchers evaluated whether these databases may have been included in another data set. Some of the pollutant release information in TRI and NEI was included in US EPA modeled data described below. In addition, an evaluation of the PC/CO data shows that many of the facilities in this database were included in the modeling data described below.

NYS DOH researchers concluded that the information in these emissions databases would be of limited use in evaluating exposures in the CMP area .

3) ***Modeled Data***

For CEP and NATA, US EPA estimated chemical-specific air concentrations for small geographic areas known as census tracts across the continental 48-state region. For CEP, US EPA estimated outdoor air concentrations for 148 hazardous air pollutants based on 1990 emissions data. For NATA, US EPA estimated outdoor air concentrations of 32 hazardous air pollutants and diesel particulates based on 1996 emissions data. US EPA

obtained emissions data from databases such as the Toxic Release Inventory and the National Emissions Inventory to compile 1990 and 1996 emissions. Additionally, for the NATA emissions inventory, US EPA obtained facility emissions information from state source inventories and therefore the quality of the NATA estimates is considered better than CEP. US EPA developed outdoor air concentrations using a complex computer program (called a dispersion model) that merges the emissions data with meteorological data, such as wind speed and wind direction, to estimate pollutant concentrations in air. This model accounted for emissions from large industrial facilities, such as power plants and manufacturing facilities, and smaller facilities, such as dry cleaners and gas stations. US EPA included emissions also from mobile sources such as motor vehicles, trains, planes, and boats, and emissions from farming and construction equipment in the modeling estimates.

To examine the accuracy of the CEP and NATA estimates, US EPA researchers compared the estimated concentrations for some chemicals with monitored (measured) concentrations from various locations throughout the country (none of which are in the CMP area). US EPA concluded that, for the specific pollutants evaluated, the modeled results showed fairly good agreement with the measured results. This analysis was somewhat more rigorous for NATA. Therefore, NYS DOH researchers have more confidence in the NATA estimates than the CEP estimates.

In applying the screening criteria, NYS DOH researchers found many aspects of these two databases better than the others for evaluating exposures. Details of the application of the screening criteria for CEP and NATA can be found in *Appendix IV-1.* These databases provide modeled estimates for the entire region so the information for the CMP area was considered complete and that the exposure information was considered widespread. By modeling the concentrations of chemicals in air, a medium to which everyone is exposed, the inhalation exposure pathway would be considered complete for the residents. Also, the modeled estimates in both databases include a comprehensive inventory of sources (which was of higher quality in NATA than in CEP).

However, there are shortcomings of using these data sets. First, the modeled estimates are only for 1990 and 1996, a time period that is nearly concurrent with cancer incidence data. Also, because the data are modeled estimates of chemicals in outdoor air, they are considered indirect measures of exposure.

4) ***Conclusions***

Among all the air databases evaluated, NYS DOH researchers chose to focus their efforts on US EPA's CEP, NATA and Air Quality System databases because they provide the most useful information for characterizing potential exposures in the CMP area. Additionally, many of the concerns raised by the residents about emissions from specific sources would be included in these data sets.

There are certain limitations of using these data for this investigation. The US EPA air monitoring data provide data on pollutants that are not considered risk factors for breast cancer. The CEP and NATA data represent modeled estimates of outdoor air concentrations of some contaminants that may be breast cancer risk factors, but that may or may not represent the contaminant concentrations to which people may actually have been exposed. We have no knowledge of actual release patterns from the sources modeled, and are not able to verify that the emissions

information used in the models reflect actual emissions from sources. The modeled estimates represent only outdoor concentrations over one-year time periods. Because the estimates are annual averages, possible potential acute exposures would not be reflected in those estimates. Additionally, these estimates were produced for census tracts and would not reflect the varying exposures that people would have when they move about during the day between home, work or school. The CEP and NATA data also represent concentration estimates for 1990 and 1996, respectively, and do not necessarily reflect possible exposures for other years.

e. Evaluation of Exposure to Air Contaminants in the CMP Area

NYS DOH researchers used the CEP and NATA data to evaluate whether exposure to air contaminants in the CMP area was higher than exposure in other comparison areas of New York State. The comparison areas used were Suffolk County without the CMP region, New York State, and New York State exclusive of New York City. This evaluation was performed on a chemical-specific basis by calculating the ratio of the concentration estimate for the CMP area to the concentration estimate for each comparison area. A ratio greater than one indicates the modeled concentration was higher in the CMP area than in the comparison area. For most analytes, all three ratios were below or within 10 percent of one, suggesting little or no increased exposure in the CMP area. An increase in exposure to a trace contaminant in outdoor air of less than 10% is unlikely to elicit an increase in adverse health effects that can be detected epidemiologically. Therefore, DOH researchers conservatively chose to evaluate contaminants for which a comparison ratio exceeded 1.1 in any comparison area. Twenty-seven contaminants in the CEP data set and four contaminants in the NATA data set have ratios that exceed 1.1 for at least one comparison area, suggesting an elevated exposure in the CMP region. *Table 16* shows the concentration estimates and the comparison ratios for all the pollutants included in the CEP data set. *Table 17* shows the same information for NATA. The absence of a comparison ratio indicates no modeled estimates were provided in the CMP area or one of the comparison areas. Concentration estimates expressed with a "–" indicate that no modeled estimates were provided in the data for that region.

Table 16. CEP comparison ratios and concentration estimate

CEP (1990)	Comparison ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Ethylene thiourea	2.55	18.43	33.88	$8.95x10^{-8}$	$3.51x10^{-8}$	$4.86x10^{-9}$	$2.64x10^{-9}$
Acrylic acid	1.97	10.96	6.78	$1.02x10^{-3}$	$5.17x10^{-4}$	$9.27x10^{-5}$	$1.50x10^{-4}$
Hexane	4.60	7.66	3.85	4.64	1.01	0.606	1.20
Methyl tert-butyl ether (MTBE)	3.44	4.84	2.39	0.871	0.254	0.180	0.364
Propionaldehyde	2.55	4.33	1.91	0.534	0.209	0.123	0.280
1,2,4-Trichlorobenzene	0.68	3.14	4.55	$8.83x10^{-5}$	$1.30x10^{-4}$	$2.82x10^{-5}$	$1.94x10^{-5}$
Methylene diphenyl diisocyanate	1.17	2.90	0.61	$2.51x10^{-4}$	$2.14x10^{-4}$	$8.63x10^{-5}$	$4.13x10^{-4}$
Acetaldehyde	1.96	2.89	1.18	1.54	0.787	0.533	1.31
Hydrofluoric acid	1.51	2.86	2.81	$6.73x10^{-2}$	$4.46x10^{-2}$	$2.36x10^{-2}$	$2.40x10^{-2}$
Ethylbenzene	1.90	2.22	1.21	0.753	0.397	0.340	0.623
2,2,4-Trimethylpentane	1.95	2.16	1.14	1.59	0.814	0.736	1.39
Methyl ethyl ketone	1.52	2.10	0.94	1.44	0.947	0.683	1.53
Benzene	1.71	1.88	1.09	3.52	2.06	1.87	3.22
Dimethyl phthalate	0.74	1.80	3.21	$4.02x10^{-4}$	$5.43x10^{-4}$	$2.23x10^{-4}$	$1.25x10^{-4}$
Toluene	1.53	1.74	0.83	5.74	3.75	3.29	6.90
Beryllium	1.27	1.67	0.84	$2.51x10^{-5}$	$1.98x10^{-5}$	$1.50x10^{-5}$	$2.99x10^{-5}$
Xylene	1.32	1.54	0.78	3.72	2.82	2.41	4.75
Diethanolamine	5.24	0.08	0.06	$2.13x10^{-6}$	$4.06x10^{-7}$	$2.68x10^{-5}$	$3.73x10^{-5}$
Aniline	0.61	1.01	1.53	$1.67x10^{-3}$	$2.74x10^{-3}$	$1.66x10^{-3}$	$1.09x10^{-3}$
Trichloroethene	0.86	1.12	0.80	0.416	0.485	0.372	0.516
1,1,1-trichloroethane	0.90	1.11	0.70	2.92	3.23	2.62	4.18
Hydrochloric acid	1.18	1.35	0.65	0.939	0.795	0.696	1.44
Arsenic	1.07	1.18	0.46	$3.32 x10^{-4}$	$3.10 x10^{-4}$	$2.82 x10^{-4}$	$7.17 x10^{-4}$
1,3-Dichloropropene	0.93	1.12	0.46	$5.46 x10^{-2}$	$5.89 x10^{-2}$	$4.86 x10^{-2}$	0.119
Glycol ethers	0.85	1.10	0.45	0.612	0.722	0.554	1.37
Acrylamide	0.97	1.37	0.41	$4.14 x10^{-8}$	$4.27 x10^{-8}$	$3.03 x10^{-8}$	$1.00 x10^{-7}$
1,1-Dichloroethene	1.33	0.16	0.24	$1.13 x10^{-5}$	$8.50 x10^{-6}$	$6.85 x10^{-5}$	$4.68 x10^{-5}$
Air contaminants above have been given priority for integration							
Phosgene	1.03	1.08	1.02	$7.05x10^{-2}$	$6.84x10^{-2}$	$6.56x10^{-2}$	$6.91x10^{-2}$

**These values are expressed in scientific notation: a number such as 1.02 x 10^{-3} is equivalent to 0.00102*

CEP (1990)	Comparison ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Chlordane	1.00	1.00	1.00	$9.89x10^{-6}$	$9.89x10^{-6}$	$9.89x10^{-6}$	$9.89x10^{-6}$
Hexachlorobenzene	1.00	1.00	1.00	$9.32x10^{-5}$	$9.32x10^{-5}$	$9.32x10^{-5}$	$9.32x10^{-5}$
1,2-Dibromoethane	1.00	1.00	1.00	$7.69x10^{-3}$	$7.69x10^{-3}$	$7.69x10^{-3}$	$7.69x10^{-3}$
Methyl chloride	1.00	1.00	1.00	1.25	1.25	1.25	1.25
Methyl iodide	1.00	1.00	1.00	$1.16x10^{-2}$	$1.16x10^{-2}$	$1.16x10^{-2}$	$1.16x10^{-2}$
Carbonyl sulfide	1.00	1.00	1.00	1.23	1.23	1.23	1.23
Lindane	1.00	1.00	1.00	$2.50x10^{-4}$	$2.50x10^{-4}$	$2.51x10^{-4}$	$2.50x10^{-4}$
Hexachloroethane	1.00	1.00	1.00	$4.84x10^{-3}$	$4.84x10^{-3}$	$4.85x10^{-3}$	$4.85x10^{-3}$
Carbon tetrachloride	1.00	1.00	0.99	0.884	0.885	0.885	0.890
Hexachlorobutadiene	1.00	0.98	0.99	$1.81x10^{-3}$	$1.81x10^{-3}$	$1.84x10^{-3}$	$1.83x10^{-3}$
Methyl bromide	1.00	0.94	0.96	$3.90x10^{-2}$	$3.90x10^{-2}$	$4.15x10^{-2}$	$4.06x10^{-2}$
Polychlorinated biphenyls	1.00	0.93	0.96	$3.78x10^{-4}$	$3.78x10^{-4}$	$4.05x10^{-4}$	$3.93x10^{-4}$
Chloroform	1.00	0.98	0.94	$8.48x10^{-2}$	$8.52x10^{-2}$	$8.62x10^{-2}$	$9.03x10^{-2}$
Bis(2-ethylhexyl)phthalate	1.01	1.01	0.90	$1.50x10^{-3}$	$1.48x10^{-3}$	$1.48x10^{-3}$	$1.66x10^{-3}$
Bromoform	1.00	0.96	0.84	$2.10x10^{-2}$	$2.11x10^{-2}$	$2.19x10^{-2}$	$2.50x10^{-2}$
1,2-Dichloroethane	0.97	1.01	0.79	$8.54x10^{-2}$	$8.82x10^{-2}$	$8.46x10^{-2}$	0.108
Methylene chloride	1.01	0.84	0.77	0.430	0.425	0.515	0.561
Cumene	1.10	1.08	0.75	$2.59x10^{-2}$	$2.35x10^{-2}$	$2.39x10^{-2}$	$3.43x10^{-2}$
Carbon disulfide	1.00	0.59	0.69	$5.24x10^{-2}$	$5.21x10^{-2}$	$8.87x10^{-2}$	$7.54x10^{-2}$
Mercury Compounds	1.02	1.03	0.69	$2.01x10^{-3}$	$1.97x10^{-3}$	$1.95x10^{-3}$	$2.92x10^{-3}$
Dibutylphthalate	0.92	0.95	0.63	$1.70x10^{-3}$	$1.84x10^{-3}$	$1.80x10^{-3}$	$2.70x10^{-3}$
Biphenyl	0.69	0.59	0.57	$9.02x10^{-5}$	$1.31x10^{-4}$	$1.52x10^{-4}$	$1.58x10^{-4}$
Cresol	0.93	0.95	0.57	$6.74x10^{-2}$	$7.26 x10^{-2}$	$7.14 x10^{-2}$	0.119
Phenol	0.92	0.82	0.57	$9.64 x10^{-2}$	0.105	0.117	0.170
Ethyl chloride	0.89	1.05	0.53	$1.28 x10^{-2}$	$1.43 x10^{-2}$	$1.21 x10^{-2}$	$2.43 x10^{-2}$
Acrylonitrile	0.90	1.05	0.53	$9.32 x10^{-3}$	$1.04 x10^{-2}$	$8.86 x10^{-3}$	$1.77 x10^{-2}$
Chloroprene	0.86	1.00	0.52	$1.31 x10^{-2}$	$1.52 x10^{-2}$	$1.31 x10^{-2}$	$2.51 x10^{-2}$
Tetrachloroethene (PERC)	0.86	0.92	0.52	0.469	0.543	0.508	0.900
Vinyl chloride	0.90	1.01	0.51	$1.68 x10^{-2}$	$1.85 x10^{-2}$	$1.66 x10^{-2}$	$3.26 x10^{-2}$
Phthalic anhydride	0.90	1.01	0.51	$9.08 x10^{-3}$	$1.01 x10^{-2}$	$8.98 x10^{-3}$	$1.77 x10^{-2}$
1,1,2-Trichloroethane	0.91	0.45	0.51	$2.41 x10^{-4}$	$2.64 x10^{-4}$	$5.40 x10^{-4}$	$4.72 x10^{-4}$

CEP (1990)	Comparison ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Ethyl acrylate	0.88	1.01	0.50	4.89×10^{-3}	5.58×10^{-3}	4.85×10^{-3}	9.73×10^{-3}
Maleic anhydride	0.87	0.92	0.50	2.61×10^{-3}	3.01×10^{-3}	2.85×10^{-3}	5.23×10^{-3}
Chlorobenzene	0.91	1.08	0.49	5.82×10^{-2}	6.36×10^{-2}	5.41×10^{-2}	0.118
Vinyl acetate	0.87	1.00	0.48	2.23×10^{-2}	2.56×10^{-2}	2.24×10^{-2}	4.63×10^{-2}
Naphthalene	0.95	0.98	0.48	0.124	0.131	0.127	0.258
Methanol	0.95	1.01	0.46	0.966	1.01	0.961	2.09
Cyanide compounds	0.87	0.86	0.46	6.47×10^{-2}	7.40×10^{-2}	7.50×10^{-2}	0.142
Propylene oxide	0.57	0.39	0.46	4.28×10^{-4}	7.53×10^{-4}	1.10×10^{-3}	9.38×10^{-4}
1,4-Dichlorobenzene	0.87	1.05	0.44	9.49×10^{-2}	0.109	9.02×10^{-2}	0.215
Polycyclic organic matter	0.97	1.04	0.43	0.281	0.288	0.271	0.648
Polychlorinated dibenzo-p-dioxins/polychlorinated dibenzofurans	0.89	0.95	0.43	2.31×10^{-8}	2.59×10^{-8}	2.42×10^{-8}	5.37×10^{-8}
Ethylene glycol	0.86	1.06	0.43	0.397	0.461	0.374	0.926
Methyl isobutyl ketone	0.84	0.76	0.41	0.147	0.174	0.194	0.358
Formaldehyde	0.91	0.92	0.39	1.01	1.11	1.09	2.57
Chromium	0.98	0.76	0.39	5.09×10^{-4}	5.18×10^{-4}	6.68×10^{-4}	1.32×10^{-3}
Nickel	1.03	1.02	0.38	8.48×10^{-3}	8.26×10^{-3}	8.34×10^{-3}	2.25×10^{-2}
Styrene	0.75	0.66	0.37	3.91×10^{-2}	5.24×10^{-2}	5.93×10^{-2}	0.107
Methyl methacrylate	0.43	0.44	0.34	1.68×10^{-3}	3.90×10^{-3}	3.80×10^{-3}	5.00×10^{-3}
Selenium	0.88	0.59	0.34	2.66×10^{-4}	3.03×10^{-4}	4.47×10^{-4}	7.90×10^{-4}
Ethylene oxide	0.92	0.40	0.32	1.87×10^{-3}	2.04×10^{-3}	4.66×10^{-3}	5.94×10^{-3}
Acrolein	0.88	1.00	0.31	0.153	0.174	0.153	0.492
1,3-Butadiene	0.78	0.69	0.30	8.53×10^{-2}	0.110	0.124	0.284
Manganese	0.93	0.82	0.30	3.02×10^{-3}	3.25×10^{-3}	3.69×10^{-3}	1.01×10^{-2}
Antimony	0.95	0.98	0.29	3.74×10^{-4}	3.95×10^{-4}	3.82×10^{-4}	1.28×10^{-3}
Cobalt	0.73	0.72	0.29	1.23×10^{-4}	1.69×10^{-4}	1.71×10^{-4}	4.28×10^{-4}
Lead	0.87	0.66	0.29	2.57×10^{-3}	2.94×10^{-3}	3.88×10^{-3}	8.99×10^{-3}
Cadmium	0.98	0.83	0.28	1.37×10^{-4}	1.41×10^{-4}	1.65×10^{-4}	4.92×10^{-4}
Dimethyl formamide	0.20	0.09	0.14	1.41×10^{-5}	7.11×10^{-5}	1.49×10^{-4}	1.01×10^{-4}
1,2-Dichloropropane	0.89	0.08	0.13	8.04×10^{-4}	9.00×10^{-4}	9.57×10^{-3}	6.28×10^{-3}
Nitrobenzene	1.06	0.48	0.11	9.92×10^{-6}	9.36×10^{-6}	2.06×10^{-5}	8.73×10^{-5}
Hydroquinone	0.22	0.01	0.02	1.15×10^{-6}	5.30×10^{-6}	1.23×10^{-4}	7.09×10^{-5}

CEP (1990)	Comparison ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Hydrazine	0.97	0.01	0.00	2.07×10^{-8}	2.13×10^{-8}	4.12×10^{-6}	1.46×10^{-5}
Acetonitrile	0.14	0.00	0.00	1.50×10^{-7}	1.09×10^{-6}	2.10×10^{-3}	1.94×10^{-3}
1,4-Dioxane				-	2.12×10^{-5}	1.09×10^{-3}	6.01×10^{-4}
N,N-diethyl/dimethylaniline				-	8.54×10^{-9}	9.81×10^{-4}	5.67×10^{-4}
2-Nitropropane				-	4.06×10^{-7}	5.30×10^{-5}	2.23×10^{-4}
Propoxur				-	7.96×10^{-7}	2.19×10^{-6}	1.90×10^{-4}
Diethyl sulfate				-	1.27×10^{-8}	8.20×10^{-6}	5.49×10^{-5}
1,2-Epoxybutane				-	1.08×10^{-5}	5.80×10^{-5}	3.43×10^{-5}
Chloroacetic acid				-	4.25×10^{-9}	1.55×10^{-5}	1.29×10^{-5}
Captan				-	9.39×10^{-10}	1.13×10^{-7}	6.15×10^{-8}
Epichlorohydrin				-	-	1.87×10^{-5}	3.83×10^{-4}
1,1,2,2-Tetrachloroethane				-	-	1.82×10^{-4}	1.00×10^{-4}
2,4-Toluene diisocyanate				-	-	1.34×10^{-5}	9.64×10^{-5}
Hexachlorocyclopentadiene				-	-	1.13×10^{-4}	6.12×10^{-5}
Benzyl chloride				-	-	6.07×10^{-7}	3.91×10^{-5}
Allyl chloride				-	-	4.01×10^{-5}	3.28×10^{-5}
Dimethyl sulfate				-	-	1.08×10^{-5}	2.40×10^{-5}
Benzotrichloride				-	-	3.41×10^{-5}	1.86×10^{-5}
Urethane				-	-	3.10×10^{-6}	1.19×10^{-5}
4,4'-Methylenedianiline				-	-	1.34×10^{-5}	7.27×10^{-6}
O-toluidine				-	-	1.24×10^{-5}	6.76×10^{-6}
Styrene oxide				-	-	2.14×10^{-7}	6.31×10^{-6}
2,4-Dinitrophenol				-	-	1.09×10^{-5}	5.95×10^{-6}
1,2-Propylenimine				-	-	6.66×10^{-7}	3.59×10^{-6}
Carbaryl				-	-	6.07×10^{-7}	2.76×10^{-6}
4,4'-Methylene bis(2-chloroaniline)				-	-	1.65×10^{-6}	8.95×10^{-7}
Quinoline				-	-	1.04×10^{-6}	5.64×10^{-7}
Catechol				-	-	1.00×10^{-6}	5.44×10^{-7}
Methyl hydrazine				-	-	2.97×10^{-7}	1.65×10^{-7}
Trifluralin				-	-	2.07×10^{-7}	1.34×10^{-7}
3,3'-Dichlorobenzidine				-	-	4.82×10^{-8}	1.33×10^{-7}

CEP (1990)	Comparison ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Acetophenone				-	-	1.27×10^{-7}	6.90×10^{-8}
2,4-Diaminotoluene				-	-	6.47×10^{-8}	4.64×10^{-8}
3,3'-Dimethoxybenzidine				-	-	4.50×10^{-9}	2.48×10^{-8}
Heptachlor				-	-	3.17×10^{-9}	1.72×10^{-9}
4-nitrophenol				-	-	5.82×10^{-10}	3.17×10^{-10}
P-phenylenediamine				-	-	1.46×10^{-10}	7.93×10^{-11}
Pentachlorophenol				-	-	1.55×10^{-11}	8.44×10^{-12}
Quinone				-	-	3.41×10^{-12}	1.85×10^{-12}
Dichloroethyl ether				-	-	-	6.83×10^{-7}
Anisidine				-	-	-	-
Parathion				-	-	-	-
Pentachloronitrobenzene				-	-	-	-
2,4,6-Trichlorophenol				-	-	-	-
Vinyl bromide				-	-	-	-
Acetamide				-	-	-	-
Bis(chloromethyl) ether				-	-	-	-
Calcium cyanamide				-	-	-	-
Chloramben				-	-	-	-
Chloromethyl methyl ether				-	-	-	-
2,4-Dichlorophenoxyacetic acid				-	-	-	-
Dichlorvos				-	-	-	-
1,1-Dimethyl hydrazine				-	-	-	-
4,6-Dinitro-o-cresol				-	-	-	-
2,4-Dinitrotoluene				-	-	-	-
1,1-Dichloroethane				-	-	-	-
Methoxychlor				-	-	-	-
Methyl isocyanate				-	-	-	-

Table 17. NATA comparison ratios and concentration estimate.

NATA (1996)	Comparison Ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
1,2-Dibromoethane	1.04	4.05	5.37	5.44×10^{-5}	5.22×10^{-5}	1.34×10^{-5}	1.01×10^{-5}
Ethylene oxide	1.50	0.52	0.20	3.99×10^{-4}	2.66×10^{-4}	7.66×10^{-4}	2.03×10^{-3}
Diesel particulate matter	0.88	1.32	0.44	2.37	2.69	1.80	5.39
Cadmium	1.19	0.40	0.30	1.21×10^{-4}	1.02×10^{-4}	3.02×10^{-4}	4.08×10^{-4}
Air contaminants above, have been given priority for integration							
Carbon tetrachloride	1.00	1.00	1.00	0.880	0.881	0.881	0.882
1,1,2,2-Tetrachloroethane	0.81	1.09	0.90	1.76×10^{-3}	2.18×10^{-3}	1.62×10^{-3}	1.97×10^{-3}
Chloroform	0.98	0.98	0.88	8.57×10^{-2}	8.75×10^{-2}	8.73×10^{-2}	9.69×10^{-2}
Vinyl chloride	0.80	0.72	0.65	4.41×10^{-3}	5.52×10^{-3}	6.14×10^{-3}	6.75×10^{-3}
Acrylonitrile	0.80	0.51	0.54	3.32×10^{-3}	4.17×10^{-3}	6.57×10^{-3}	6.10×10^{-3}
1,2-Dichloropropane	0.41	0.43	0.54	2.53×10^{-4}	6.13×10^{-4}	5.85×10^{-4}	4.66×10^{-4}
1,2-Dichloroethane	1.06	0.40	0.52	5.90×10^{-4}	5.56×10^{-4}	1.49×10^{-3}	1.13×10^{-3}
Benzene	0.90	0.92	0.51	1.09	1.21	1.18	2.14
Tetrachloroethene	0.85	0.87	0.48	0.236	0.279	0.272	0.492
Methylene chloride	0.92	0.87	0.48	0.415	0.451	0.475	0.872
Trichloroethene	0.83	0.76	0.46	0.122	0.148	0.161	0.264
Quinoline	0.74	0.48	0.45	8.10×10^{-7}	1.09×10^{-6}	1.68×10^{-6}	1.81×10^{-6}
1,3-Dichloropropene	0.81	0.91	0.35	8.86×10^{-2}	0.110	9.74×10^{-2}	0.255
Polycyclic organic matter	0.78	0.81	0.34	7.07×10^{-2}	9.12×10^{-2}	8.76×10^{-2}	0.209
Arsenic	0.88	0.64	0.34	5.78×10^{-5}	6.59×10^{-5}	8.96×10^{-5}	1.72×10^{-4}
Beryllium	0.79	0.79	0.33	1.09×10^{-5}	1.38×10^{-5}	1.39×10^{-5}	3.32×10^{-5}
Acetaldehyde	0.84	1.00	0.30	0.648	0.769	0.647	2.14
Manganese	0.73	0.59	0.29	1.34×10^{-3}	1.82×10^{-3}	2.25×10^{-3}	4.54×10^{-3}
Nickel	0.93	0.66	0.29	1.12×10^{-3}	1.20×10^{-3}	1.68×10^{-3}	3.80×10^{-3}
Acrolein	0.77	0.82	0.28	8.62×10^{-2}	0.113	0.105	0.303
1,3-Butadiene	0.81	0.59	0.25	2.93×10^{-2}	3.61×10^{-2}	4.97×10^{-2}	0.117
Chromium	0.74	0.44	0.25	8.53×10^{-4}	1.16×10^{-3}	1.96×10^{-3}	3.47×10^{-3}
Formaldehyde	0.77	0.88	0.24	0.739	0.957	0.836	3.06
Mercury	0.55	0.61	0.17	3.98×10^{-4}	7.24×10^{-4}	6.57×10^{-4}	2.33×10^{-3}
Lead	0.37	0.27	0.10	1.70×10^{-3}	4.54×10^{-3}	6.31×10^{-3}	1.68×10^{-2}

** These values are expressed in scientific notation: a number such as 1.02 x 10^{-3} is equivalent to 0.00102*

NATA (1996)	Comparison Ratios			Concentration estimate microgram per cubic meter (mcg/m^3)*			
	CMP/ Suffolk	CMP/NYS w/o NYC	CMP/ NYS	CMP	Suffolk	NYS w/o NYC	NYS
Hydrazine	0.71	0.04	0.01	1.60×10^{-7}	2.27×10^{-7}	3.81×10^{-6}	1.09×10^{-5}
Hexachlorobenzene	0.19	0.03	0.01	1.19×10^{-8}	6.15×10^{-8}	3.42×10^{-7}	1.84×10^{-6}
Coke oven emissions				-	-	1.39×10^{-3}	7.56×10^{-4}
Polychlorinated biphenyls				-	-	4.59×10^{-8}	2.61×10^{-8}

In addition to the CEP and NATA contaminants listed above, the air pollutant ozone will also be further evaluated in *Chapter V. Integration* because ozone levels exceed the NAAQS.

f. Contaminants Considered for Integration

Twenty-seven contaminants from the CEP data set and the four contaminants from the NATA data set have ratios that exceed 1.1 for any comparison area. An error in the modeling results was found for six contaminants in CEP and these contaminants will not be evaluated further. An explanation of that error is provided below. The remaining contaminants, a total of 26 including ozone, were evaluated in terms of potential risk factors for breast cancer and non-cancer health effects by integrating the concentration estimates provided in CEP and NATA and air monitoring data with the toxicological data collected as part of the toxicological evaluation. This information is presented in *Chapter V. Integration*.

A number of contaminants from the CEP data set that were initially selected for integration, will not be evaluated further because of an error in the modeling results. This error is linked to the data for a gasoline terminal in the CMP region (Northville Industries—East Setauket location) and the contaminants associated with the error are common constituents of gasoline. Using benzene as an example, US EPA's modeling of benzene emissions from this facility are based on an emission rate of 472,000 pounds of benzene per year. NYS DEC records indicate that 223,000 pounds of gasoline were emitted from the facility in 1990. Since benzene is a constituent of gasoline, benzene emissions from the facility should be much smaller than total gasoline emissions. Assuming US EPA incorrectly overestimated benzene emissions from the facility, the CEP concentration estimates for benzene in the CMP area also would be overestimated. Likewise, the CEP concentration estimates for the other gasoline constituents (including hexane, ethylbenzene, toluene, xylene and 2,2,4-trimethylpentane) also would be overestimated. The NATA modeling results for benzene and other gasoline constituents for the CMP area looked much different, therefore only the CEP gasoline constituents were evaluated.

Using emissions information provided to NYS DOH researchers by NYS DEC, emission rates for benzene and the other gasoline constituents were calculated and that information is presented in *Table 18*. This table lists the contaminants and a comparison of the emission rates as found in the CEP data and calculated emission rates

based on information from NYS DEC for the facility. Had the correct emission rates been used for the gasoline constituents in CEP, it is unlikely the modeled concentrations of these contaminants would have had a comparison ratio greater than 1.1 in any of the comparison areas. Therefore, these six contaminants were eliminated from further analysis.

Table 18. Gasoline related contaminant emissions, CEP compared to NYS DEC

Contaminant	CEP reported emissions (pounds/year)	NYS DEC calculated emissions (pounds/year)
Benzene	472,000	2,000
Hexane	1,124,200	3,600
Ethylbenzene	108,770	220
Toluene	700,000	2,900
Xylene	350,000	1,100
2,2,4-trimethylpentane	238,000	1,800

Although NYS DOH researchers have concerns about the accuracy of the modeled air concentrations for diesel particulate matter, it will be considered for integration. Concern arises from the fact that these estimates were based on 1996 data, the first year that US EPA included diesel particulate matter in the National Air Toxics Assessment. They were based on an inventory of diesel sources (e.g., cars, trucks, trains, planes, and farming and construction equipment) and approximation techniques to estimate the contribution of their emissions to air concentrations of diesel particulate matter. Many simplifying assumptions were made to make these estimations and these assumptions reduce confidence in the modeled air concentrations.

4. Pesticide Use

a. Introduction

Residents who attended the public availability meeting expressed concerns about past and current use and possible exposure to pesticides. The Initial Environmental Inventory identified two data sources that could provide information about the use of pesticides and possible human exposure to pesticides in the CMP area. Those sources of data are New York State's Pesticide Sales and Use Reporting Database and the Suffolk County pesticide groundwater and drinking water monitoring database. Both of these data sets were selected for further evaluation (as indicated in *Appendix IV-1).*

People can be exposed to pesticides both during and after their application. Pesticides have been used for a long time. Organochlorine chemicals such as DDT became available after World War II. Since then, newer pesticides have become available and some of the older ones have been banned or phased out. Many of the older pesticides were banned because of environmental or health-related concerns.

Little quantitative information is available about historical pesticide use in the CMP area or elsewhere in New York State. NYS DOH researchers identified New York State's Pesticide Sales and Use Reporting Database as the most comprehensive source of information for evaluating pesticide use. An evaluation of pesticide application patterns based on the commerical (certified, professional) applicator portion of the database is summarized in this section. Suffolk County's data on the presence of pesticides and pesticide breakdown products in private and public drinking water supplies are discussed in *Section IV E-6. Water Supply.*

Suffolk County has a long-standing vector control program that sometimes uses pesticides for insect control. The County's use of pesticides for this program is reported to the New York State Pesticide Sales and Use Reporting Database. In addition, Suffolk County provides information about this program on its web site at http://www.co.suffolk.ny.us/ (search for "Pesticide Application Notification"). Health information about spraying for mosquitoes can also be obtained by visiting the State Health Department's web site at http://www.health.state.ny.us/nysdoh/westnile/index.htm or calling 800-458-1158.

b. Sources of Pesticide Data

The most comprehensive information on pesticide sales and use in New York is contained in New York State 's Pesticide Sales and Use Reporting Database established under New York's Pesticide Reporting Law, enacted in 1996. The law requires that commerical pesticide applicators to report pesticide use for each pesticide application. Records include the EPA registration number for the product, the product name, the quantity of pesticide used, the date applied and the location of application by address. The law also requires those who sell restricted use pesticides to report each sale of a restricted use pesticide or a general use pesticide used in agricultural crop

production. Private pesticide applicators, those applying restricted use pesticides for the purpose of producing agricultural commodities on their own or their employer's property, must maintain records of the purchase and application of restricted use pesticides and make those records available for inspection, but do not need to report the data. Those involved in manufacturing or importing restricted use pesticides must report sales of restricted use pesticides.

DOH researchers used the commercial application part of the database because it is the only set of data reported to the state that contains application location information. At the time this report was prepared finalized data were available for the years 1997 through 2001. Because the use of pesticides such as chlordane, heptachlor, aldrin, dieldrin, and DDT ended prior to 1997, there are no records available for applications of these pesticides in this database. DOH researchers used ZIP Code level data to evaluate regional differences in pesticide application rates.

Pesticide sales and use data are reported to NYS DEC, however, the database is managed by Cornell University and can be accessed at http://pmep.cce.cornell.edu/psur/ (January 2005). Data summaries also are available at this website. Additional information on New York State's Pesticide Reporting Law can be found at http://www.dec.state.ny.us/website/dshm/prl/weblaw.htm (January 2005).

c. Evaluation of Data Quality

The application data in New York State's Pesticide Sales and Use Reporting Database is provided by the commercial applicator who must properly identify time, place, and quantity of pesticide applied. Applicators report the quantity of pesticides applied in either pounds or gallons, depending on the form in which it is purchased. The quality of the reporting of the data has improved over the years as the reporting system has been improved and as applicators and NYS DEC gained experience. Database managers also have worked with the data to correct some reporting or data entry errors.

Appendix IV-1 summarizes NYS DOH researchers' assessment of the New York State Pesticide Sales and Use Reporting Database to evaluate pesticide exposures in the CMP area. NYS DOH researchers concluded that this database is of limited use to estimate pesticide exposures from commercial pesticide applications in the CMP area. It provides no direct evidence of actual exposure to pesticides, but regional patterns of pesticide applications can be evaluated. The temporal relevance of these data also is poor for characterizing a relationship between pesticide use and breast cancer incidence in the CMP area because this data set contains no records prior to 1997. However, given the lack of any alternative data for characterizing pesticide exposures in the CMP area, and given that there may be a relationship between historical pesticide applications and those applied since 1997, researchers retained this database for further evaluation.

d. Characterization of Potential Elevated Exposures to Pesticides

NYS DOH researchers used the Pesticide Sales and Use Reporting Database to compare the total number of pounds and total number of gallons of reported commercial pesticide applications in Suffolk County to those

reported for other counties in New York State. In 2000, Suffolk County ranked first in the number of pounds of dry-pesticide commercial applications reported and second in the number of gallons of liquid-pesticide commercial applications reported among the 62 counties in New York State.

Table 19 shows the reported data of dry-pesticide commercial applications in New York State, Suffolk County and in the CMP area. These data show that a relatively small number of products labeled for use for lawn care and landscaping are responsible for about 90% of the total pounds of pesticide products applied in Suffolk County and the CMP area (see *Table 19*).

Table 19. Number and pounds reported of dry-pesticide commercial applications in New York State, Suffolk County and CMP area in 2000

	Number of pesticide products reported	Pounds of pesticide-containing products reported	Number of pesticide products accounting for 90% of all pounds	Number of products reported for lawn care and landscaping	Pounds reported for lawn care and landscaping	Percent of pounds reported for lawn care and landscaping
NYS	2,313	16,000,000	214	123	8,800,000	54
Suffolk County	811	2,400,000	65	59	2,100,000	86
CMP	277	220,000	19	19	200,000	90

Source: New York State's Pesticide Sales and Use Reporting Database

Most of the weight of lawn care and landscaping products consists of fertilizer and inert ingredients, with only a small percentage (often less than 1%) of the total weight attributable to the active pesticide ingredients. This means that the pounds of active ingredient actually applied is much less than the total pounds of product applied.

To compare pesticide use in the CMP area to use in New York State and Suffolk County, researchers calculated and compared "*rates*" of use, because, in total numbers of pounds or gallons, clearly more pesticides would be applied in the State and County than in the CMP area. ***Websters Dictionary*** defines *rate* as, *"a quantity measured with respect to another measured quantity:* e.g. pounds of pesticides applied per square mile, or $\frac{lb.\ pesticide}{mile^2}$

Choice of the rate to use for comparison can greatly affect the conclusions drawn from the data. If possible, researchers would select an exposure related comparison to evaluate pesticide use between different areas. Because of the difficulty in doing this for each pesticide and because data were not available for many types of comparisons (e.g. pesticide use per house, or per acre of lawn), total land area data were used to compare pesticide use, expressed as pesticide use in pounds or gallons per square mile.

The evaluation of the data shows that in the year 2000, the number of pounds per square mile of pesticide products applied was higher in the CMP area compared to the rest of Suffolk County and NYS (about 4280 pounds per square mile compared to about 2540 pounds per square mile and 162 pounds per square mile, respectively).

Commercial pesticide applications vary between the CMP area, Suffolk County and New York State for several reasons, among which may be type of land use (agricultural, vacant land or wooded land, suburban development) and property value. The heavier use of pesticide products in the CMP area, and, especially, pesticides used for lawn care and landscaping, may be because landscaped areas constitute a larger portion of the land area in the CMP area compared to other areas of Suffolk County. Areas further east in Suffolk County (e.g. Riverhead, Southhampton, East Hampton and Shelter Island) have more agricultural and undeveloped areas. These areas use less landscaping products on a per unit area basis (e.g. per square mile) than areas that have more landscaping. Therefore, the total rate of commercially applied pesticides in Suffolk County is made up of a combination of areas with higher rates to the west and lower rates to the east (see *Figure 4).*

Table 20 shows commercially reported data, for liquid-pesticide applications in gallons. The total gallons of commercially applied pesticide products, the number of products applied, and the quantities applied statewide, in Suffolk County and in CMP are shown. In the CMP area, seven products, six insecticides and one soil fumigant, account for 90% of the total gallons reported in the year 2000. One of those seven products, VAPAM HL soil fumigant, was applied for agricultural purposes exclusively in one ZIP Code in the CMP area. This product accounted for about 77% (33,000 gallons) of the total gallons applied in the CMP area in 2000. Four horticultural oil products account for 14% of the gallons applied. Horticultural oils are manufactured from petroleum distillates and contain nearly 99% light oil. The oils are typically sprayed on plants and trees while they are dormant to kill insects and other soft-bodied invertebrates by suffocation. The remaining two products were Sevin, an insecticide registered for a number of residential, commercial and agricultural uses, and Dragnet, an insecticide registered for use as a termiticide and for lawn care and landscaping. Sevin contains the active ingredient carbaryl and Dragnet contains the active ingredient permethrin.

Table 20. Number and gallons reported of liquid-pesticide commercial applications in New York State, Suffolk County and the CMP area in 2000

	Number of pesticide products reported	Gallons of pesticide products reported	Number of pesticide products accounting for 90% of all gallons reported
New York State	1,490	2,700,000	97
Suffolk County	438	340,000	32
CMP	106	42,000	7

Source: New York State's Pesticide Sales and Use Reporting Database

e. Characterization of Potentially Elevated Exposures to Specific Pesticides

Table 21. Summary of Suffolk County Pesticide Applications: 2000-2001

	Pounds of Active Ingredient		Loading (Pounds per square mile)		
Pesticide	**Suffolk County excluding CMP**	**CMP**	**Suffolk County excluding CMP**	**CMP**	**Ratio of pounds per square mile (CMP/Suffolk County excluding CMP)**
Termiticides[1]	378,700	8,200	440	160	0.4
Horticultural oil (gallons)	90,900	2,800	100	50	0.5
Carbaryl	84,600	7,500	100	140	1.5
2,4-D	35,000	4,000	40	80	1.9
Dicamba	3,300	400	4	8	2.0
Mecoprop	10,700	1,400	12	30	2.2

Source: New York State's Pesticide Sales and Use Reporting Database

[1] The termiticides evaluated are permethrin, cypermethrin, fenvalerate, and chlorpyrifos

1. 2,4-D, dicamba, & mecoprop

NYS DOH researchers evaluated data from the top 90% of commercially applied pesticide products reported for Suffolk County in the New York State Pesticide Sales and Use Reporting Database to determine which pesticide active ingredients [1] were represented and for how long they have been used. Recently developed pesticide active ingredients were screened out from further evaluation because they were not considered temporally relevant to breast cancer rates in the CMP area. Researchers also screened out active ingredients that were used in small quantities or only in a few localized areas. The active ingredient 2,4-dichlorophenoxyacetic acid (2,4-D) was selected for further evaluation. DOH researchers selected 2,4-D for several reasons: 2,4-D has been widely used in landscaping for several decades including the time period relevant to breast cancer, 2,4-D is commonly used throughout CMP as well as other regions of Suffolk County and NYS, and the total area treated with 2,4-D is large and is typically accessible by people.

Researchers evaluated data for 10 commercially applied products containing 2,4-D that were determined to be commonly used in the CMP area from 1997 through 2001. Many of the products that contain 2,4-D also contain the active ingredients 2-(4-chloro-2-methylphenoxy) propionic acid (mecoprop) and 3,6-dichloro-o-anisic acid (dicamba). These products are used to control broadleaf weeds in lawns. NYS DOH used New York State's

[1] An active ingredient is defined as the chemical or substance component of a pesticide product that can kill, repel, attract, mitigate or control a pest or that acts as a plant growth regulator, desiccant, or nitrogen stabilizer. The remainder of a formulated pesticide product consists of one or more inert ingredients (such as water, solvents, emulsifiers, surfactants, clay and propellants), which are there for reasons other than pesticidal activity.

Pesticide Sales and Use Reporting Database for the years 1997-2001 for products containing these three pesticides.

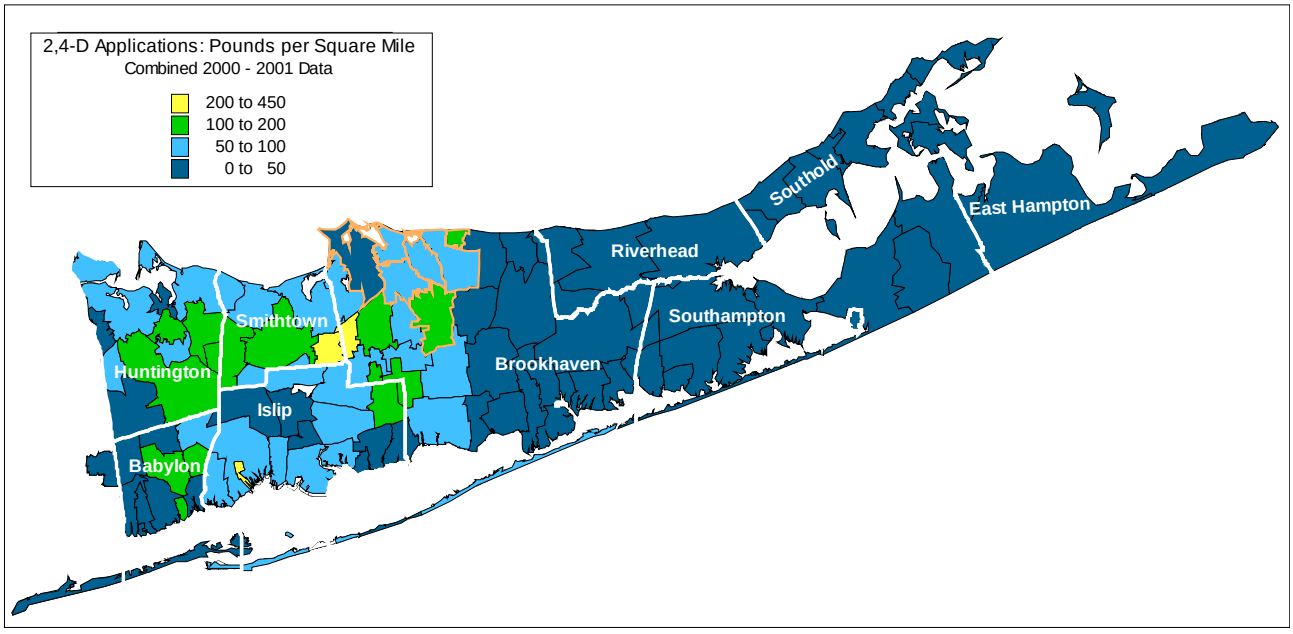
A preliminary evaluation of the data showed that in the year 2000, the amounts of 2,4-D, mecoprop and dicamba commercially applied per square mile in CMP was higher than New York State and the rest of Suffolk County. These data do not provide direct information about exposure to any of these pesticides.

As a follow-up to the preliminary evaluation state health researchers investigated reporting errors in the Pesticide Sales and Use Reporting Database that affected the preliminary estimates of the quantities of 2,4-D, dicamba, and mecoprop applied in Suffolk County for the years 2000 and 2001. The errors were associated with the manner in which pesticide products were reported. A number of liquid products were reported as both solid and liquid. Once the reporting issue was addressed, the comparison was repeated. Although the difference was reduced, the amounts of 2,4-D, mecoprop and dicamba professionally applied per square mile in CMP remained higher than New York State and the rest of Suffolk Count (*Table 21)*.

Application rates of 2,4-D across Suffolk County by ZIP code are illustrated in *Figure 4.* The figure indicates that the lower application rates of 2,4-D in the eastern part of Suffolk County reduce the overall application rates for the whole county. Mecoprop and dicamba follow the same pattern. Application rates of these pesticides in CMP appear to be similar to other areas of western Suffolk County.

Based on the comparison of the CMP area with the rest of Suffolk County, 2,4-D is evaluated in *Chapter V. Integration*. Furthermore, because mecoprop and dicamba often are applied with 2,4,D and are similarly elevated compared to the rest of Suffolk County *(Table 21),* they also are evaluated in Chapter V.

Figure 4. Map of Suffolk County depicting application rates of 2,4-D in Suffolk County Towns and ZIP Codes for the years 2000 & 2001.



2. Termiticides

Researchers reviewed data from the New York State Pesticide Sales and Use Reporting Database to evaluate whether the reported professional applications of pesticides used for termite control are greater in the CMP area than other parts of New York State.

In 2000 and 2001, nine pesticides were commercially applied in Suffolk County that are registered for use as termiticides. Of these, permethrin, chlorpyrifos, cypermethrin, and fenvalerate account for 99% of the total pounds of all nine active ingredients applied in the county during this time period. Researchers compared the amounts of these products in the CMP area to the rest of Suffolk County. The results show that commercial applicators reported applying fewer pounds per square mile of termiticides in the CMP area than in the rest of Suffolk County in 2000 and 2001 (*Table 21)*. Because applications of termiticides in the CMP area are lower than in the rest of Suffolk County, they will not be investigated further.

3. Horticultural Oils and Carbaryl

Because of their widespread use in the CMP area, the use of horticultural oils and pesticide products containing the active ingredient carbaryl in the years 2000 - 2001 was evaluated. On a pounds per square mile basis, CMP applied less horticultural oils and more carbaryl compared to the rest of Suffolk County during the years 2000 and 2001 *(Table 21)*.

Because applications of horticultural oils in the CMP area are lower than those in the rest of Suffolk County, they were not investigated any further. Carbaryl was further evaluated in *Chapter V. Integration* because applications in the CMP area are higher than in the rest of Suffolk County.

4. VAPAM HL

To follow up on the use of VAPAM HL in CMP within the CMP area, the Pesticide Sales and Use Reporting Database was examined for applications of this product throughout New York State. For the years 1997 to 2001, this product was applied almost exclusively in Suffolk County, with CMP accounting for nearly 90 percent of the total quantity applied statewide. Applications in the area were reported for 1998, 1999, 2000, and 2001. The total quantity reported within a single year ranged from 1,800 gallons to 33,000 gallons. Applications were also reported in Eastern Suffolk County for the years 1997, 1998, 2000, and 2001 with the total quantity applied over a single year ranging from 55 to 3,000 gallons. The active ingredient in VAPAM HL, metam sodium, is the third most commonly used agricultural pesticide in the United States and has held this rank since 1995 with more than 50 million pounds applied nationwide each year (USEPA Pesticide Industry Sales and Usage Report).

However, because VAPAM only was used for agriculture in one small area, exposures, if any, would be limited and would not affect the overall CMP area. Therefore, VAPAM will be not be further evaluated.

f. Choosing a rate for comparing pesticide use

The State Health Department received comments on the Pesticide Use evaluation after preliminary results in the *Working Draft Report* were released in June 2004. Commenters noted that the reason that pesticide use was higher in the CMP area was because Suffolk County and New York State were used as comparison areas and use in the CMP area is more similar to other communities in Western Suffolk County and Nassau County.

Suffolk County and New York State were used as the comparison areas because the use of those areas was consistent with methods used to identify the elevation in breast cancer incidence as well as the other evaluations conducted as part of this investigation. It is true that the pesticide application *rates on a per square mile basis* are similar between the CMP area and other areas of Western Suffolk County and Nassau County, when pesticide use is divided by total land area (see *Figure 4).*

As discussed earlier, if it were possible, a different rate might be used to compare the use of different pesticides based on how and where the pesticides are applied and that analysis may have different results. For example, 2,4-D is used in landscaped areas, mostly lawns. If data on residential lot size and the size of each lawn were available for all ZIP Codes in New York State, researchers might have compared pounds of 2,4-D applied per acre of lawn in the CMP area to the rest of Suffolk County and New York State. However, those data were not available. Because 2,4-D is applied to the surface of lawns, expressing application rates as pounds of 2,4-D per square mile seemed reasonable to compare pesticide use given the limitations of available data.

Our evaluation of the pesticide use data suggests that a number of factors, including land use, lot size, population size, property values and other demographic characteristics may influence commercial pesticide application rates. Work continues as part of ongoing research activities at NYS DOH to evaluate the NYS Pesticide Sales and Use database to identify factors that influence pesticide use patterns in New York State. In addition, researchers are evaluating the data in an effort to identify other rate options to use in comparing different commercial pesticide applications in different areas.

g. Conclusion

The commercial pesticide application data for the year 2000 show that Suffolk County accounts for between 12% and 15% of the total professional pesticide applications statewide. The commercial application of total pesticide containing products in the CMP investigation area is higher on a per square mile basis than in the rest of Suffolk County, although totals vary in Suffolk County with higher rates in the western Suffolk County than in eastern Suffolk County. The majority of the pounds reported were in the form of dry fertilizer-pesticide combination products used on lawns and other landscape areas.

Commercial applications of products containing the active ingredient 2,4-D, mecoprop and dicamba were higher in the CMP area than in Suffolk County on a pound per square mile basis. Reported applications of termiticides and horticultural oils were lower in CMP than in the rest of Suffolk County. Reported applications of carbaryl were higher in CMP than in the rest of Suffolk County. Although CMP accounts for more than 90 percent of the total applications of VAPAM HL in New York State for the years 1997 to 2001, it was applied to a small area within CMP and would

not represent a wide-spread exposure. Carbaryl, dicamba, mecoprop and 2,4-D are evaluated further in *Chapter V. Integration.*

5. Industrial and Inactive Hazardous Waste Sites and Spills

a. Introduction

The Initial Environmental Inventory identified major industrial and waste sites and existing environmental databases for the CMP area. Area residents also identified some of these industries, waste sites and spills as concerns during the public availability session held in June 2002. NYSDOH staff evaluated all of this information *(Appendix IV-1).* What follows is a discussion of available information for these sources for the CMP area.

NYS DOH researchers referenced existing site evaluations to determine potential exposures from the release of chemicals from sites in the CMP area. Only exposures for people living within the CMP investigation area are discussed here.

- Lawrence Aviation is both an active business and a hazardous waste site. The US EPA is investigating environmental contamination at Lawrence Aviation. The EPA, DOH, DEC, ATSDR and Suffolk County Department of Health Services (SCDHS) continue to evaluate the potential for exposures. Lawrence Aviation is discussed further in this section.
- Brookhaven National Laboratory (BNL) is an active research facility that is outside the CMP area. Because many residents expressed concerns about the facility, state health researchers used existing sampling data to evaluate exposures from radioactive air emissions. More details about this evaluation are provided in this section.
- The Port Jefferson Power Station is an active power plant with ongoing permitted air emissions that were considered as part of the evaluation of air quality in Section IVE-3. Because of its importance in the CMP area and community concerns about historical operations of this plant, additional information about the Port Jefferson Power Station is provided in this section. However, no data were found to estimate the impact of historical exposures associated with air emissions from this power plant.
- Four areas are within the seven ZIP Codes that have undergone investigation and/or cleanup and are no longer classified as hazardous waste sites: Heins Landfill, Suffolk Materials Mining Corporation, Brookhaven Aggregates and Pine Road Ecology Site. Additional details about these sites are provided in this section.
- One hazardous waste site, RCA-Rocky Point Landfill, is outside of the area but near the seven ZIP Code boundaries. Soil sampling showed that contamination is contained on the site and does not affect the CMP area. No further evaluation was done on this site.
- DOH researchers reviewed data about leaking underground storage tanks in NYS DEC's Major Oil Storage Facility and Spill Incidents databases. Based on a review of these data and discussion with NYS DEC and

SC DHS staff, no widespread human exposures were identified. The Northville Industries gasoline leak, which is one of the largest spill incidents in the area, is discussed further below.

b. Lawrence Aviation Industries

Lawrence Aviation Industries (LAI) is in Port Jefferson Station in the Town of Brookhaven. Groundwater contamination potentially related to LAI was first detected in private wells north of the site in the 1970s. The groundwater contained trichloroethene, 1,1-dichloroethene, cis-1,2-dichloroethene and tetrachloroethene. Samples collected from a private drinking well in 1985 and 1986 contained trichloroethene levels ranging from 43-83 micrograms per liter (mcg/L). In 1987, four additional private wells north of the site were sampled and contained trichloroethene levels ranging from 10-910 mcg/L, cis-1,2-dichloroethene from 1-15 mcg/L and tetrachloroethene from 0-6 mcg/L. By 1987, five private wells contained levels of trichloroethene exceeding the current New York State drinking water standard of 5 mcg/L. Residents north of LAI with contaminated private water wells were supplied bottled water until they were connected to public water.

In 1997, the Suffolk County Department of Health Services identified 10 additional residential wells north of LAI that were potentially affected. Five wells were identified as contaminated and five wells were identified as vulnerable to contamination by tetrachloroethene, trichloroethene, and cis-1,2-dichloroethene in the groundwater. These homes were connected to the public water supply between 1997 and 1999. We do not know for how long private drinking water wells have been contaminated. The contamination would probably have been introduced no earlier than the 1950s. Residents using water during that time may have been exposed to trichloroethene, tetrachloroethene and cis-1,2-dichloroethene.

In 1991, trichloroethene contamination potentially related to LAI was found in Brook Road Pond and a small stream down-gradient from LAI. People may have contacted contaminated water in Brook Pond and the stream. However, in 1993, signs reading “Warning, contaminated waters; do not drink; avoid prolonged contact with skin” were posted around the pond to warn residents about potential exposures. Chemicals in the groundwater also may vaporize, migrate through pore spaces in the soil (soil vapor) and may affect nearby buildings. US EPA sampled soil vapor in the area, but results are not available at this time.

Limited data suggest that areas of soil contamination are present on the LAI site. The site is fenced; thereby limiting the potential for the public to contact contaminated soil. The US EPA began an on-site investigation in the fall of 2003, the results of which should provide more information on soil contamination.

Currently, documented exposures from the LAI site are due to trichloroethene, cis-1,2-dichloroethene, and tetrachloroethene in private drinking water. These data are discussed in the private drinking water evaluation in *Section IV E-7.*

Because there is currently no other information indicating that exposures have occurred, the Lawrence Aviation site is not discussed further. Nevertheless, investigations at the LAI site continue, and NYS DOH staff will continue to evaluate whether site-related exposures are occurring.

For information on US EPA's on-going investigations at the Lawrence Aviation site, please contact US EPA's project manager, Mr. Salvatore Badalamenti, at 212-637-3314.

c. Brookhaven National Labs

Residents were concerned about emissions of radioactive substances from Brookhaven National Laboratory (BNL). BNL is a 5,265-acre site in Upton, New York, about five miles southeast of the CMP area. BNL is owned by the US Department of Energy (US DOE) and has operated since 1947. The laboratory is used for multi-disciplinary research in high-energy physics, nuclear medicine, biology and chemistry. There are three inactive nuclear reactors on-site that were historically used for research. The site includes disposal facilities containing hazardous chemicals and nuclear waste. An inter-agency agreement was negotiated between BNL, US DOE, US EPA and New York State to address environmental issues.

NYS DOH researchers reviewed data related to emissions of radioactive substances from BNL to determine whether these emissions might have affected the CMP area. The winds near BNL come mainly from the southwest and the northwest, and typically carry air away from the CMP area. However, less frequent southeasterly winds may transport air from BNL towards the CMP area. An examination of air monitoring results for the years between 1973 and 1994 showed the levels of gross Beta particulates in the air (an indicator of release of radioactive substances) near BNL were well below statewide averages. We expect that levels in CMP are similar.

Groundwater contamination on- and off-site has been well documented. Some people living near BNL used private well water from the contaminated aquifer and were exposed to VOCs and low levels of tritium. Some of the VOCs in the off-site wells may have originated from another facility. The Suffolk County Water Authority wellfields near the site were not contaminated. The US DOE provided public water hookups to homes and businesses in the off-site area affected by groundwater contamination

Groundwater flow from the site is towards the south. While groundwater under the site is contaminated, it does not flow towards the CMP area, and therefore does not affect people living in the CMP area.

The ATSDR prepared a Public Health Assessment for BNL that addresses potential exposures and public health implications of the BNL site in greater detail. For more information about this public health assessment, please contact Mr. Andy Dudley of ATSDR at 404-498-0340 or Mr. Arthur Block of ATSDR's Region 2 office in New York City at 212-637-4307. For more information on the ongoing investigation at BNL, please contact the US EPA site project manager, Mary Logan at 212-637-4321.

d. Port Jefferson Power Station

Residents identified the effect of pollutant emissions from the Port Jefferson Power Station on breast cancer incidence as a community concern.

Information about the Port Jefferson Power Station was obtained from the Long Island Power Authority's November 2001 Environmental Assessment for the Port Jefferson Station Energy Project (Long Island Power Authority, 2001 Combustion Turbine Project Environmental Assessment; http://www.lipower.org/projects/turbines.html (January

2004)). The power station, which is located on 73 acres, is bounded by Village of Port Jefferson residential properties to the south, light industrial property within the Town of Brookhaven to the west, Village of Poquott residential properties to the north and Port Jefferson Harbor to the east. The power plant has been in operation for over 50 years. Through the 1950's, two coal-fired units were used to generate electricity. By the 1960's, two larger coal-fired electric generating units were added. In the late 1960's, coal firing stopped when all of the units were converted to fire #6 fuel oil and natural gas. By the mid-1990s, the two original units stopped operating and remain on permanent standby. In 2002, two new units were added to the facility. These units burn natural gas or low-sulfur oil when natural gas is unavailable. Other emission sources at the facility include a small gas-fired combustion turbine (used to provide "peaking" power) and a 500 horsepower diesel generator that is used to start this turbine.

The US EPA included emissions from the Port Jefferson Power Station in the models it used to evaluate air quality. However, these modeling studies do not reflect historical emissions of hazardous air pollutants associated with using coal as a primary fuel. Clearly, the emissions of some hazardous air pollutants such as polycyclic aromatic hydrocarbons would have been greater when coal was used as fuel than when fuel oil and natural gas were used. Emissions of regularly monitored air pollutants (e.g., particulate matter and sulfur dioxide) would also have been greater during those years. The data available to evaluate the higher levels of pollutants in the 1950s or 1960s are limited. Any evaluation based on those data would be too uncertain to yield meaningful estimates of the levels of pollutants to which people might have been exposed or to identify the areas in which those exposures might have occurred. Based on the evaluation criteria developed by our research team, our researchers used more recent air pollution estimates from the US EPA to characterize the impacts from this power plant. The US EPA air modeling studies and the results of these studies for the CMP area are discussed in Section IV E-3 Air Quality.

References

Long Island Power Authority, 2001 Combustion Turbine Project Environmental Assessment; http://www.lipower.org/projects/turbines.html (January 2004)

e. Heins Landfill

The Heins landfill site is in Port Jefferson Station. In 1985, allegations of improper waste disposal at Heins Landfill were reported to the NYSDEC. Soil samples collected at different times from 1985 to 1990 did not identify any hazardous wastes on the site. Because no contamination was found on the site, Heins Landfill was not identified as the source of the groundwater contamination. In 1991, Heins Landfill was removed from the Registry of Inactive Hazardous Waste Sites in New York and referred to the NYSDEC Division of Solid Waste for final closure. Because no hazardous wastes were identified at the Heins Landfill, it will not be given further consideration in this investigation.

f. Suffolk Material and Mining Corporation

Suffolk Material and Mining Corporation is in East Setauket. In 1984, the NYSDEC and Suffolk County Department of Health Services (SCDHS) received complaints from local residents concerning activities at the site, including

allegations of hazardous waste disposal. The complaints resulted in a site investigation and the subsequent listing of Suffolk Materials and Mining Corp on the Registry of Inactive Hazardous Waste Sites in New York.

Sampling of soil, groundwater, and soil gas conducted up until 1989 failed to identify any hazardous wastes on the site. Groundwater samples collected did reveal contamination of several nearby private drinking water wells by volatile organic compounds. Because no contamination was found on the site, Suffolk Materials and Mining Corp. was not identified as the source of the groundwater contamination. In 1993, Suffolk Materials and Mining Corp. was removed from the Registry of Inactive Hazardous Waste Sites in New York and referred to the NYSDEC Division of Solid Waste for final closure. The landfill is scheduled to be capped in the Summer of 2004. Because no hazardous wastes were identified at the Suffolk Materials and Mining Corp., it will not be given further consideration in this investigation. The drinking water contamination in the area is discussed in the private drinking water evaluation in Section IV E-7.

g. Brookhaven Aggregates

Brookhaven Aggregates is in Miller Place. In 1984, allegations of improper disposal practices led to an investigation by NYSDEC at Brookhaven Aggregates Landfill. Samples of soil and groundwater were collected in 1984 and in 1986. The site was classified as a hazardous waste site in 1988, based on the results of the laboratory analysis of the 1986 samples. Upon further review, however, inaccuracies in the laboratory results were discovered and subsequent samples collected in 1997 were not found to contain hazardous chemicals. In July 1998, the site was removed from the Registry of Inactive Hazardous Waste Sites in New York and referred to the NYSDEC Division of Solid Waste for final closure. A passive landfill gas collection system is currently in operation at this site and groundwater monitoring is conducted routinely. Because no hazardous wastes were identified at the Brookhaven Aggregates Landfill, it will not be given further consideration in this investigation.

h. Pine Road Ecology Landfill

Pine Road Ecology is in the Hamlet of Coram. Sampling of a private drinking water well near the Pine Road Ecology Site detected the presence of volatile organic compound contaminants. The site was listed in the Registry of Inactive Hazardous Waste Sites in New York, and an investigation into disposal practices at the site was conducted. The investigation did not find any evidence of hazardous waste disposal at the Pine Road Ecology Site, so it was removed from the Registry of Inactive Hazardous Waste Sites in New York and referred to the NYSDEC Division of Solid Waste for final closure. Because no hazardous wastes were identified at the Pine Road Ecology Site, it will not be given further consideration in this investigation. The drinking water contamination in the area is discussed in the private drinking water evaluation in Section IV E-7.

i. Northville Industry Corporation East Setauket Terminal Gasoline Leak

In November 1987, gasoline contamination of the groundwater beneath the Northville Industry Corporation's East Setauket Terminal was discovered. A leak in one of the facility's underground pipes was the source of the gasoline release. Approximately 1.2 million gallons of gasoline leaked into the ground and groundwater that is about 100 feet

below the surface. While it is not known when the leak began, it could have occurred over a period of 25 to 30 years. The pipe was repaired, and an inspection of the facility's storage tanks and underground pipes was conducted. No other leaks were discovered.

Northville Industries took steps to identify the extent of contamination and to implement remedial measures. The remedial procedures included steps to recover gasoline from the groundwater and prevent the contamination from spreading farther. On October 13, 1994, Northville Industries entered into a Consent Order with New York State. The agreement defined actions necessary for the completion of site remediation and closure.

As a result of gasoline entering the groundwater, exposures to local residents in East Setauket could have occurred through contamination of drinking water and the intrusion of vapors into local residences. An evaluation of public and private drinking water that evaluated contaminants from this site and other sites is found in Section IV E-6. Water Supply. Records indicate that most nearby homes were served by public water. A review of the public drinking water data did not detect drinking water contaminants associated with this gasoline spill. The review of private water drinking databases also did not show these contaminants in private wells near the facility. Testing for vapor intrusion was undertaken in some affected residences as part of the actions required by New York State and the SCDHS. Levels of indoor air contaminants associated with the gasoline spill were sufficiently low in these samples so that no further action was required to address vapor intrusion in local residences.

6. Public Water Supply

a. Background

The majority of the CMP area is served by public water supply provided by the Suffolk County Water Authority (SCWA), which is the largest provider of public drinking water in Suffolk County and in the CMP area. In the 1970s and 1980s, several public water suppliers provided water to small areas, including both *community* (residential) and *non-community* (non-residential, e.g. commercial, government) drinking water. The SCWA now serves all of these customers, although a few non-community suppliers are still active. *Table 23* shows all community water systems that have or continue to serve customers in the CMP area.

The SCWA provides drinking water from 44 groundwater wells. These wells serve two service areas also called *pressure zones*. Pressure zones are portions of the water distribution system that are under separate control mechanisms. The majority of the CMP area falls within the SCWA's pressure zone number 15. The northwest portion of the study area, including western Port Jefferson, Poquott, Setauket and Old Field, are served by pressure zone 14. No wells are located in zone 14. Water in pressure zone 14 is provided by groundwater wells in pressure zone 15. Though specific wells tend to be used for individual pressure zones, water can be exchanged across pressure zone boundaries depending on operational pressure and demands on the system.

Public drinking water is provided by operating a number of facilities in response to distribution system water pressure or by timed delivery by pumping systems. Wells are not all in use at any given time, but are brought into production based on consumer use, maintenance needs and active roles in eliminating entry of contaminants into

the distribution system. Although much of the data analyzed in this report is limited to point-of-entry samples to the distribution system, the amount of water provided from any given well can vary substantially over time, which is not reflected when using sampling data. A retrospective analysis of the contribution of specific wells to the distribution system or how much finished water may cross pressure zones was not conducted as part of this evaluation.

Table 22. **Public water systems in CMP Area**

Community Systems	Non Community Systems
Suffolk County Water Authority (SCWA)	112 Exxon Service Station
Sound View Association*	555 Proffessional Bldg
Scott's Beach Association*	600 Middle Country Road*
Crest Hall*	7 11 Store #25627 (Mt Sinai)*
Terrace on the Sound*	Amici's Restaurant
Culross Corporation (Culross Beach)*	Brookhaven Bathing Association
	Carvalho Concrete Corp.
	Central Brookhaven Head Start
	Colonial Shop Center*
	Commerce Center
	Coram Garden Shopping Ctn
	Coram Municipal Office*
	Coram Sunoco
	Coram Towers*
	Estonian Educational Soc
	Gaslight Motor Lodge
	Imperial Nurseries*
	Local 66 Training Facility
	Lombardi's Restaurant*
	North Shore Mall
	Pickwick Beach Club*
	Port Jeff. Village Beach West
	Weir's Corner Deli

**Indicates Public Water Systems no longer active: either closed or receiving water from SCWA*

In addition to public drinking water, a portion of the study area's population receives drinking water from private wells (see *Section IV E-6*). Census data indicate that approximately 2000 private wells in the investigation area were in service in 1980 and 1990. Fewer private wells are in service in the CMP area today as the trend is to change from private wells to public water service.

b. Drinking Water Standards

New York State and the federal government regulate drinking water to protect public health. Regulations have evolved over time for a variety of volatile organic compounds (VOCs), metals, pesticides and pathogens.

In 1974, Congress passed the Safe Drinking Water Act that standardized the protection of drinking water on a national level. States that previously had established drinking water standards were required to make their standards at least as stringent as the national standards promulgated by the US EPA. These national drinking water standards first went into effect in 1977.

Beginning with the discovery of synthetic organic chemicals in some public water supplies in Nassau County in 1976, State and County resources were used to sample all community public drinking water wells in Nassau and Suffolk Counties for a variety of volatile organic compounds (NYSDOH, 1981). Most of the commonly found VOCs are chemicals that have been used as solvents, cleaners and degreasers in a variety of industrial and household settings. They can enter drinking water supplies through accidental spills and improper disposal practices. The permeable soils in Suffolk County make the underlying aquifer susceptible to the introduction of VOCs from these kinds of activities.

Historically, the guideline for most VOCs in New York State was 50 micrograms per liter (mcg/L). The US EPA, through amendments to the Safe Drinking water in mid-1987, promulgated federal rules for eight specific VOCs commonly found in groundwater contamination. These federal rules were enacted in New York State in 1989, when more stringent standards for public drinking water went into effect changing the standard for many VOCs to 5 mcg/L. As part of New York State's drinking water standards, maximum contaminant levels (MCLs) for contaminants are codified in regulation (NYCRR Part 5-1). Even before New York State enacted the more stringent health-based standards, voluntary restrictions were put into place where contaminants were found to exceed the anticipated standards, removing sources of water from distribution to the public.

The SCWA historically has provided drinking water that met or was a higher quality than state and federal drinking water standards. SCWA monitors for water contaminants through a comprehensive monitoring program that takes samples more frequently than New York State or the federal government require. The monitoring results are used to manage each water supply well and apply necessary treatment to comply with drinking water standards.

When chemical contaminants have been detected in smaller community and non-community systems in the CMP area, these smaller suppliers usually opted to close the well and connect with the SCWA rather than treat their own well water and continue operation. Expanding the SCWA service area has proven to be the most economically efficient option to address contaminated drinking water wells and assure high quality drinking water throughout the service area. A detailed description of historical contaminants found in these supplies is provided later in this section.

c. Sources of Drinking Water Supply Contaminant and Potential Exposure Information

NYS DOH researchers evaluated four data sources to assess historical chemical contamination of public and private drinking water in the CMP area. These analytical data sets, though providing some of the best proxies for exposure in the study area, have been collected for a variety of purposes including regulatory compliance and targeted responses to specific needs to address contamination issues. None of these data were collected as part of

a specific survey designed to evaluate or measure human exposure to environmental contaminants. These data sources are described as follows.

1. **Suffolk County Department of Health Services (SCDHS) public drinking water supply data (1971-1996).** This is the largest data set with 1,369 finished water samples from public community water systems and 225 water samples from public non-community water systems. This data set is maintained as part of the Public Water Supply Supervision Program for all public drinking water and conforms to monitoring requirements for regulatory purposes providing sample data at regular frequencies and at established locations. The data set also includes additional samples measuring contaminants in raw water, which refers to water coming directly out of the well prior to treatment. Raw water sample data would be less indicative of potential exposure and are not included in analyses here. Water sample data collected since 1996 are maintained in a separate database. The more recent data set, though not directly relevant to this study since the elevated incidence of breast cancer precedes the date of this data, was also evaluated to determine if any substantial omissions would occur based on using the earlier data set alone.
2. **The United States Geological Survey (USGS) National Water Information System Data (1977-1993).** This database is a subset of the USGS national water information system, which contains historical water quality information about groundwater. It includes information about both public water supply wells and other wells, such as monitoring wells. For the most part, these data measure contaminants found in groundwater and do not consider water treatment so they are not an ideal measure of drinking water at the tap. Analytes include metals, VOCs and pesticides, but the amount of data for each well varies considerably.
3. **NYS DOH drinking water system samples (1976-1995).** This data set includes a variety of water quality information from samples taken to independently survey water systems for contamination. Many of these samples were taken by the SCDHS and submitted to the State's laboratory for analysis, while other samples may have originated from special interest studies. This data set includes water quality information directly from public drinking water wells, from within the public distribution system and from a limited number of private wells.

In addition to the data sets that were evaluated for the quality of their information about drinking water contamination, spatial data were used as part of this evaluation. These data were used to delineate public water service areas and to provide specific well locations and associated sample data. Water district and pressure zone boundaries were provided by the SCWA or developed by NYS DOH researchers based on water distribution records available from Annual Water Quality Reports and their predecessors, prior service area maps for SCWA and other community water systems, and from water purveyor files maintained by the Department of Health. Locations of wells were provided from:

- Safe Drinking Water Information System (SDWIS) data maintained by the NYS DOH (NYS DOH, 2003)
- SCWA well location and associated parcel data (SCWA, 2003); and
- Results of the recent Source Water Assessment Study for Long Island (NYS DOH, 2003).

Well locations were validated within the study area and locations were corrected as needed using standard validation techniques relying on agreement of multiple data sets including tax parcel data, street address data, digital orthoimagery and direct consultation with SCWA staff. Researchers used data from SDWIS to join well locations to the water quality sampling data set based on re-creation of unique identifiers for each source. Inactive wells that did not have specific geographic locations in the SDWIS data set were all successfully located to apply historical data to the study area. All sample data were successfully associated with specific wells within the study area.

d. Results of Applying Screening Criteria

Each of the three public drinking water supply information data sets was assessed for its use in evaluating historical environmental exposures using the criteria described in *Section IV-C.* The results of this evaluation are summarized in *Appendix IV-1* and described in more detail below.

1. **SCDHS public drinking water supply data (1971-1996)** provides the most comprehensive data set of public drinking water quality in the CMP area and Suffolk County for both consistent sampling locations and regular sampling intervals. Many different analytes are represented in this data set, with each individual sample record tending to have complete information for groups of analytes. In addition, the number of analytes reported has increased over time as a result of new monitoring requirements and improvements in analytical methods. This data set provides sufficient information for an evaluation of the extent and duration of contamination associated with public drinking water in the CMP area based on complete geographic coverage of the study area, including samples from all drinking water wells serving the area. Because most people were served by public water, these data also provide information about a large portion of the population within the CMP area that may have been exposed to contaminants.

   The quality of environmental contaminant data and potential for exposure is high, providing direct evidence of the presence or absence of specific contaminants. The actual pathway for exposure is not complete for these data sets, however, as neither definitive information regarding actual contaminant levels reaching households is available, nor is actual consumption information available for what contaminants may have reached specific consumers. An analysis of the data did not indicate the presence of unexpected values due to data entry errors or questionable sample results. Locations of wells were confirmed and corrected as necessary based on ancillary data sets such as road address and historical records along with proximity of the well location to service areas and well fields. Because the amount of data about various water quality contaminants varies over time, researchers further evaluated this data set by grouping classes of chemical contaminants in drinking water, VOCs, metals and pesticides. This evaluation is described below.

   - *VOC data.* Many wells were sampled and the water analyzed for a variety of potential contaminants, including VOCs, even prior to the implementation of federal and state laws requiring such monitoring, thus providing data for the early years in this evaluation. The range of sample data for VOCs was from 1971 to 1996, a time period directly relevant to the CMP Investigation. This data set meets the criteria established by ATSDR to provide evidence of a completed exposure pathway (see *Section IV-C)* These data, which are

among the most thorough historical environmental data sources available, indicate that low levels of contaminants were historically present, although the level of contamination did not exceed the 50 mcg/L water quality guideline in place prior to 1989. Researchers further analyzed these data, which is described in more detail in later in this section.

- *Inorganics data.* Information about metals in drinking water was collected along with a variety of other inorganic analytes. Most samples measured the physical characteristics of the water such as alkalinity, conductivity and total dissolved solids. Of the metals that were routinely tested for, iron and manganese were the most commonly detected in groundwater samples with 102 of 897 and 46 of 841 detections, respectively. Lead was detected in 30 out of 604 samples taken in the early 1980s. Copper also was reported with 48 detections in 862 samples. Lead and copper enter drinking water from pipes within the distribution system as the result of corrosion, which was a common occurrence within water distribution systems. These sample data do not adequately represent potential exposure to lead and copper as the sample locations are near the source and not at the consumer water tap at the end of plumbing systems. Metals have not been detected at significant levels in drinking water in the CMP area since the implementation of corrosion control programs in the mid-1980s. Other than lead and copper, which were not unique exposures in the CMP area, no other metals were detected even though a large number of samples were taken (>500). Specifically, no detections were found for arsenic, and only single detections, which were not reproduced in further sampling, were reported for selenium, cadmium and chromium. These data do not indicate long-term exposure to metals above the detection limit in the CMP area. As a result, no further evaluation was done with these metals data.

  In addition to metals, data was available for other inorganic compounds including nitrate and ammonia. Though other areas of Long Island may experience elevations in these compounds, the study area did not present unusual values for either. Of 903 nitrate samples taken, 630 resulted in positive detections (70%), with a single sample of 21 milligrams per liter (mg/L) exceeding the health-based standard of 10 mg/L. Ammonia was detected at a lower frequency with 75 out of 877 samples resulting in positive detections, with a maximum value of 1.4 mg/L. The CMP area has never exceeded drinking water guidelines for asbestos and no asbestos piping (also known as AC-Pipe) was used to carry public water in the CMP area and water provided by the SCWA.
- *Pesticide data.* Data from a substantial number of samples analyzed for pesticide contaminants were available for community and non-community public water supply systems. Data were assessed for historical pesticides (largely chlorinated hydrocarbons) from 1966 through 1996 and for additional pesticides from 1991 through 1996. Separate results for Aldicarb also were available for community (1989-1996) and non-community systems (1987-1996) and from special studies conducted between 1980 and 1996. These data did not detect any pesticides in community public drinking water systems. Data for a single non-community water system, North Shore Mall, reported three instances of Aldicarb in drinking water between 1992 and 1993. These data do not indicate long-term exposure to these pesticides. As a result, no further evaluation was done with these data.

2. **SCDHS public drinking water supply data (1996-present).** The more recent SCDHS data set includes results for 251 analytes and a total of 35,381 samples within the study area. This includes sample results for 34 additional analytes not tested for in the prior data set. These analytes largely include additional pesticides and their metabolites. Of these additional analytes, detections occurred for Metolachlor metabolites (two detections out of 423 samples with a maximum of 0.4 mcg/L for Metolachlor oxanilic acid) and for Alachlor and its metabolites (13 detections of 397 samples, with five detections of Alachlor with a maximum value of 0.76 mcg/L). With the exception of three samples, detections for these two analytes and their metabolites occurred near Miller Place. The assessment of these data did not indicate that significant omissions in the analysis would occur if the focus remained on the historically relevant data sets.
3. **USGS National Water Information System Data (1977-1993).** Results for several hundred samples were available for the CMP area from the USGS. The data are not as comprehensive as the SCDHS public water supply data both in terms of its geographic coverage and frequency of samples. By analyzing VOC data, researchers determined that contaminants were present in raw water from wells used for public drinking water, however they could not determine whether these data measured the quality of drinking water at the tap, because the samples reflect water quality prior to any treatment within the public water system. Though not as comprehensive as the SCDHS drinking water data, researchers used this data set to validate their assessment of VOC contamination. Data for inorganics, including heavy metals and pesticides, also were evaluated with no detections reported. Because there were no detections and because historical information about pesticides was not in the data set, researchers did not use this data set other than to verify results of the VOC assessment.
4. **NYS DOH public water system samples (1976-1995).** Researchers reviewed the results of 3,028 samples from 292 public water distribution sample sites in the CMP area from the NYS DOH. The data are not as comprehensive as the SCDHS public water supply data, both in terms of their geographic coverage and frequency of samples. Many of the samples report results for only a single analyte in comparison to the more comprehensive monitoring data in the SCDHS data sets. Roughly half of the samples are taken at source wells providing limited confirmation of the monitoring data set. The other half of the data are from specific locations within the distribution system. The extent of duplicate samples between the NYS DOH and SCDHS data sets was not determined as there is no common identifier to relate the two data sets. This data set was included to provide data to assess contaminants within the distribution system, rather than within individual drinking water wells. The only use of this data set was to trace contaminants within the distribution system adjacent to wells with contaminant detections. This data set was used in preference to the distribution sample information in the SCDHS data set, based on the availability of complete address information. The addresses allowed researchers to identify sample locations accurately. The number of samples collected across the CMP area varied, with some areas covered by a greater number of samples than others. Only three private wells are included in these data. Location information was recorded by address of the sample site and lacks detail to determine accurate sample locations. As a result, researchers determined that these data were incomplete with respect to assessing possible environmental exposures. However, these data were used in conjunction with

SCDHS data in a geographic assessment of contaminants from specific water supply wells throughout the distribution system, which is described further later in this section.

e. Results

Based on the review of the water supply data sets, NYS DOH researchers found evidence of a low level of VOC contamination in the CMP area. These levels did not exceed any drinking water standards in place during the time of the exposure, nor were they unusual compared to data for the rest of Suffolk County. However, given the high quality of the exposure information, researchers were able to identify geographic areas where water containing low levels of VOCs may have been distributed through the water system.

1) Summary Data. VOC Concentrations in Community and Non-Community Supplies.

In this section, summaries of SCDHS and USGS data for community and non-community and private water supplies are presented to provide an overview of the maximum levels of contamination detected in the CMP area.

Detections of VOCs exceeding 5 mcg/L from the SCDHS community public water supply data are summarized in *Table 23.* As many as 76 compounds were analyzed in 1324 samples. Fifty-three of the 76 analytes were never detected in any of the samples. Seven compounds had one detection that was not reproduced and eight other compounds were detected several times but never exceeded today's regulatory standard of 5 mcg/L.

Of all analytes, 1,1,1-trichloroethane was detected in the largest number of samples (133 samples). Chloroform was detected in 60 samples followed by 1,1-dichloroethane in 58 samples. Six analytes were detected above 5 mcg/L. These were 1,1,1-trichloroethane, 1,1-dichloroethane, carbon tetrachloride, benzene, xylenes and chlorobenzene. Although chloroform is included, it is considered a constituent of total trihalomethanes (TTHMs) with a higher regulatory standard of 80 mcg/L, which is based on a running annual average of quarterly samples. Chloroform is a common by-product of chlorination of organic matter and was retained as a potential indicator of organic matter in groundwater. All VOC detections for non-community water systems are summarized in *Table 24.* Of 69 analytes with sample records in the data set, six analytes were detected.

Table 23. Community water system sample results exceeding 5 mcg/L (1971-1996)

Analyte	Maximum concentration (mcg/L)	Number of detections	Number of samples analyzed	Percent detected
1,1,1-trichloroethane	13	133	1260	10.6
Chloroform*	12	60	1272	4.7
carbon tetrachloride	9	38	1272	3.0
1,1-dichloroethane	8	57	984	5.8
Benzene	12	9	894	1.0
1, 2-dichloropropane	5	9	993	0.9
Chlorobenzene	6	1	822	0.1
xylenes (o,p,m, total)	8	5	890	0.6

Source: SCDHS public drinking water supply data

Table 24. Non-community water system sample results exceeding 5 mcg/L (1971-1996)

Analyte	Maximum concentration (mcg/L)	Number of detections	Number of samples analyzed	Percent detected
1,1,1-trichloroethane	18	23	46	50.0.
1,1,1-dichloroethane	7	4	26	15.4.
Chloroform*	5	2	47	4.3

Source: SCDHS public drinking water supply data

** is considered a constituent of total trihalomethanes (TTHMs) with a higher regulatory standard of 80 mcg/L*

VOC detections in USGS Water Information System Data is presented in *Table 25.* Of 102 analytes tested, four had detections four or more times. Out of the remaining data, 84 analytes had no detections, 13 had either only one or two detections, and one addressed the presence of bacteria. The presence of chloroform in raw groundwater samples suggests that this contaminant may not be a disinfection by-product due to chlorination, but is likely present as a contaminant from other sources.

Table 25. USGS water information system data summary (1977-1993)

Analyte	Maximum concentration (mcg/L)	Number of detections	Number of samples analyzed	Percent detected
1,1,1-trichloroethane	11	24	275	8.7
Chloroform	16	4	276	1.4
carbon tetrachloride	7	5	276	1.8
Toluene	7	4	143	2.8

Source: USGS National Water Information System Data

The community and non-community water data sets show that 1,1,1-trichloroethane was the principal contaminant found in public drinking water in the CMP area. In addition, 1,1-dichloroethane, a degradation product of 1,1,1-trichloroethane, was found in the public water supply data. Chloroform also is common among the public water supply data sets. Carbon tetrachloride and xylenes appear in two of the public water supply data sets.

2) Comparisons with other areas

In this section, researchers evaluated if the level and frequency of drinking water contamination in the CMP area was higher than in the remainder of Suffolk County. This evaluation showed that no unusual exposures existed in either frequency of contamination or level of contamination.

Data from the study area were compared to data for the remainder of Suffolk County. The reliance on groundwater as the sole source of drinking water is unique to Long Island, making it inappropriate to compare water quality in the CMP area with New York State as a whole. Most other areas of the state serving major populations rely on surface water sources, which typically contain a different set of contaminants than those found in groundwater. Researchers opted to use Suffolk County as the comparison area, rather than all of Long Island to be consistent with comparisons for other environmental media. To make comparisons for community and non-community water supplies, researchers calculated the median values for all sample results where contamination was detected in the CMP area and in the remainder of Suffolk County. The medians of detected contaminants were then compared in a ratio, where a ratio of 1.00 would indicate no difference from the remainder of the County. In all instances the median level of contaminant in community water supplies was lower in the CMP area than in the remainder of the county *(Table 26).* The median value used in this comparison is not a valid indicator of either the level of environmental contaminants or potential exposure, since the level of sampling effort clearly focused on wells with contaminants. As a result, the larger number of samples taken at wells with contaminant issues would inflate the median value. Nevertheless, this is a useful measure for comparing like data sets with similar biases to determine if any contaminant stands out in the study area.

Researchers also compared the frequency of contamination or *rate of detection,* in the CMP area and the remainder of Suffolk County for community and non-community water systems by dividing the number of detections

by the number of samples analyzed for each contaminant in both areas. The results for community water systems are presented in *Table 27* as Percent Detected. This evaluation indicates that for community water supplies the frequency of contamination in the CMP area also is lower than in the rest of Suffolk County for all but three of the listed analytes: carbon tetrachloride, benzene and chloroform. Because sampling efforts are increased when contamination is detected, these comparisons may be biased based on more frequent sampling of areas of known contamination. As such, the frequency of contamination measure does not provide an overall measure of environmental contamination. However, collectively, comparisons of both the concentration and detection rate generally indicate that the CMP area has not experienced substantial public water supply contamination in community water systems relative to the remainder of Suffolk County that could reasonably be related to any observed increase in disease incidence.

Table 26. Community public water system data – median contaminant levels in the CMP area versus the remainder of the county (1971-1996)

Analyte	CMP area median concentration detected (mcg/L)	Suffolk County median concentration detected (mcg/L)*	CMP area/ Suffolk County ratio
1,1,1 –trichloroethane	2	4	0.5
1,1-dichloroethane	1	2	0.5
carbon tetrachloride	2.5	3	0.8
Benzene	2	2	1.0
1,2-dichloropropane	3	3	1.0
Tetrachloroethene	1	4	0.25
Trichloroethene	0.75	5	0.15
Chloroform	1.0	1.0	1.0

Source: SCDHS public drinking water supply data

**Excluding the Coram / Mount Sinai / Port Jefferson Station study area*

Table 27. Community public water system data – detection rates in the study area versus rates in the remainder of the county (1971-1996)

	CMP area			Suffolk County*		
Analyte	Number of detections	Samples analyzed	Percent detected	Number of detections	Samples analyzed	Percent detected
1,1,1-trichloroethane	133	1260	10.6	1451	9836	14.8
1,1-dichloroethane	57	984	5.8	641	7470	8.6
carbon tetrachloride	38	1272	3.0	25	9842	0.3
Benzene	9	894	1.0	36	6577	0.6
1,2-dichloropropane	9	993	0.9	136	7502	1.8
Tetrachloroethene	11	1257	0.9	518	9803	5.3
Trichloroethene	8	1273	0.6	585	9892	5.9
Chloroform	60	1272	4.7	392	9911	4.0

Source: SCDHS public drinking water supply data

**Excluding the CMP investigation area*

Compared to the remainder of the County, the median concentration of detected contaminants in non-community water systems is considerably lower in the CMP area than the remainder of Suffolk County, with the exception of 1,1,1-trichloroethane *(Table 28).* In general, the number of samples collected varies considerably among different non-community wells based on whether contaminants have been detected in that well. Analysis of the rate of detection in the CMP area and the remainder of the County largely reflects sampling bias introduced by repeated sampling at known sources of contamination and by a relatively low number of samples *(Table 29).*

Table 28. Non-community public water system data – median contaminant levels in the CMP area versus the remainder of the county (1971-1996)

	CMP area median concentration detected (mcg/L)	Suffolk County* median concentration detected (mcg/L)	CMP area/ county ratio
Analyte			
1, 1,1-trichloroethane	8.0	3.0	2.7
1,1-dichloroethane	3.0	2.0	1.5
carbon tetrachloride	ND	3.0	NA
Benzene	ND	2.0	NA
1,2-dichloropropane	0.5	3.0	0.2
Tetrachloroethene	ND	4.0	NA
Trichloroethene	ND	6.0	NA
Chloroform	3.0	3.0	1.0

Source: SCDHS public drinking water supply data

**Excluding the CMP investigation area*

Table 29. Non-community public water system data – detection rates in the study area versus rates in the remainder of the county (1971-1996)

	CMP area			Suffolk County*		
Analyte	**Number of detections**	**Samples analyzed**	**Percent detected**	**Number of detections**	**Suffolk County* samples analyzed**	**Percent detected**
1,1,1-trichloroethane	23	46	50.0	144	1699	8.5
1,1-dichloroethane	4	26	15.4	10	1234	0.8
carbon tetrachloride	0	46	0.00	1	1696	0.1
Benzene	0	36	0.00	7	3455	0.2
1,2-dichloropropane	3	26	11.5	17	1230	1.4
Tetrachloroethene	0	47	0.00	38	1700	2.2
Trichloroethene	0	47	0.00	40	3455	6.0
Chloroform	5	47	10.6	113	1687	6.7

Source: SCDHS public drinking water supply data

**Excluding the CMP investigation area*

3) Characterization of Areas with Elevated Contaminant Levels and Potential for Exposure

In this section, researchers evaluated whether low-level VOC contaminants from public drinking water wells were widespread throughout the CMP area, or were localized to specific areas within the CMP area. Researchers analyzed records to locate drinking water wells where contaminants were detected. The current drinking water standard of 5 mcg/L was used as the measure to indicate exceedances, and the frequency of detections were used to determine if any instance of chronic, low level contamination below the drinking water standard existed in the study area. Once a well was identified with contaminants exceeding 5 mcg/L, the data were re-analyzed to include all analyte detections for that well, whether or not the detection exceeded the drinking water standard. The purpose in including all detections for wells with contaminant exceedances was to provide a conservative view of the extent of contamination so that locations where the potential for synergistic effects among contaminants could be identified. No instance of low-level chronic contamination was found in wells that did not also have contaminants exceeding drinking water standard. After specific wells were identified, researchers evaluated the proximity of contaminated wells to determine whether any common geographic or distribution areas existed with contaminant detections.

4) Analysis by Constituent Occurrence at Wells

Each of the contaminants reported in the SCHD data set and their occurrence at specific wells are listed here.

1) 1,1,1-trichloroethane and 1,1-dichloroethane were detected in 29 community wells. Exceedances of 5 mcg/L for 1,1-dichloroethane occurred at wells 05447 (1) and 12430 (3), while 21 exceedances of 5 mcg/L for 1,1,1-trichloroethane MCL occurred at wells 23827 (1), 05070 (1), 08736 (2), and 15962 (17). One non-community well (Coram Municipal Office Building) had 19 exceedances of 5 mcg/L for 1,1,1-trichloroethane and one exceedance of 5 mcg/L for 1,1-dichloroethane.

2) Carbon tetrachloride was detected in four wells (51953, 61910, 40838, 30088). In two of these wells (61910 & 51953) it was detected above 5 mcg/L. In well 61910 it was detected above 5 mcg/L in one sample collected on 6/7/1980. In well 51953 carbon tetrachloride was detected above the current MCL seven times in samples collected between 1979 and 1981.

3) 1,2-dichloropropane was detected nine times in two community wells (47219, 52451) and three times in one non-community well (North Shore Mall). Detections at 5 mcg/L occurred in 1981 and 1982 at well 47219 (2) and in the single detection for well 52451 (1) that was not reproduced.

4) Benzene was detected in only five wells (61910, 19465, 32180, 32325, 51953). In well 61910, two samples on 12/23/1987 and one on 1/6/1987 were found to be above 5 mcg/L.

5) Chlorobenzene was detected in only one sample above 5 mcg/L at well number 47310 on 3/1/1981. No other detection for this compound was found in any other well.

Table 30 shows the number of detections that exceeded 5 mcg/L. Of the 16 wells that had multiple contaminant detections, nine of these wells had exceedances. In all instances but one, the trend is to have multiple contaminants present at each source. 1,1,1-trichloroethane is often accompanied by 1,1-dichloroethane, and

carbon tetrachloride is always accompanied by benzene in these sample data. The exception to this trend is 1,2-dichloropropane, which is likely to have originated from application of this compound as an agricultural fumigant on lands adjacent to the well.

Table 30. Constituent wells with exceedances of 5 mcg/L

Well Identifier	Analytes									
	1,1,1-trichloroethane		1,1-dichloroethane		carbon tetrachloride		1,2-dichloropropane		benzene	
	Number of detections	*Number of exceedances of 5 mcg/L*	*Number of detections*	*Number of exceedances of 5 mcg/L*	*Number of detections*	*Number of exceedances of 5 mcg/L*	*Number of detections*	*Number of exceedances of 5 mcg/L*	*Number of detections*	*Number of exceedances of 5 mcg/L*
5070	4	1	3	-	-	-	-	-	-	-
5447	7	5	5	1	-	-	-	-	-	-
8736	3	2	1	-	-	-	-	-	-	-
12430	6	4	3	3	-	-	-	-	-	-
15962	19	17	8	-	-	-	-	-	-	-
23827	14	1	-	-	-	-	-	-	-	-
47219	-	-	-	-	-	-	8	2	-	-
51953	9	-	7	-	25	7	-		1	-
61910	9	-	1	-	11	1	-		3	3
Coram Municipal Office Building	23	19	3	1	-	-	-	-	-	-

Source: SCDHS public drinking water supply data

5. Analysis by Common Geographic (Distribution) Areas

Additional analysis of the community and non-community well data revealed that the study area could be further divided into six smaller geographic regions based upon a small subset of wells showing an exceedance of 5 mcg/L for one of five VOCs *(Table 31.).* These areas were identified as the Scott's Beach, Coram, Setauket, Sound View Association, Crystal Brook and Sequoia Park, north of Coram.

1) Scott's Beach. The Scott's Beach Water Company originally provided residents of Scott's Beach, in the northeastern section of the study area with drinking water. Results of samples collected between October 1979 and July 1992 showed that two wells (#05070, Hilltop Road Well #1 and #8736, Beach Street Well #2) exceeded 5 mcg/L for 1,1,1-trichloroethane in samples collected on May 4, 1987. The sample collected from the Beach St. well had a 1,1,1-trichloroethane concentration of 15 mcg/L with the Hilltop Rd well showing a concentration of 16 mcg/L. A total of four detections of 1,1-dichloroethane were reported for these wells, none of which exceeded 5 mcg/L. These two wells were closed in December 1992 and the area is now served by the SCWA.

The Scott's Beach Water Company served between 80 and 250 people while it was in operation. Sample results from between 1979 and 1987 were available from the NYS DOH public water system sample data characterizing the water quality within the distribution system. Based on the likely isolation of this system from

exchanges with other sources of water, direct well source samples with confirmed exceedances and corroborating sample results from the distribution system, the potential for exposure to the resident population existed in this area. Further assessment of toxicological data is warranted.

2) Coram, at the southern tip of the study area, also had public water supply wells with detectable levels of 1,1,1-trichlorethane. Two SCWA wells in the same wellfield (well #'s 23828 & 23827, Meehan Lane) had numerous detections of 1,1,1-trichloroethane in samples collected between 1978 and 1988. During this time period well #23827 had 14 samples with detectable levels of 1,1,1-trichloroethane. One of these samples collected on April 22, 1982 exceeded 5 mcg/L with a concentration of 6 mcg/L. Well #23828 had eight samples with detectable levels of 1,1,1-trichloroethane with the highest concentration being 4 mcg/L. One non-community water system in this region, the Coram Municipal Office Building, had a greater number of detects of 1,1,1-trichloroethane than any other well sampled in the study area. Twenty-three water samples collected from this non-community well between 1981 and 1987 had detects of 1,1,1-trichloroethane. Of these, 19 samples had concentrations greater than 5 mcg/L with a maximum concentration of 18 mcg/L being detected on February 5, 1981. There were three detects less than 1 mcg/L occurring in 1991 (2) and 1992 (1). The Coram Municipal Office Building well also had three detects of 1,1-dichloroethane, two occurring in 1984 and one occurring in 1981 that exceeded 5 mcg/L. The Coram Municipal Office Building well was inactivated in February 2003, with the building now receiving water from SCWA.

Service in the Coram area is provided by the listed wells and a number of nearby wells, none of which had exceedances for 1,1,1-trichloroethane. Sample results from the NYS DOH independent survey of water systems for locations within the distribution system showed sporadic detections at different locations and times, but did not produce any pattern of contaminants in the distribution system that could be associated with the listed wells. It is very likely that operation of the wells in the Coram area resulted in a blending of water from different sources. As a result, the likelihood of exposure is very low. No further assessment of toxicological data is warranted. With respect to the Coram Municipal Office Building, the potential for exposure to the contaminants associated with this isolated well existed. Although it is not known what portion of the population that worked at this location also lived in the CMP area, an evaluation of potential health-effects is warranted and is addressed in *Chapter V.* of this report.

3) Setauket. Several wells within the Setauket region of the study area had detects of 1,1,1-trichloroethane. Of the six wells operated by the SCWA in this region three had detects of 1,1,1-trichloroethane in samples collected between 1976 and 1988. Of these wells, well #15962 (Mud Road well #1) had 19 samples with 1,1,1-trichloroethane and of those 17 had concentrations greater than 5 mcg/L. The highest concentration recorded from this well was 13 mcg/L in a sample collected January 19, 1988. Eight detections of 1,1-dichloroethane also occurred at this well, all below 5 mcg/L. This well was subsequently taken out of service on August 10, 1988 and permanently sealed on March 9, 1994. Two other wells just outside the study region boundary but within close geographic proximity to well 15962 in the Setauket region, also showed detects of 1,1,1-trichloroethane in water samples collected between 1979 and 1989. These wells (#'s 05447 and 12430) were operated by the Soundview Association and were found to have a maximum 1,1,1-trichloroethane concentration of 8 and 16 mcg/L, respectively. These wells also had the highest levels of 1,1-dichloroethane with a total of eight detections, four of which exceeded 5 mcg/L. Records indicate that the Soundview Association wells became inactive in December 1990 and the area now is served by SCWA.

The SCWA well with relatively high frequency of detections in the Setauket area is part of a well field with two other wells. One of these adjacent wells was in production during the time frame for this analysis, while the third well was more recently installed. Three other SCWA wells located to the east of the Mud Road wellfield (at Sherry Drive) also serve the area. Sample data for the distribution system show a single exceedance along Ridgeway Avenue. Other distribution sample results in the proximity of Mud Road did not detect the contaminant. It is likely that operation of the wells in the Setauket area resulted in a blending of water from different sources that resulted in a lower level of potential exposure.

4) Sound View Association. Similar to Scott's Beach, the Sound View Association area relied on an independent water source that was not blended with other sources. This area served 236 people while it was in

operation. Although no sample results from the distribution system were available to corroborate delivery of contaminants, a conservative assessment suggests that the likelihood of exposure in this area warrants further evaluation.

5)Crystal Brook. Unlike the other three regions where 1,1,1-trichloroethane was the most frequently or only detected compound, samples from two wells within this region (#'s 51953 and 61910) were found to contain detectable levels of several VOC compounds. These two wells are part of the same well field and are operated by the SCWA. Analysis of water samples collected from these wells between 1977 and 1995 detected carbon tetrachloride, benzene, 1,1,1-trichloroethane, 1,1-dichloroethane, chloroform, tetrachloroethene, toluene, 1,2-dibromomethane, vinyl chloride, 1,2,4-trimethylbenzene, 1,2,4-trichlorobenzene and xylene. The most frequently detected compound in both wells was carbon tetrachloride. This compound was detected in 25 samples collected from well #51953 and in 11 samples collected from well #61910. Of these, the concentration of seven samples from well #51953 exceeded the MCL for carbon tetrachloride while only one sample from well #61910 was above 5 mcg/L. 1,1,1-trichlorethane was detected in nine samples collected from both wells but none of the concentrations exceeded 5 mcg/L. Benzene was detected in one sample from well #51953 and in three samples from well #61910. All three samples with detectable benzene from well #61910 had concentrations exceeding 5 mcg/L. In January, 1987, well #61910 was restricted from service and well #51953 was restricted from service in July 1988. In 1989, the SCWA installed granular activated carbon (GAC) units to treat the water from wells #51953 and #61910. Since that time there have been no detectable concentrations of any VOCs in water samples collected from either of these two wells and no MCL exceedances have occurred at these wells since that time.

The Crystal Brook wellfield does not include wells other than those listed, nor are there other SCWA wells in the immediate area to indicate that the area served by these wells would have received blended water from multiple sources. The NYS DOH pubic water system sampling data was used in this area to identify a number of MCL exceedances within the distribution system for carbon tetrachloride that may be associated with the two listed wells. Based on the distribution sample results, these wells appeared to have served the area immediately to the southwest of Crystal Brook, extending to east of Port Jefferson and portions of Port Jefferson Station. The specific area receiving water from these wells would depend on operation of adjacent wells, as blending would occur along the margins of delivery from these different sources. Based on the combination of direct well source samples with confirmed exceedances and corroborating sample results from the distribution system, the potential for exposure to the resident population was high in this area. Distribution sample data and piping infrastructure data were used to estimate areas that would have been served by these wells. A likely range in population that may have been exposed to these contaminants is between 1400 and 2200 people based on 1980 census data. In addition to the results for carbon tetrachloride, the number of other contaminants detected, along with the frequency and level of these detections, indicates that further evaluation of toxicological data is warranted.

6) *Sequoia Park, north of Coram.* The area has a single well field comprised of three wells at Strathmore Court that are operated by SCWA. One well (#47219) had eight detections of 1,2-dichloropropane, two of which were at 5 mcg/L. A second well (#52451) had one detection at 5 mcg/L while the last well (#47310) had no detections. All detections at 5 mcg/L occurred in 1981 or 1982, with more recent detections below the MCL between 1986 and 1989. Information regarding when treatment was applied to these wells was not confirmed. Though sparse in this area, the NYS DOH water systems data did not indicate detections of this contaminant. The low levels of this contaminant, plus the existence of a third well that would have likely provided blended water in the distribution system, suggests that the likelihood of exposure from these wells is relatively low. No additional evaluation of toxicological data is warranted.

Table 31. Areas and contaminant analytes integrated with toxicological data *(Chapter V. Integration)*

	Geographic area			
Analyte	Scott's Beach	Sound View Association	Crystal Brook	Coram Municipal Office Building
1,1,1-trichloroethane	X	X	X	X
1,1-dichloroethane	X	X	X	X
carbon tetrachloride			X	
Benzene			X	

f. References

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7. Private Water Supply

a. Background

While the majority of residents in the CMP area are served by public drinking water, some people use water from their own private wells. Census data from 1990 estimate that about 6%, 2000 out of a total of about 33,200 households, were served by private wells in the CMP area. Public water has been extended to more of the CMP area over time and as development has occurred. A greater number and percentage of residents now use public water supplies for drinking water than in the past.

Private drinking water wells are sometimes sampled in response to problems identified by the well owner. New York State agencies and the Suffolk County Department of Health Services (SCDHS) also sample private wells as part of investigations of specific sites or identified groundwater contamination. The SCDHS, in cooperation with the NYSDEC, has also been conducting a monitoring program to detect pesticides and their decomposition products in the groundwater of Nassau and Suffolk Counties. That program began in the late 1990s. Almost all of the private well water sampling data available and used in this investigation was collected by the SCDHS.

Water from both private and public drinking water wells is analyzed using similar, approved analytical methods. For evaluating public health implications, sample results from private wells usually are evaluated using the same drinking water standards that are applied to public drinking water.

b. Sources of Drinking Water Supply Exposure Information

NYS DOH researchers evaluated data sources to assess historical chemical contamination of private drinking water in the CMP area. The NYS DOH data set had few samples, so these were considered with the SCDHS data. SCDHS data sources include the following:

SCDHS private drinking water data (1971- 2001). Four subsets of SCDHS private drinking water quality data were reviewed: SCDHS historical (1971-1996), SCDHS recent (1996-2001), SCDHS historical pesticide (1980-1995) and SCDHS pesticide monitoring program (1998-2001). These data include water quality information from samples taken in the CMP area from 161 private drinking water wells (SCDHS historical), 324 private drinking water wells (SCDHS historical pesticide), 140 private drinking water (SCDHS recent), and 15 private drinking water wells (SCDHS pesticide monitoring program). Data from a few site-specific investigations were not included in this database, however, they were in the State's files and incorporated into this evaluation.

c. Results of Applying Screening Criteria

Each subset of the private water supply information was assessed for its use in evaluating historical environmental exposures using the criteria described in Section IV-C. The results of this evaluation are summarized in Appendix IV-1 and described in more detail below.

The SCDHS historical (1971-1996) data are temporally relevant for this investigation and useful for evaluating exposure. However, these data may not be representative of the overall quality of the private drinking water

supplies in the CMP area because the samples were usually taken in response to some concern that was identified either by the owner or by a government agency. In addition, site-specific investigations result in a clustering of samples in areas of potential contamination, which might exaggerate the degree of private well water contamination in the CMP area.

The length of time people may have been exposed to the contamination can be difficult to estimate because only one or two samples were usually taken for each well. When contamination is detected in a private well, the State and SCDHS usually recommend that the landowner connect to public water to eliminate any exposure. In many cases we do not know how quickly public water was extended to the property. We also do not know for how long the well was contaminated before the sample was collected. Since most private wells serve only one or two households, the number of people exposed per well is small.

The SCDHS historical pesticide (1980-1995) data are temporally relevant and useful for evaluating exposures. However, these wells were selected based on proximity to agricultural areas where the chemicals were used and are unlikely to represent pesticide contamination in private wells across the CMP area.

The recent SCDHS data (1996-2001) are less temporally relevant because the exposures are recent. However, the wells that were sampled during those years may have been contaminated in the past but were not sampled previously. Thus, any contamination found in these wells may have been present in the past and may be temporally relevant. The recent data has information on 107 additional analytes that were not tested for in the historical data set. We do not know how long any of the contaminants that were detected were present, although for some recently-developed chemicals, the earliest that they might have been used can be estimated. Therefore, these data were retained for further analyses.

Likewise, data from the Suffolk County Pesticide Monitoring Program (1998-2001) were reviewed but we do not know how long these chemicals were in the water. Only 15 wells in the CMP area were tested under this program. These wells were tested because they were among those most likely to contain those contaminants because of proximity to agricultural areas. Therefore, the testing results for these 15 wells may not be representative of the rest of the private wells in the CMP area. Nevertheless, these data were retained for further analyses.

d. Results

All chemicals detected in private well samples are provided in *Tables 34 and 35. Table 32* shows those chemicals that were detected in at least one sample from a well in at least 5% of the wells that were tested in the CMP area, keeping the historical and recent subsets of data separate.

Table 32. Frequency of Detection of VOCs and Pesticide Data in Private Water in the CMP Area (micrograms per liter (mcg/L)

Analyte	Number of wells with chemical detected		Number of wells tested for chemical		Percent of wells with chemical detected	
	historical	recent	historical	recent	historical	recent
1,1,1-trichloroethane	25	15	161	140	16	11
Methyl tert butyl ether (MTBE)	4	18	89	140	4	13
chloroform	15	6	161	140	9	4
Dichlorodifluoromethane	4	0	60	140	7	0
1,1-dichloroethane	8	9	150	140	5	6
Tetrachloroterephthalic acid	na	6	0	63	-	10
aldicarb	22	0	324	63	7	-
Aldicarb - sulfoxide	68	0	284	63	24	-
aldicarb - sulfone	68	0	284	63	24	-
carbofuran	41	0	292	63	14	-
alachlor	na	7	0	140	-	5
alachlor ESA	na	5	0	15	-	33
alachlor OA	na	2	0	15	-	13
metalachlor ESA	na	2	0	15	-	13

na – not analyzed

Source: (Source: SCDHS 1971-2001)

Comparison Area

As in the public water supply evaluation, data for the CMP area were compared to the rest of Suffolk County. We do not have similar data for the rest of New York State, and the SCDHS database for the remainder of Suffolk County is extensive and suitable for comparison to the CMP area. This comparison is shown in *Table 33* for each chemical that is shown in *Table 32.* We used whichever database has more data for that chemical (e.g., for 1,1,1-trichloroethane we used the historical database and for tetrachloroterephthalic acid we used the recent database). In addition to showing the frequency of detects, the average (geometric mean) is shown for all the samples analyzed for that chemical, as well as the drinking water standard for that chemical. For the calculation of an average level, one-half of the laboratory's detection limit was substituted for the value when the chemical was not detected.

Table 33. Comparison of VOC and Pesticide Contaminants in Private Drinking Water in CMP and the Remainder of Suffolk County

		CMP				Suffolk County, without CMP			
Chemical	Standard (mcg/L)	Maximum Detect (mcg/L)	Geometric Mean (mcg/L)	Percent Detect	Number of wells tested	Maximum Detect (mcg/L)	Geometric Mean (mcg/L)	Percent Detect	Number of wells tested
1,1,1-trichloroethane	5	14	0.72	16.2	161	9900	7.98	17	43345
methyl tertiary butyl ether	10	2	0.5	4.5	89	1300	1.8	9	5347
Chloroform	80	7	0.60	9.3	161	420	1.76	6	43604
Dichlorodifluoromethane	5	4	0.38	8.3	60	110	0.42	2	3418
1,1-dichloroethane	5	37	0.36	6.3	150	770	0.85	8	19425
Tetrachloroterepthalic acid	50	230	9.5	9.5	63	1054	9.72	5.6	4727
Aldicarb	3	13	0.70	7	324	515	1.54	9	30898
Aldicarb-sulfoxide	4	13	1.0	24	284	266	2.77	33	22057
Aldicarb-sulfone	2	16	1.0	24	284	153	2.82	33	22051
Carbofuran	40	17	1.6	14	292	176	1.54	27	22709
Alachlor	2	5.8	0.41	5	140	42	0.26	1	5501
Alachlor ESA	50	17.5	3.0	33	15	16.2	0.22	10	1169
Alachlor OA	50	3.28	0.55	13	15	18.2	0.24	1	1169
Metalachlor ESA	50	2.07	0.30	13	15	32.46	4.1	28	1169

(Source: SCDHS 1971-2001)

Overall, the private wells that were tested in the CMP area appear to be similar to the private wells that were tested in the remainder of Suffolk County in terms of the specific chemicals, frequency of detection and average (geometric mean) level of the contaminants. The volatile organic compounds were found in approximately the same percentage of wells and approximately the same average concentration (or slightly lower) in the CMP area compared to the rest of Suffolk County excluding the CMP area.

Further analysis

The contaminants were further evaluated based on the frequency of detection in CMP area versus Suffolk County, number of wells tested, number of wells with detections, and concentrations above standard, criteria or guidance values.

Volatile organic compounds detected in more than 5% of the wells in the CMP area were found at levels averaging below drinking water standards. I,I,I-trichloroethane was the most commonly occurring volatile organic compound. Chloroform and dichlorodifluoromethane were found slightly more frequently in CMP compared to the rest of Suffolk County, however the maximum levels detected in the CMP area were also below drinking water standards.

The pesticide alachlor and its breakdown products (ESA and OA) were found somewhat more frequently in the CMP area, but levels averaged below drinking water standards. For alachlor ESA and OA the number of wells tested is also too small to make any conclusions about the presence of these chemicals in the CMP area.

Tetrachloroterephthalic acid was detected slightly more frequently in the CMP area than the rest of Suffolk County, however, the average concentration was below NYS DOH drinking water standards. Only two wells exceeded drinking water standards.

The private drinking water wells tested for pesticides were not uniformly distributed across Suffolk County or the CMP investigation area. As a result, the higher frequency of detect of some of the pesticides and the pesticide breakdown products (e.g. aldicarb sulfoxide, aldicarb sulfone, carbofuran and alachlor ESA), for both the CMP area and the rest of Suffolk County, likely reflects the purposeful choice of sampling wells near agricultural areas. Most of the wells sampled for pesticides were in Miller Place. Because the SCDHS recommends residents connect to public water when contaminants are detected in their private drinking water wells, potential pesticide exposures through drinking water from private wells have been greatly reduced. Because sampling results do not indicate widespread or significant exposures to these pesticides, no further analysis will be done with these data.

Inorganics

Most of the inorganic compounds that were tested in the wells in the CMP area have not been a significant public health concern in private drinking water in Suffolk County. Many of the metals occur naturally in groundwater. Some of the metals do not have drinking water standards. Few samples exceeded any drinking water guidelines. Data on metals and other inorganic compounds will not be further analyzed.

Nitrates

Because nitrates are produced primarily by human activity (e.g. fertilizers and septic waste), these data are presented in *Table 34.* Data for the CMP area were compared to the rest of Suffolk County. Overall, nitrates in the CMP area are similar, or a little lower than the rest of Suffolk County. No further analysis will be done with these data.

Table 34. Comparison of Nitrates in Private Water in CMP and Remainder of Suffolk County

		CMP				Suffolk County			
Chemical	**Standard (mg/L)**	**Maximum Detect (mg/L)**	**Geometric Mean (mg/L)**	**Percent Detect**	**Number of wells tested**	**Maximum Detect (mg/L)**	**Geometric Mean (mg/L)**	**Percent Detect**	**Number of wells tested**
nitrates –historical	10	15.7	3.8	90	150	132	4.5	82	46134
nitrates –recent	10	4.7	3.4	80	5	22.2	5.2	89	323

(Source SCWD 1971-2001)

Based on the private drinking water data review discussed above, no chemicals are being passed to integration because of their presence in private drinking water wells.

Table 35. Private Drinking Water Sample Results in the CMP Area Reported in Micrograms per Liter (mcg/L) Unless Otherwise Noted (Recent Data 1996-2001)

Sorted first by data set (recent, historical) then by analytes with at least one detect above drinking water guidelines, analytes with detects below drinking water guidelines, finally by analytes with no detections, and in each category, analytes are sorted alphabetically.

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
1,1,1-Trichloroethane	5	0.5	6	0.4	15	140	11
Alachlor	2	0.3	5.8	0.4	7	140	5
Iron	0.3*	0.1	22	0.5	69	140	49
Iron + Manganese (Combined, Calc)	0.5*	0.001	22	0.5	129	140	92
Lead	15	1	21.5	1.6	62	140	44
Methyl-tert-butyl-ether (MTBE)	10	0.5	13	0.6	18	140	13
Nitrites + Nitrates	10*	0.2	18	3.8	125	136	92
Tetrachloroterephthalic acid	50	10	230	10	6	63	10
Trichloroethene	5	0.9	27	0.4	5	140	4
1,1-Dichloroethane	5	0.5	3	0.3	9	140	6
1,1-Dichloroethene	5	1	1	0.3	2	140	1
1,2-Dichloropropane	5	2	2	0.3	1	140	1
Alachlor ESA	50	0.8	17.5	3.1	5	15	33
Alachlor OA	50	2.4	3.3	0.5	2	15	13
Aluminum		5.1	325	20.9	90	140	64
Ammonia		20	9740	127	22	140	16
Arsenic	50	2.9	3.3	1	3	140	2
Barium	2000	1.4	144	27.3	133	140	95
Bromide		0.4	0.4	0.1	1	5	20
Bromodichloromethane	80	0.5	0.5	0.3	1	140	1
Chlordane	2	1.2	1.2	0.5	1	140	1
Chloride	250*	0.004	0.2	0.03	137	137	100
Chlorodifluoromethane		0.7	15	0.4	3	140	2
Chloroform	80	1	2	0.3	6	140	4
Chromium	100	1	32	3.1	132	140	94
cis-1,2-Dichloroethene	5	0.7	2	0.3	3	140	2
Cobalt		1.1	3.8	0.5	2	140	1
Copper	1300	1.1	810	116.4	136	140	97

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Gross beta (PC/L)		1.5	3.9	1.4	4	14	29
Manganese	300	1.1	247	20.6	26	140	19
Mercury	2	0.4	0.4	0.4	1	135	1
Metolachlor ESA (CGA-354743)	50	0.5	2.1	0.3	2	15	13
Molybdenum		1.1	2.6	0.5	2	140	1
Nickel	100	1.0	16.4	1.8	96	140	69
Nitrate	10*	0.001	0.005	0.003	4	5	80
Nitrite	1*	0.02	0.18	0.01	12	133	9
Selenium	50	2	2.2	1	3	140	2
Silver	100	0.5	3	2.5	9	140	6
Sodium	no limit**	1900	126000	17166	140	140	100
Sulfate	250*	4	66	20.1	134	140	96
Surfactants-MBAS		400	400	113.6	1	4	25
Tetrachloroethene	5	0.5	4	0.3	7	140	5
Thallium	2	1.2	1.9	0.5	2	140	1
Thorium		1.1	11	0.6	3	140	2
Titanium		1	3.1	0.7	37	140	26
Toluene	5	0.5	2	0.3	3	140	2
Vanadium		1	5.1	0.9	53	140	38
Zinc	5000	0.5	11	0.6	3	140	2
1,1-Dichloropropene	5	nd ***	nd	nd	0	140	0
1,1,1,2-Tetrachloroethane	5	nd	nd	nd	0	140	0
1,1,2-Trichloroethane	5	nd	nd	nd	0	140	0
1,1,2,2-Tetrachloroethane	5	nd	nd	nd	0	140	0
1,2-Dichlorobenzene (o)	5	nd	nd	nd	0	140	0
1,2-Dichloroethane	5	nd	nd	nd	0	140	0
1,2,3-Trichlorobenzene	5	nd	nd	nd	0	140	0
1,2,3-Trichloropropane	5	nd	nd	nd	0	140	0
1,2,4-Trichlorobenzene	5	nd	nd	nd	0	140	0
1,2,4-Trimethylbenzene	5	nd	nd	nd	0	140	0
1,2,4,5-Tetramethylbenzene		nd	nd	nd	0	140	0
1,2-dibromo-3-chloropropane	0.2	nd	nd	nd	0	140	0
1,2-dibromoethane	5	nd	nd	nd	0	140	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
1,3-Dichloropropane	5	nd	nd	nd	0	140	0
1,3,5-Trimethylbenzene	5	nd	nd	nd	0	140	0
1,4-Dichlorobutane	5	nd	nd	nd	0	135	0
1-Bromo-2-Chloroethane	5	nd	nd	nd	0	140	0
1-Methylethylbenzene	5	nd	nd	nd	0	140	0
1-Naphthol	50	nd	nd	nd	0	63	0
2,2-Dichloropropane	5	nd	nd	nd	0	140	0
2,3-Dichloropropene	5	nd	nd	nd	0	140	0
2,4,5-T	50	nd	nd	nd	0	18	0
2,4,5-TP (SILVEX)	10	nd	nd	nd	0	18	0
2,4-D	50	nd	nd	nd	0	18	0
2,4-DB		nd	nd	nd	0	18	0
2-Bromo-1-Chloropropane	5	nd	nd	nd	0	140	0
2-Butanone (MEK)	50	nd	nd	nd	0	140	0
2-Chlorotoluene	5	nd	nd	nd	0	140	0
2-HydroxyAtrazine (G-34048)	50	nd	nd	nd	0	15	0
3,5-Dichlorobenzoic Acid	50	nd	nd	nd	0	18	0
3-Chlorotoluene		nd	nd	nd	0	140	0
3-Hydroxycarbofuran	50	nd	nd	nd	0	63	0
4,4-DDD	5	nd	nd	nd	0	140	0
4,4-DDE	5	nd	nd	nd	0	140	0
4,4-DDT	5	nd	nd	nd	0	140	0
4-Chlorotoluene	5	nd	nd	nd	0	140	0
4-Nitrophenol	50	nd	nd	nd	0	18	0
Acetochlor		nd	nd	nd	0	22	0
Acifluorfen	50	nd	nd	nd	0	18	0
Acrylonitrile		nd	nd	nd	0	5	0
Aldicarb	3	nd	nd	nd	0	63	0
Aldicarb-Sulfone	2	nd	nd	nd	0	63	0
Aldicarb-Sulfoxide	4	nd	nd	nd	0	63	0
Aldrin	5	nd	nd	nd	0	140	0
Allyl chloride		nd	nd	nd	0	5	0
alpha-BHC	5	nd	nd	nd	0	140	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Antimony	6	nd	nd	nd	0	140	0
Atrazine	3	nd	nd	nd	0	39	0
Azoxystrobin		nd	nd	nd	0	1	0
Benfluralin	50	nd	nd	nd	0	17	0
Bentazon		nd	nd	nd	0	18	0
Benzene	5	nd	nd	nd	0	140	0
Benzo(a)pyrene	0.2	nd	nd	nd	0	39	0
Beryllium	4	nd	nd	nd	0	140	0
beta-BHC	5	nd	nd	nd	0	140	0
Bis(2-ethylhexyl)adipate	50	nd	nd	nd	0	39	0
Bis(2-ethylhexyl)phthalate	6	nd	nd	nd	0	38	0
Bloc	50	nd	nd	nd	0	14	0
Bromacil		nd	nd	nd	0	39	0
Bromobenzene	5	nd	nd	nd	0	127	0
Bromochloromethane	5	nd	nd	nd	0	127	0
Bromoform	80	nd	nd	nd	0	138	0
Bromomethane	5	nd	nd	nd	0	140	0
Butachlor		nd	nd	nd	0	41	0
Cadmium	5	nd	nd	nd	0	140	0
Carbaryl		nd	nd	nd	0	63	0
Carbofuran	40	nd	nd	nd	0	63	0
Carbon Disulfide		nd	nd	nd	0	5	0
Carbon Tetrachloride	5	nd	nd	nd	0	140	0
Chloramben	50	nd	nd	nd	0	18	0
Chlorobenzene	5	nd	nd	nd	0	140	0
Chlorodibromomethane		nd	nd	nd	0	140	0
Chloroethane	5	nd	nd	nd	0	140	0
Chloromethane	5	nd	nd	nd	0	140	0
Chlorothalonil	5	nd	nd	nd	0	17	0
Chlorpyriphos		nd	nd	nd	0	22	0
cis-1,3-Dichloropropene	5	nd	nd	nd	0	140	0
Cyanazine		nd	nd	nd	0	14	0
Cyfluthrin		nd	nd	nd	0	17	0
Dacthal		nd	nd	nd	0	140	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Deisopropylatrazine (G-28279)	50	nd	nd	nd	0	15	0
delta-BHC	5	nd	nd	nd	0	140	0
Desethylatrazine (G-30033)	50	nd	nd	nd	0	15	0
Diazinon		nd	nd	nd	0	39	0
Dibromomethane	5	nd	nd	nd	0	140	0
Dicamba		nd	nd	nd	0	18	0
Dichlorbenil		nd	nd	nd	0	14	0
Dichlorodifluoromethane	5	nd	nd	nd	0	140	0
Dichloroprop	50	nd	nd	nd	0	18	0
Didealkylatrazine (G-28273)		nd	nd	nd	0	15	0
Dieldrin	5	nd	nd	nd	0	140	0
Diethyl Ether	50	nd	nd	nd	0	5	0
Dimethyldisulfide	50	nd	nd	nd	0	135	0
Dinoseb	7	nd	nd	nd	0	31	0
Dissolved Phosphate		nd	nd	nd	0	4	0
Disulfoton		nd	nd	nd	0	22	0
Disulfoton sulfone		nd	nd	nd	0	14	0
d-Limonene		nd	nd	nd	0	5	0
Endosulfan I	50	nd	nd	nd	0	140	0
Endosulfan II	50	nd	nd	nd	0	140	0
Endosulfan Sulfate	50	nd	nd	nd	0	44	0
Endrin	2	nd	nd	nd	0	140	0
Endrin aldehyde	5	nd	nd	nd	0	140	0
EPTC	50	nd	nd	nd	0	14	0
Ethenylbenzene (Styrene)	5	nd	nd	nd	0	140	0
Ethofumesate		nd	nd	nd	0	22	0
Ethyl Parathion	50	nd	nd	nd	0	1	0
Ethylbenzene	5	nd	nd	nd	0	140	0
Ethylmethacrylate		nd	nd	nd	0	5	0
Fluoride	2200	nd	nd	nd	0	7	0
Freon 113		nd	nd	nd	0	140	0
gamma-BHC (Lindane)	0.2	nd	nd	nd	0	140	0
Gross alpha E	5	nd	nd	nd	0	14	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Heptachlor	0.4	nd	nd	nd	0	140	0
Heptachlor epoxide	0.2	nd	nd	nd	0	140	0
Hexachlorobenzene	1	nd	nd	nd	0	39	0
Hexachlorobutadiene	5	nd	nd	nd	0	140	0
Hexachlorocyclopentadiene	5	nd	nd	nd	0	39	0
Imidacloprid		nd	nd	nd	0	15	0
Iodofenphos		nd	nd	nd	0	17	0
Iprodione		nd	nd	nd	0	17	0
Isofenphos		nd	nd	nd	0	22	0
Isopropyltoluene (p-Cymene)		nd	nd	nd	0	140	0
Kelthane	50	nd	nd	nd	0	14	0
m,p-Dichlorobenzene		nd	nd	nd	0	140	0
Malaoxon	50	nd	nd	nd	0	1	0
Malathion		nd	nd	nd	0	22	0
MCPA	50	nd	nd	nd	0	18	0
MCPP		nd	nd	nd	0	18	0
Metalaxyl		nd	nd	nd	0	29	0
Methacrylonitrile		nd	nd	nd	0	5	0
Methiocarb	50	nd	nd	nd	0	63	0
Methomyl		nd	nd	nd	0	63	0
Methoprene	50	nd	nd	nd	0	14	0
Methoxychlor	40	nd	nd	nd	0	140	0
Methyl isothiocyanate		nd	nd	nd	0	5	0
Methyl Methacrylate	50	nd	nd	nd	0	5	0
Methyl parathion		nd	nd	nd	0	1	0
Methyl Sulfide		nd	nd	nd	0	140	0
Methylene Chloride	5	nd	nd	nd	0	140	0
Metolachlor		nd	nd	nd	0	39	0
Metolachlor metabolite (CGA-3773)		nd	nd	nd	0	15	0
Metolachlor metabolite (CGA-4017)		nd	nd	nd	0	15	0
Metolachlor metabolite (CGA-4163)		nd	nd	nd	0	15	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Metolachlor metabolite (CGA-6712)		nd	nd	nd	0	15	0
Metolachlor OA (CGA-51202)		nd	nd	nd	0	15	0
Metribuzin	50	nd	nd	nd	0	39	0
Monomethyltetrachloroterephthalate	50	nd	nd	nd	0	63	0
m-Xylene	5	nd	nd	nd	0	140	0
Naphthalene	50	nd	nd	nd	0	135	0
Napropamide		nd	nd	nd	0	14	0
n-Butylbenzene	5	nd	nd	nd	0	140	0
n-Propylbenzene	5	nd	nd	nd	0	140	0
Orthophosphate		nd	nd	nd	0	6	0
Oxamyl	50	nd	nd	nd	0	63	0
o-Xylene	5	nd	nd	nd	0	140	0
p-Diethylbenzene		nd	nd	nd	0	140	0
Pendimethalin		nd	nd	nd	0	17	0
Pentachlorobenzene		nd	nd	nd	0	22	0
Pentachloronitrobenzene		nd	nd	nd	0	17	0
Pentachlorophenol	1	nd	nd	nd	0	18	0
Perchlorate		nd	nd	nd	0	8	0
Permethrin		nd	nd	nd	0	17	0
Picloram	50	nd	nd	nd	0	18	0
Piperonyl butoxide	50	nd	nd	nd	0	1	0
Prometon	50	nd	nd	nd	0	36	0
Propachlor		nd	nd	nd	0	39	0
Propiconazole (Tilt)		nd	nd	nd	0	1	0
Propoxur (Baygon)	50	nd	nd	nd	0	63	0
p-Xylene	5	nd	nd	nd	0	140	0
Resmethrin	50	nd	nd	nd	0	14	0
sec-Butylbenzene	5	nd	nd	nd	0	140	0
Simazine	4	nd	nd	nd	0	39	0
Sumithrin		nd	nd	nd	0	14	0
T. Chlorotoluene		nd	nd	nd	0	140	0
Tebuthiuron	50	nd	nd	nd	0	22	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Terbacil		nd	nd	nd	0	14	0
Terbufos		nd	nd	nd	0	14	0
tert-Amyl-Methyl-Ether		nd	nd	nd	0	101	0
tert-Butylbenzene	5	nd	nd	nd	0	140	0
tert-Butyl-Ethyl-Ether	50	nd	nd	nd	0	101	0
Tetrahydrofuran		nd	nd	nd	0	140	0
Total Aldicarb (calc)	3	nd	nd	nd	0	63	0
Total Organic Carbon		nd	nd	nd	0	4	0
Total Triazines + Metabolites		nd	nd	nd	0	39	0
Total Xylenes	5	nd	nd	nd	0	140	0
trans 1,2 Dichloroethene	5	nd	nd	nd	0	140	0
trans-1,3-Dichloropropene	5	nd	nd	nd	0	140	0
Tridimefon	50	nd	nd	nd	0	17	0
Trichlorofluoromethane	5	nd	nd	nd	0	140	0
Trifluralin		nd	nd	nd	0	17	0
Tritium		nd	nd	nd	0	14	0
Vinclozolin		nd	nd	nd	0	17	0
Vinyl Chloride	2	nd	nd	nd	0	140	0
1,1,1-trichloroethane	5	0.2	420	8.8	25	161	16
1,1-dichloroethane	5	0.1	7	1.2	8	150	17
1,2,4-trimethylbenzene	5	10	10	1.8	1	117	1
1,2-dichlorobenzene-o	5	9	9	2.2	1	113	1
1,2-dichloropropane	5	25	25	1.7	1	29	3
1,3,5-trimethylbenzene	5	15	15	1.8	1	106	1
Aldicarb	3	1	13	0.7	22	324	7
Aldicarb-Sulfone	2	1	16	1	68	284	24
Aldicarb-Sulfoxide	4	1	13	1	68	284	24
Benzene	5	77	110	3	2	134	1
Chloride	250*	0.02	297	22	150	150	100
cis-1,2-dichloroethene	5	5	18	2	2	94	2
Copper	1300	100	21000	296.6	67	150	45
Ethylbenzene	5	7	7	1.8	1	131	1
Iron	0.3*	0.1	29	0.7	103	149	69

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Lead	15	1	63.9	3.7	68	123	55
Manganese	0.3*	0.05	17	0.2	36	149	24
Nitrate	10*	0.2	15.7	3.4	136	150	91
Sulfate	250*	2	3670	203.5	149	149	100
Tetrachloroethene	5	0.4	610	10.4	15	174	9
Toluene	5	11	11	1.9	1	134	1
Total Aldicarb (Calc)	3	1	29	5.3	95	95	100
total xylenes	5	53	53	26.6	1	2	50
Trichloroethene	5	4	340	7	10	173	6
Zinc	5000	400	14000	881	51	150	34
1,1-dichloroethene	5	4	4	1.0	1	28	4
Ammonia		20	7360	10	34	150	23
Barium	2000	8.4	8.4	8.4	1	1	100
Bromoform		0.1	0.1	2.2	1	152	1
Cadmium	5	1.1	1.1	1.0	1.1	120	1
Carbofuran	40	1	17	1.6	41	292	14
Chloroform	80	1	18	2.3	15	161	9
Dichlorodifluoromethane	5	0.5	4	0.4	5	60	8
Freon 113		4	4	1.8	1	172	1
m,p-dichlorobenzene	5	9	9	2.3	1	112	1
Methyl-tertiary-butyl-ether (MTBE)	10	0.5	2	5	4	89	4
Nitrite	1*	0.06	0.07	0.01	3	57	5
Sodium	No limit**	1800	155000	16603	150	150	100
Surfactants MBAS		0.3	0.4	0.1	3	52	6
T Chromium		2.14	3	4.6	2	11	18
Total Conductivity		43	3670	206	150	150	100
1,1,1,2-tetrachloroethane	5	nd	nd	nd	0	31	0
1,1,1,2-tetrachloropropane	5	nd	nd	nd	0	27	0
1,1,2,2-tetrachloroethane	5	nd	nd	nd	0	83	0
1,1,2-trichloroethane	5	nd	nd	nd	0	104	0
1,2,2,3-tetrachloropropane	5	nd	nd	nd	0	28	0
1,2,3-trichlorobenzene	5	nd	nd	nd	0	81	0
1,2,3-trichloropropane	5	nd	nd	nd	0	26	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
1,2,4,5-Tetramethylbenzene		nd	nd	nd	0	92	0
1,2,4,-trichlorobenzene	5	nd	nd	nd	0	81	0
1,2-dibromo-3-dichloropropane	5	nd	nd	nd	0	1	0
1,2-dibromoethane	5	nd	nd	nd	0	30	0
1,2-dichloroethane	5	nd	nd	nd	0	29	0
1,3-dichloropropane	5	nd	nd	nd	0	1	0
1-Bromo-2-chloroethane	5	nd	nd	nd	0	29	0
1-methylethylbenzene	5	nd	nd	nd	0	1	0
1-Naphthol	50	nd	nd	nd	0	112	0
2,2-dichloropropane	5	nd	nd	nd	0	1	0
2,3-dichloropropene	5	nd	nd	nd	0	29	0
2-Bromo 3-Chloropropane	5	nd	nd	nd	0	27	0
2-butanone (MEK)	50	nd	nd	nd	0	1	0
2-chlorotoluene	5	nd	nd	nd	0	118	0
3-chlorotoluene		nd	nd	nd	0	118	0
3-Hydroxycarbofuran	50	nd	nd	nd	0	448	0
4-chlorotoluene	5	nd	nd	nd	0	118	0
Acenaphthene	50	nd	nd	nd	0	3	0
Anthracene	50	nd	nd	nd	0	3	0
Arsenic	50	nd	nd	nd	0	10	0
Benzo(A)Anthracene	50	nd	nd	nd	0	3	0
Benzo(a)pyrene	0.2	nd	nd	nd	0	3	0
Benzo(B)Fluoranthene	50	nd	nd	nd	0	3	0
Benzo(GHI)Perylene	50	nd	nd	nd	0	3	0
Benzo(K)Fluoranthene	50	nd	nd	nd	0	3	0
Bromobenzene	5	nd	nd	nd	0	126	0
Bromochloromethane	5	nd	nd	nd	0	16	0
Bromodichloromethane	80	nd	nd	nd	0	172	0
Carbaryl		nd	nd	nd	0	447	0
Carbon Tetrachloride	5	nd	nd	nd	0	174	0
Chlorobenzene	5	nd	nd	nd	0	132	0
Chlorodibromomethane		nd	nd	nd	0	154	0
Chlorodifluoromethane		nd	nd	nd	0	1	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Chrysene	50	nd	nd	nd	0	3	0
cis-1,3-dichloropropene	5	nd	nd	nd	0	27	0
Dibenzo(A,H)Anthracene	50	nd	nd	nd	0	3	0
Dibromomethane	5	nd	nd	nd	0	28	0
Ethylbenzene-styrene	5	nd	nd	nd	0	1	0
Fluoranthene	50	nd	nd	nd	0	3	0
Fluorene	50	nd	nd	nd	0	3	0
Fluoride	2200	nd	nd	nd	0	4	0
Hexachlorobutadiene	5	nd	nd	nd	0	1	0
Indo(1,2,3-CD)Pyrene	50	nd	nd	nd	0	3	0
Isopropyltoluene-p-Cymene		nd	nd	nd	0	1	0
Mercury	2	nd	nd	nd	0	1	0
Methiocarb	50	nd	nd	nd	0	32	0
Methomyl		nd	nd	nd	0	405	0
Methylene Chloride	5	nd	nd	nd	0	29	0
m-xylene	5	nd	nd	nd	0	133	0
Naphthalene	50	nd	nd	nd	0	3	0
n-butylbenzene	5	nd	nd	nd	0	1	0
n-propylbenzene	5	nd	nd	nd	0	1	0
Oxamyl	50	nd	nd	nd	0	448	0
o-xylene	5	nd	nd	nd	0	133	0
p-diethylbenzene		nd	nd	nd	0	101	0
Phenanthrene	50	nd	nd	nd	0	3	0
Propoxur (Baygon)	50	nd	nd	nd	0	30	0
p-xylene	5	nd	nd	nd	0	133	0
Pyrene	50	nd	nd	nd	0	3	0
sec-butylbenzene	5	nd	nd	nd	0	1	0
Selenium	50	nd	nd	nd	0	11	0
Silver	100	nd	nd	nd	0	11	0
T-chlorotoluene		nd	nd	nd	0	1	0
tert-Butylbenzene	5	nd	nd	nd	0	1	0
Tetrahydrofuran		nd	nd	nd	0	1	0
trans 1,2-dichloroethene	5	nd	nd	nd	0	29	0
trans-1,3-dichloropropene	5	nd	nd	nd	0	27	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Vinyl Chloride	2	nd	nd	nd	0	27	0

**Value reported in mg/L*

*** Sodium is naturally occurring in groundwater and can also come from a variety of man-made sources, most commonly road salting and water softener backwash. Although there is no maximum contaminant level for the general population, the NYSDOH recommends a limit of 20,000 mcg/L for people on severely restricted diets and 270,000 mcg/L for people on moderately restricted diets.*

**** nd indicates that analyte was not detected in sample*

*****The geometric mean was calculated using actual detected values, and when the analyte was not detected, half of the detection limit was substituted in for the value.*

Table 36. Private Drinking Water Sample Results in the CMP Area Reported in Micrograms per Liter (mcg/L) Unless Otherwise Noted (Historical Data 1971-1996)

Sorted first by data set (recent, historical) then by analytes with at least one detect above drinking water guidelines, analytes with detects below drinking water guidelines, finally by analytes with no detections, and in each category, analytes are sorted alphabetically.

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
1,1,1-trichloroethane	5	0.2	420	8.8	25	161	16
1,1-dichloroethane	5	0.1	7	1.2	8	150	17
1,2,4-trimethylbenzene	5	10	10	1.8	1	117	1
1,2-dichlorobenzene-o	5	9	9	2.2	1	113	1
1,2-dichloropropane	5	25	25	1.7	1	29	3
1,3,5-trimethylbenzene	5	15	15	1.8	1	106	1
Aldicarb	3	1	13	0.7	22	324	7
Aldicarb-Sulfone	2	1	16	1	68	284	24
Aldicarb-Sulfoxide	4	1	13	1	68	284	24
Benzene	5	77	110	3	2	134	1
Chloride	250*	0.02	297	22	150	150	100
cis-1,2-dichloroethene	5	5	18	2	2	94	2
Copper	1300	100	21000	296.6	67	150	45
Ethylbenzene	5	7	7	1.8	1	131	1
Iron	0.3*	0.1	29	0.7	103	149	69
Lead	15	1	63.9	3.7	68	123	55
Manganese	0.3*	0.05	17	0.2	36	149	24

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Nitrate	10*	0.2	15.7	3.4	136	150	91
Sulfate	250*	2	3670	203.5	149	149	100
Tetrachloroethene	5	0.4	610	10.4	15	174	9
Toluene	5	11	11	1.9	1	134	1
Total Aldicarb (Calc)	3	1	29	5.3	95	95	100
total xylenes	5	53	53	26.6	1	2	50
Trichloroethene	5	4	340	7	10	173	6
Zinc	5000	400	14000	881	51	150	34
1,1-dichloroethene	5	4	4	1.0	1	28	4
Ammonia		20	7360	10	34	150	23
Barium	2000	8.4	8.4	8.4	1	1	100
Bromoform		0.1	0.1	2.2	1	152	1
Cadmium	5	1.1	1.1	1.0	1.1	120	1
Carbofuran	40	1	17	1.6	41	292	14
Chloroform	80	1	18	2.3	15	161	9
Dichlorodifluoromethane	5	0.5	4	0.4	5	60	8
Freon 113		4	4	1.8	1	172	1
m,p-dichlorobenzene	5	9	9	2.3	1	112	1
Methyl-tertiary-butyl-ether (MTBE)	10	0.5	2	5	4	89	4
Nitrite	1*	0.06	0.07	0.01	3	57	5
Sodium	No limit**	1800	155000	16603	150	150	100
Surfactants MBAS		0.3	0.4	0.1	3	52	6
T Chromium		2.14	3	4.6	2	11	18
Total Conductivity		43	3670	206	150	150	100
1,1,1,2-tetrachloroethane	5	nd	nd	nd	0	31	0
1,1,1,2-tetrachloropropane	5	nd	nd	nd	0	27	0
1,1,2,2-tetrachloroethane	5	nd	nd	nd	0	83	0
1,1,2-trichloroethane	5	nd	nd	nd	0	104	0
1,2,2,3-tetrachloropropane	5	nd	nd	nd	0	28	0
1,2,3-trichlorobenzene	5	nd	nd	nd	0	81	0
1,2,3-trichloropropane	5	nd	nd	nd	0	26	0
1,2,4,5-Tetramethylbenzene		nd	nd	nd	0	92	0
1,2,4,-trichlorobenzene	5	nd	nd	nd	0	81	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
1,2-dibromo-3-dichloropropane	5	nd	nd	nd	0	1	0
1,2-dibromoethane	5	nd	nd	nd	0	30	0
1,2-dichloroethane	5	nd	nd	nd	0	29	0
1,3-dichloropropane	5	nd	nd	nd	0	1	0
1-Bromo-2-chloroethane	5	nd	nd	nd	0	29	0
1-methylethylbenzene	5	nd	nd	nd	0	1	0
1-Naphthol	50	nd	nd	nd	0	112	0
2,2-dichloropropane	5	nd	nd	nd	0	1	0
2,3-dichloropropene	5	nd	nd	nd	0	29	0
2-Bromo 3-Chloropropane	5	nd	nd	nd	0	27	0
2-butanone (MEK)	50	nd	nd	nd	0	1	0
2-chlorotoluene	5	nd	nd	nd	0	118	0
3-chlorotoluene		nd	nd	nd	0	118	0
3-Hydroxycarbofuran	50	nd	nd	nd	0	448	0
4-chlorotoluene	5	nd	nd	nd	0	118	0
Acenaphthene	50	nd	nd	nd	0	3	0
Anthracene	50	nd	nd	nd	0	3	0
Arsenic	50	nd	nd	nd	0	10	0
Benzo(A)Anthracene	50	nd	nd	nd	0	3	0
Benzo(a)pyrene	0.2	nd	nd	nd	0	3	0
Benzo(B)Fluoranthene	50	nd	nd	nd	0	3	0
Benzo(GHI)Perylene	50	nd	nd	nd	0	3	0
Benzo(K)Fluoranthene	50	nd	nd	nd	0	3	0
Bromobenzene	5	nd	nd	nd	0	126	0
Bromochloromethane	5	nd	nd	nd	0	16	0
Bromodichloromethane	80	nd	nd	nd	0	172	0
Carbaryl		nd	nd	nd	0	447	0
Carbon Tetrachloride	5	nd	nd	nd	0	174	0
Chlorobenzene	5	nd	nd	nd	0	132	0
Chlorodibromomethane		nd	nd	nd	0	154	0
Chlorodifluoromethane		nd	nd	nd	0	1	0
Chrysene	50	nd	nd	nd	0	3	0
cis-1,3-dichloropropene	5	nd	nd	nd	0	27	0

ANALYTE	Standard, Criteria or Guidance Value if any	Minimum Detected Value	Maximum Detected Value	Geometric Mean with half detection limit****	Number of Wells with Detections	Number of Wells Analyzed	% Detect
Dibenzo(A,H)Anthracene	50	nd	nd	nd	0	3	0
Dibromomethane	5	nd	nd	nd	0	28	0
Ethylbenzene-styrene	5	nd	nd	nd	0	1	0
Fluoranthene	50	nd	nd	nd	0	3	0
Fluorene	50	nd	nd	nd	0	3	0
Fluoride	2200	nd	nd	nd	0	4	0
Hexachlorobutadiene	5	nd	nd	nd	0	1	0
Indo(1,2,3-CD)Pyrene	50	nd	nd	nd	0	3	0
Isopropyltoluene-p-Cymene		nd	nd	nd	0	1	0
Mercury	2	nd	nd	nd	0	1	0
Methiocarb	50	nd	nd	nd	0	32	0
Methomyl		nd	nd	nd	0	405	0
Methylene Chloride	5	nd	nd	nd	0	29	0
m-xylene	5	nd	nd	nd	0	133	0
Naphthalene	50	nd	nd	nd	0	3	0
n-butylbenzene	5	nd	nd	nd	0	1	0
n-propylbenzene	5	nd	nd	nd	0	1	0
Oxamyl	50	nd	nd	nd	0	448	0
o-xylene	5	nd	nd	nd	0	133	0
p-diethylbenzene		nd	nd	nd	0	101	0
Phenanthrene	50	nd	nd	nd	0	3	0
Propoxur (Baygon)	50	nd	nd	nd	0	30	0
p-xylene	5	nd	nd	nd	0	133	0
Pyrene	50	nd	nd	nd	0	3	0
sec-butylbenzene	5	nd	nd	nd	0	1	0
Selenium	50	nd	nd	nd	0	11	0
Silver	100	nd	nd	nd	0	11	0
T-chlorotoluene		nd	nd	nd	0	1	0
tert-Butylbenzene	5	nd	nd	nd	0	1	0
Tetrahydrofuran		nd	nd	nd	0	1	0
trans 1,2-dichloroethene	5	nd	nd	nd	0	29	0
trans-1,3-dichloropropene	5	nd	nd	nd	0	27	0
Vinyl Chloride	2	nd	nd	nd	0	27	0

**Value reported in mg/L*

*** Sodium is naturally occurring in groundwater and can also come from a variety of man-made sources, most commonly road salting and water softener backwash. Although there is no maximum contaminant level for the general population, the NYSDOH recommends a limit of 20,000 mcg/L for people on severely restricted diets and 270,000 mcg/L for people on moderately restricted diets.*

**** nd indicates that analyte was not detected in sample*

*****The geometric mean was calculated using actual detected values, and when the analyte was not detected, half of the detection limit was substituted in for the value.*

8. Electromagnetic Fields (EMFs)

Several residents expressed concerns about exposure to EMFs as a possible cause for the increased incidence of breast cancer. EMF stands for electromagnetic fields and they occur wherever there is electricity. EMFs weaken with distance from the source. Sources include electric power lines, household wiring, and everyday appliances such as clothes dryers, electric blankets, water beds, hair dryers, toasters, stoves and televisions. Limited information is available to evaluate levels of EMFs in the CMP area.

To evaluate whether transmission lines from the power plant result in a higher density of transmission lines in CMP compared to other areas, the environmental exposure team reviewed the New York State Electric Transmission lines database. This database was created in 1997 by the NYS Department of Public Service. It contains New York State electric transmission lines of 115 kilovolts and above. Researchers compared total miles of transmission lines, miles of transmission lines per person and miles of transmission lines per square mile of land in the CMP area with other parts of Suffolk County. The CMP area had fewer miles of transmission lines and approximately the same number of miles per person and miles per square mile of land as other parts of Suffolk County. Based on this analysis of major transmission lines, researchers did not find evidence that people in the CMP area were exposed to higher levels of EMFs than people in other areas. No further evaluation will be done with these data.

9. Other Data Sources Listed in the Initial Environmental Inventory

A variety of data sources that were listed on the *Initial Environmental Inventory (Appendix IV-2)* contained information that was too limited to estimate environmental exposures in the CMP area. These data sets included New York State Major Oil Storage Facilities, New York State Wastewater Discharges (PCS), Spill Incidents, NYS DOH Fish Consumption Advisories, and Large Quantity Hazardous Waste Generators. A description of these data sets, including identification of what was reported for the CMP area, was provided to the communities at the June 2002 public meeting. A copy of that document is available on NYS DOH's web site at http://www.health.state.ny.us/nysdoh/cancer/sublevel/envinven.htm. The evaluation of these data sets is provided in *Appendix IV-1.*

10. Summary Results

As part of this environmental exposure evaluation, a variety of existing data sets were assessed to determine if environmental contaminants in the CMP area were higher than other parts of New York State, or if areas of contamination existed in the CMP area that should be explored further. Based on these data, researchers found that environmental quality in the CMP area is similar, or even above average, compared to the rest of New York State. Although some contaminants were found to be higher in the CMP environment than in some of the comparison areas, these differences are within the range of what environmental scientists would expect to find when doing these kinds of analyses in communities around New York State. As a result, those chemicals are being assessed further to determine whether the levels present could have contributed to breast cancer incidence or other non-cancer health effects in the CMP area. The integration of environmental exposure and toxicological information is described further in *Chapter V. Integration*. A summary list of contaminants that have been assessed further is provided in *Table 37.*

Table 37. Summary of contaminants Integrated with toxicological data in Chapter V-B and V-C

Contaminant	Environmental exposure evaluated	Health risk previously evaluated in Working Draft Report *(Chapter VB)*	Health risk evaluated for Final Report (Chapter VC)
Air contaminants			
Ethylene thiourea	X	X	
Acrylic acid	X		X
Methyl tert-butyl ether (MTBE)	X		X
Propionaldehyde	X		X
1,2,4-Trichlorobenzene	X		X
Methylene diphenyl diisocyanate	X		X
Acetaldehyde	X		X
Hydrofluoric acid	X		X
Methyl ethyl ketone	X		X
Dimethyl phthalate	X		X
Beryllium	X		X
Diethanolamine	X		X
Aniline	X		X
Trichloroethene	X		X
1,1,1-Trichloroethane	X		X
Hydrochloric acid	X		X
Arsenic	X		X
1,3-Dichloropropene	X		X
Glycol ethers	X		X
Acrylamide	X		X
1,1-Dichloroethene	X		X
1,2-Dibromoethane	X		X
Ethylene oxide	X		X
Diesel particulate matter	X		X
Cadmium	X		X
Ozone	X		X
Pesticides			
2,4-dichlorophenoxyacetic acid (2,4-D)	X	X	
Mecoprop	X		X
Dicamba	X		X

Contaminant	Environmental exposure evaluated	Health risk previously evaluated in Working Draft Report *(Chapter VB)*	Health risk evaluated for Final Report (Chapter VC)
Carbaryl	X		X
Public Water Supply contaminants			
1,1,1-trichloroethane	X	X	
1,1-dichloroethane	X	X	
carbon tetrachloride	X	X	
Benzene	X	X	

Appendix IV-1. Evaluation of use of environmental information for CMP exposure evaluation

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
NYS DOH Radon data	Yes	Widespread	High	Completed	Moderate 1986-1999	No	**Outcome**: Although data are relatively recent, they are good surrogates for historical radon levels. These data can be used to evaluate radon exposures. **Result**: Proceed with comparisons.
Air Quality							
US EPA Toxic Release Inventory (TRI)	Yes	Unknown	Moderate	Potential	Moderate (1988 - 2001)	Yes - CEP/NATA provide better quality of exposure information for years modeled)	**Outcome:** Data present limited information to characterize exposure **Result:** Data may be considered in the future.
US EPA National Emission Inventory (AIRS)	Yes	Unknown	Moderate	Potential	Moderate (1985 - 1998 criteria pollutants, 1993 - 1996 HAPs)	Yes - CEP/NATA provide better quality of exposure information for years modeled.	**Outcome:** Data present limited information to characterize exposure. **Result:** Data may be considered in the future.
US EPA National-scale Air Toxics Assessment (NATA)	Yes	Widespread	Moderate	Completed	Moderate (1996)	No	**Outcome:** Data provide estimates of ambient concentrations, which could be a useful measure of exposure. **Result:** Proceed with comparisons.
US EPA Cumulative Exposure Project (CEP)	Yes	Widespread	Moderate	Completed	Moderate (1990)	No	**Outcome:** Data provide estimates of ambient concentrations, which could be a useful measure of exposure. **Result:** Proceed with comparisons
US EPA Air Quality System (AQS)	Yes	Widespread	High	Completed	Moderate (1982 - 2001)	No	**Outcome:** Monitoring network is very limited and included only criteria pollutants, which are not relevant to this investigation **Result:** No further evaluation
NYS DEC Permit to Construct/Certificate to Operate	Yes	Unknown	Moderate	Potential	Moderate (early 1980s)	Yes - CEP/NATA provide better quality of exposure information for years modeled.	**Outcome:** Data present limited information to characterize exposure. **Result:** Data may be considered in the future.

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
Pesticides							
NYS Pesticide Sales and Use Reports Database(commercial applicator data)	Yes	Sufficient	Poor	Potential	Moderate 1997-2001	Yes	**Outcome:** Data are marginal to characterize exposure. **Result**: An evaluation of certain pesticides that were likely to be used historically was undertaken and comparisons were made on the application of these pesticides with other comparison areas of New York State.
Hazardous waste sites							
Brookhaven Aggregates	No	Localized	High	Potential	High	No	**Outcome:** Data present limited information. This site is located on the edge of the CMP area. Contamination is localized with low potential impact within CMP. **Result:** No further evaluation.
Radio Corporation of America	No	None	High	Potential	High	No	**Outcome:** Data do not provide information about the CMP area. This site is located outside of the CMP area. Contamination is localized with no impact within CMP. **Result:** No further evaluation.
Peerless Photo Products (Village of Shoram)	No	None	High	None	Moderate	No	**Outcome:** Data do not provide information about the CMP area. This site is located outside of the CMP area. Contamination is localized with no impact within CMP. **Result**: No further evaluation.
Sheridan Waste Oil (Hamlet of Medford)	No	None	High	None	High	No	**Outcome:** Data do not provide information about the CMP area. This site is located outside of the CMP area. Contamination is localized with no impact within CMP. **Result**: No further evaluation.
Heins Landfill	No	Localized	High	None	High	No	**Outcome**: Data present limited information. Nearby groundwater contamination not linked to the site; data evaluated in water quality section. **Result:** No further evaluation

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
Suffolk Material and Mining Corporation	No	Localized	High	Potential	High	No	**Outcome**: Data present limited information. Nearby groundwater contamination not linked to the site; data evaluated in drinking water section. **Result:** No further evaluation
Lawrence Aviation Industries	No	Localized	High	Completed	High	Yes, in part	**Outcome:** Data present limited information. Groundwater contamination impacted a limited number of private wells in the immediate vicinity of the site; data evaluated in water quality section. **Result:** Data may be considered in the future
Pine Road Ecology Landfill	No	Insufficient	High	Completed	High	No	**Outcome**: Data present limited information. Nearby groundwater contamination not linked to the site; data evaluated in water quality section. **Result:** No further evaluation.
Public Water Supply							
SCDHS Historical Public Water Supply (1971-1996)	Yes	Localized	High	Potential	High	No	**Outcome:** Data can be used to characterize exposures. Includes data from active and inactive sources. **Result:** Proceed with comparisons.
SCDHS Recent Public Water Supply (1996-present)	Yes	Localized	High	Potential	Poor	No	**Outcome:** Data are not temporarily relevant. **Result:** No further evaluation of this data set.
SCDHS Historical Public Water Supply Data - Volatile Organic Compounds	Yes	Localized	High	Potential	High	No	**Outcome:** Data can be used to characterize exposures. Includes data from active and inactive sources. Comprehensive data showing limited

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
							historical contamination **Result:** Proceed with comparisons.
SCDHS Historical Public Water Supply Data – Metals	Yes	Localized	High	None	High	No	**Outcome:** Data present limited information to characterize exposures; no evidence of heavy metal contamination. **Result:** No further evaluation of data set.
SCDHS Historical Public Water Supply Data – Pesticides	Yes	Localized	High	None	Moderate	No	**Outcome:** Comprehensive data set showing no evidence of pesticide contamination **Result:** No further evaluation of data set
USGS National Water Information System Data (1977-1993)	Yes	Localized	Poor	Potential	High	Yes (SCDHS Historical Public and Private Water Data	**Outcome:** Data present limited information to characterize exposure. Includes both data from both public drinking water wells, other wells and surface water. Not directly relevant to human consumption. **Result:** Data may used to support further analysis; evaluate data set for data coverage for VOCs, Pesticides and Metals.
USGS – Volatile Organic Compounds	Yes	Localized	Poor	Potential	High	See above	**Outcome:** Data present limited information. Includes both data from both public drinking water wells, other wells and surface water. Not directly relevant to human consumption. **Result:** Data may used to support further analysis
USGS- Metals	Yes	Localized	Poor	None	High	See above	**Outcome:** Data present limited information. Includes both data from both public drinking water wells, other wells and surface water. Few reports of heavy metals. **Result:** No further evaluation of data set
USGS – Pesticides	Yes	Localized	Poor	None	Poor	See above	**Outcome:** Data present limited information. Includes both data from both public drinking water wells, other wells and surface water. Few reports pesticide contaminants

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
							Result: No further evaluation of data set
Private water supply							
SCDHS Historical (1971-1996)	Yes	Localized	High	Potential	High	No	**Outcome:** Data can be used to characterize exposure. Includes data from active and inactive sources. Comprehensive data showing limited historical contamination. **Result:** Proceed with comparisons.
SCDHS Recent (1996-2002)	Yes	Localized	High	Potential	Poor	No	**Outcome:** Data not temporarily relevant to this investigation. **Result:** No further evaluation of data set.
NYS DOH independent survey of water systems (1976-1995)	No	Localized	High	Potential	Poor	No	**Outcome:** Data present very limited information to characterize exposure. **Result:** No further evaluation of data set.
Other Data sources listed in the Initial Environmental Inventory							
NYS DEC Major Oil Storage Facilities Database (MSOF)	Yes	Insufficient	Poor	Potential	Poor	No	**Outcome:** MSOF data evaluated. These data present limited information to characterize exposure. This database provides locations of facilities that store oil on site above certain quantities specified by New York State. **Result:** No further evaluation.
US EPA RCRIS Hazardous Waste Generators Database	Yes	Insufficient	Poor	Potential	Poor	No	**Outcome:** US EPA data evaluated. These data present limited information to characterize exposures. This database provides the locations of hazardous waste generators, there is no information on releases or exposures **Result:** No further evaluation.

Data Source	Completeness of information	Potentially exposed population	Quality of exposure information	Exposure pathway	Temporal relevance	Information in another data set	Outcome
US EPA Permit Compliance System (maintained by NYS DEC)	Yes	Insufficient	Moderate	Potential	Poor	No	**Outcome:** PCS data evaluated. These data present limited information to characterize exposure. Permitted wastewater discharges are regulated in a manner to minimize exposure. **Result:** No further evaluation.
NYS DEC Spill Incidents Database	Yes	Unknown	High	Potential	Poor	No	**Outcome:** Spill incidents data evaluated. These data present limited information. Spills reported to the database are through a regulatory framework designed to minimize human exposure. **Result:** No further evaluation.
NYS Fish Consumption Advisories	Yes	Unknown	High	Potential	Poor	No	**Outcome:** Data present limited information to characterize exposure. The fish advisories are issued for the Long Island Sound and are not specific for the CMP area. **Result:** No further evaluation.

Appendix IV-2. Initial Environmental Inventory

New York State Department of Health

CORAM / MT. SINAI / PORT JEFFERSON STATION AREA INITIAL ENVIRONMENTAL INVENTORY

I. Background

One of the steps in investigating the occurrence of an unusual disease pattern is to conduct an initial environmental review. The review begins by compiling a list of facilities, located within or near the community, that are known to release contaminants into the environment or use/store regulated chemicals on their premises. This list is known as a **source inventory** and is assembled from information available in federal, state, county, and local databases. In addition to the source inventory, information about potential environmental exposures in the area is collected. The potential exposure information includes monitoring data and estimations based on knowledge about pollutant sources in the area. Together, the source inventory and potential exposure information make up the *Initial Environmental Inventory*.

Upon completion, the Initial Environmental Inventory is used, along with other information, to assist in determining the direction of the unusual disease pattern investigation. If an in-depth environmental investigation is deemed necessary, the inventory is more closely evaluated for potential sources of human exposure. More information about how a follow-up investigation is conducted can be found in the "Unusual Disease Pattern Investigation" information sheet and at the DOH website: *http://www.health.state.ny.us/nysdoh/cancer/csii/nyscsii.htm*

The initial environmental inventory for the Coram/Mt. Sinai/Port Jefferson Station (CMP) area has been compiled and includes information about hazardous waste sites, hazardous waste generators, major oil storage facilities, chemical spills and facilities permitted to release chemicals into the air and water. Also included is information about indoor concentrations of radon, estimates of outdoor concentrations of air pollutants, water quality testing and fish consumption advisories. DOH researchers are using this information to become familiar with *potential* sources of contamination in the CMP area.

Chemicals are part of our everyday lives and their presence at facilities listed in this inventory does not mean that they present a threat to human health. Contact between people and potentially harmful chemicals, known as exposure, is necessary for harmful health effects to occur. For more information about exposure, please see the accompanying "What is Exposure?" information sheet.

The following is a summary of the Initial Environmental Review for the CMP area. Seven ZIP Codes, 11727, 11733, 11764, 11766, 11776, 11777 and 11789 were selected for this investigation. The communities that are located at least partially within the seven ZIP Codes are the Hamlets of Coram, East Setauket, Miller Place, Mount Sinai, Port Jefferson Station, Rocky Point, Setauket, Sound Beach, and Terryville, and the Villages of Belle Terre, Old Field, Port Jefferson and Poquott. All are located within the Town of Brookhaven.

The data sources used to compile this inventory are frequently updated, which may result in some differences between the information currently available and the information presented in this inventory.

II. Source Inventory

Hazardous Waste Sites: Often, one of the major environmental concerns within a community is the presence of hazardous waste. An inactive hazardous waste disposal site is defined by the State of New York in 6 NYCRR Part

375 as any “area or structure used for the long-term storage or final placement of hazardous waste including, but not limited to, dumps, landfills, lagoons, and artificial treatment ponds, as to which area or structure no permit or authorization issued by Department of Environmental Conservation (DEC) or a federal agency for the disposal of hazardous waste was in effect after August 25, 1979.”

There is currently one hazardous waste site, Lawrence Aviation, located within the seven ZIP Codes. There are also four sites within the seven ZIP Codes that have undergone investigation and/or cleanup and are no longer classified as hazardous waste sites: Heins Landfill, Suffolk Materials Mining Corporation, Brookhaven Aggregates and Pine Road Ecology Site. There is one hazardous waste site, RCA-Rocky Point Landfill, located outside of the area but near the seven ZIP Code boundaries. More information regarding these and other hazardous waste sites in NY can be found at the following websites:
http://www.epa.gov/superfund/sites/npl/npl.htm
http://www.epa.gov/enviro/html/em/index.html
http://www.atsdr.cdc.gov/HAC/PHA/

Major Oil Storage Facilities: There are two Major Oil Storage Facilities (MOSF) in the seven ZIP Codes. A MOSF is defined as a facility with a combined capacity of over 400,000 gallons. The facilities are: the Port Jefferson Power Plant, operated by KeySpan Energy, and the Tosco Pipeline Company facility in East Setauket. Both of these oil storage facilities have a license to operate from DEC.

Hazardous Waste Generators: There are 326 facilities in the seven ZIP Codes that generate and/or store hazardous waste and are required to report to regulatory agencies in accordance with the Resource Conservation and Recovery Act (RCRA). Nearly all of the RCRA facilities in the CMP area are the types of small quantity generators that are found in nearly every community. These types of facilities include auto repair shops, photo developing labs (including the 1-hour labs in department and drug stores), hospitals, dry cleaners, dental offices, medical offices, and even local schools.
Large quantity generators of hazardous waste must also report to the Federal Government under the Biennial Reporting System (BRS) and to DEC. *(Generating more than 2.2 pounds per month of certain wastes can classify a facility as a large-quantity generator.)* A total of 11 facilities of this type were identified within the seven ZIP Codes: Long Island Railroad Old Town Bridge, two Genovese drug stores, Setauket Exxon Automotive Center, Port Jefferson Marine Maintenance Incorporated, LI Diagnostic Imaging, North Island Mini Lab Incorporated, Guaranteed Returns Incorporated (a pharmaceutical wholesale company), the Port Jefferson School District, the Port Jefferson Power Plant and the Mobil Oil Terminal. Additional information about these facilities can be found at the following websites:
http://www.epa.gov/enviro/html/em/index.html
http://www.epa.gov/enviro/index_java.html

Further information about RCRA can be found at these websites:
http://www.epa.gov/rcraonline/
http://www.epa.gov/epaoswer/general/orientat/
http://www.dec.state.ny.us/website/dshm/index.html

Toxics Release Inventory: The Federal Emergency Planning and Community Right-to-Know Act (EPCRA) was passed in 1986 for the purpose of informing communities and citizens of chemical hazards in their areas. The data collected by federal and state agencies concerning releases and transfers of certain chemicals from certain types of industrial facilities are made available to the public in the Toxics Release Inventory (TRI). There are two facilities within the seven ZIP Codes listed in the US Environmental Protection Agency’s (EPA) TRI database for the year 2000: Port Jefferson Power Plant and Lawrence Aviation. There are no additional facilities located outside of the area but near the seven ZIP Code boundaries. Information about these and other TRI facilities can be found at the following websites:
http://www.epa.gov/enviro/html/em/index.html
http://www.epa.gov/enviro/html/tris/tris_query.html
http://toxnet.nlm.nih.gov/index.html

Air Emissions: The Aerometric Information Retrieval System (AIRS) is a federal computer-based repository for information about air pollution in the United States. The AIRS databases contain information about facilities that are regulated under the Clean Air Act and also contain pollutant concentration data measured at monitoring stations across the US. There are 24 facilities listed within the seven ZIP Codes: A-1 Cleaners, Andree Cleaners, Broadway Cleaners, Brookstone Cleaners, Exxon, Mather Memorial Hospital, Lilco - Port Jefferson Substation, Majic Cleaners, Martin Cleaners, Mobil/Port Jefferson, PK Scrap Metals, Port Jefferson Power Plant, Rason Asphalt Incorporated, St. Charles Hospital, Sundial Asphalt Company Incorporated, Three Roads Cleaners, Tosco Pipeline Company, three US Postal Service facilities, Village Drive In Cleaners, Wall Mates Vinyls, Ward Melville Senior High School, and Your Dry Cleaners.

There are three AIRS facilities located outside of the area but near the seven ZIP Code boundaries: one cogeneration plant, one dry cleaner and one oil company. All of these facilities are presently listed as being in compliance with the requirements of the regulatory programs. More information about facilities listed in the federal and state databases for air emissions is available at the following websites:
http://www.epa.gov/air/data/
http://www.epa.gov/enviro/html/em/index.html

Wastewater Discharges: The Permit Compliance System (PCS) is a federal (EPA) database system that provides information about facilities that have been issued permits to discharge wastewater into surface or groundwater. The PCS data regarding discharges in the State of New York are obtained from DEC.

There are 24 such facilities within the seven ZIP Codes listed in the PCS database. Eighteen of the facilities are condominiums or laundromats whose discharges are similar to those of a home septic system. Six of the PCS facilities are permitted to discharge metals and/or organic solvents: the Port Jefferson School District, the Selden School District, Keyspan Energy, Tosco Pipeline Company, Port Jefferson Diesel Yard and Collaborative Group, Ltd. More information about facilities listed in the federal and state databases for wastewater discharges is available at the following websites:
http://www.epa.gov/enviro/index_java.html
http://www.epa.gov/enviro/html/em/index.html

Pesticide Use: A database containing information about the *professional* application of pesticides is currently under review at DOH. The DEC website has information about statewide pesticide use and sales at the county level. Links to data at the ZIP Code level can also found at *http://www.dec.state.ny.us/website/dshm/prl/.*

Golf courses: Golf courses have been included in the Initial Environmental Inventory because of their possible pesticide use. Three golf courses have been identified within the seven ZIP Codes: Port Jefferson Country Club (116 acres), Heatherwood Golf Course (38 acres) and St. George's Country Club (136 acres). Five golf courses have been identified outside of the area but near the ZIP Code boundaries: Nissequogue River Golf Course (126 acres), Middle Island Country Club (212 acres), Spring Lake Golf Club (187 acres), Mill Pond Golf Course (87 acres) and Tall Tree Golf Course (52 acres).

Spill Incidents: DEC maintains a database of spill incidents involving one gallon or more under Article 12 of the New York State Navigation Law. The law prohibits the unregulated discharge of petroleum on land and water, but the database includes spills of raw sewage as well. The database includes incidents reported since January 1, 1978. As of February 27, 2002, the number of spills for each of the communities included in the seven ZIP Codes is Belle Terre (8), Coram (229), Miller Place (96), Mount Sinai (122), Old Field (16), Port Jefferson (468), Port Jefferson Station (131), Poquott (11), Rocky Point (201), Setauket/East Setauket (217), Sound Beach (79), and Terryville (16). The greatest number of reported spills occurred during home heating oil deliveries, and most of the remaining spills occurred during gasoline and diesel fuel deliveries. Other spills included raw sewage, jet fuel, kerosene, waste oil, transformer oil, pesticides, and PCB oil. More detailed spill information can be found at *http://www.dec.state.ny.us/apps/derfoil/index.cfm?pageid=2*

III. Data on Potential Environmental Exposures

Estimated Air Concentrations: Two important sources of air concentration data are EPA's Cumulative Exposure Project (CEP) and National-scale Air Toxics Assessment (NATA). Outdoor air concentrations for 148 hazardous air

pollutants based on 1990 emission data are provided in CEP, while the more recently released NATA has concentrations for 33 hazardous air pollutants (plus diesel particulates) based on 1996 emission data. Both CEP and NATA provide estimates of annual average concentrations for these pollutants at nineteen point locations within the seven ZIP Codes. These estimates are based on the emissions from a variety of sources including: gas stations, factories, power plants, cars, and lawnmowers. The procedures that EPA used to estimate pollutant concentrations considered source characteristics, local weather patterns and other factors. In areas of the State where hazardous air pollutant monitoring data are available, comparisons with estimated concentrations from CEP have shown reasonable agreement for some pollutants, although not as good agreement for others. Also, for some pollutants, estimated concentrations in some regions of the State may be more representative of actual air concentrations than in other regions. Nonetheless, CEP and NATA provide valuable information about potential exposure to hazardous air pollutants in outdoor air. For more information about the air pollution estimates, visit the EPA websites at *http://www.epa.gov/region4/air/airtoxic/index.htm*

Air Monitoring Stations: For various time periods since 1965, ten air quality monitoring stations operated in or near the CMP area and reported data to the EPA. Five of the air monitoring stations operated within the area, and another five operated outside of the area but near the seven ZIP Code boundaries. All five of the monitoring stations within the seven ZIP Codes ceased operating by 1984. Three of the stations measured sulfur dioxide (SO_2), one measured total suspended particulate matter (TSP) and one measured sulfate (SO_4^{2-}) and nitrate (NO_3^-) concentrations in TSP. Four of the five air monitoring stations located outside of the seven ZIP Codes also ceased operating by 1984. One of the stations measured TSP, two measured SO_2 and dustfall, and the fourth measured ozone (O_3). The remaining active monitoring station is located in Holtsville, NY and began operating in January 2000. This station collects data on SO_2, O_3, carbon monoxide (CO), nitric oxide (NO), nitrogen dioxide (NO_2), total non-methane hydrocarbons (TNMHC), methane (CH_4), particulate matter less than 2.5 microns in diameter ($PM_{2.5}$), and meteorological data. Annual summaries of the data collected can be found at the EPA website at *http://www.epa.gov/aqspubl1/annual_summary.html*

Fish Consumption Advisories: DOH issued several fish consumption advisories for waters adjacent to the Coram/Mt. Sinai/Port Jefferson Station area in 2001. An advisory is in effect for Spring Pond, located to the east of ZIP Code 11727, due to elevated levels of chlordane in carp and goldfish. An advisory is in effect for St. James Pond (Mills Pond), located to the west of ZIP Code 11733, for all fish species due to chlordane and DDT contamination. An advisory is also in effect for marine bluefish, American eels, and marine striped bass taken from the Long Island Sound due to polychlorinated biphenyl (PCB) contamination. The most recent advisories about the consumption of sportfish and game can be found on the DOH website at *http://www.health.state.ny.us/nysdoh/fish/fish.htm*

Drinking Water: DOH is currently working with the Suffolk County Department of Health Services to gather information about historic public and private drinking water quality and historic industrial waste discharges in the area. Recent drinking water quality reports for the CMP area can be found on the Suffolk County Water Authority's website at *http://www.scwa.com/press/pressreleases.cfm*

In addition Suffolk County has an extensive monitoring program to detect the presence of pesticides in groundwater. The most recent annual report summarizing the results can be found at *http://www.dec.state.ny.us/website/dshm/prl/suffolk.pdf*

Radon: As of June 1999, the New York State Department of Health's Bureau of Environmental Radiation Protection had measured radon levels in 53 homes in Brookhaven Town, which includes the CMP area. Approximately 0.8% of the homes in the area exceed the action level (4 pCi/L) for radon in the living area, and 5% of the homes exceed the action limit for radon in the basement. More information about the health effects of radon and data for the indoor radon measurements taken throughout New York State can be found at *http://www.health.state.ny.us/nysdoh/radon/radonhom.htm.*

IV. Conclusion: DOH staff has compiled an Initial Environmental Inventory, as outlined in Step 2 of the Unusual Disease Patterns Follow-up Activities Protocol. In view of the fact that the epidemiologic investigation continued to suggest an elevation of breast cancer in the area, DOH concludes that the available information on *potential*

sources of environmental exposure justifies proceeding with Step 3 of the Protocol. In Step 3, DOH will seek community input and evaluate available information on potential environmental exposures. Important information includes, but is not necessarily limited to, facility location and type, location of smokestacks, unusual odors, documented or suspected drinking water contamination, chemicals used, waste handling practices and chemical spills. Individuals can report such information by completing a Community Environmental Concerns Reporting Sheet that will be distributed at community meetings and will be available upon request.

V. Contact Information: If you would like to receive additional information, or have specific environmental concerns about your community, please call the toll-free Environmental Health Information Line at 1-800-458-1158, extension 27530. More information about the Cancer Surveillance Improvement Initiative can be found on the NYS DOH web site at www.health.state.ny.us.

Information concerning the environmental quality in your community can be found at the following websites:

1. The United States Environmental Protection Agency's (EPA) Enviromapper website allows you to locate sites of interest, such as superfund sites, RCRA facilities, and air emission sources anywhere in the US. This site is searchable by ZIP Code: *http://www.epa.gov/enviro/html/em/index.html*
2. EPA's "Envirofacts Warehouse" provides access to several EPA databases that provide you with information about environmental activities that may affect air, water, and land anywhere in the United States. This site is searchable by ZIP Code: *http://www.epa.gov/enviro/index_java.html*
3. EPA's Office of Solid Waste provides more information about the Resource Conservation and Recovery Act (RCRA): http://www.epa.gov/rcraonline/*, http://www.epa.gov/epaoswer/general/orientat/*
4. EPA's Superfund website provides additional information about sites on the National Priorities List (NPL): *http://www.epa.gov/superfund/sites/npl/npl.htm*
5. EPA's AIRDATA provides access to air monitoring data and is searchable by state, county, and city. Annual summaries are available for specific locations and on a regional basis:*http://www.epa.gov/air/data/reports.html*
6. EPA's Office of Air Quality Planning and Standards website has information about modeled ambient concentrations of hazardous air pollutants: *http://www.epa.gov/ttn/atw/nata/natsa2.html*
7. EPA's Toxics Release Inventory Program website provides information about facilities and their releases into the environment: *http://www.epa.gov/tri/index.htm*
8. The U.S. Agency for Toxic Substances and Disease Registry (ATSDR) maintains a website where you can locate public health assessments for Federal superfund sites: *http://www.atsdr.cdc.gov/HAC/PHA/*
9. The National Library of Medicine's TOXNET website provides access to the EPA Toxics Release Inventory (TRI) database and a number of other chemical information sources: *http://toxnet.nlm.nih.gov/index.html*
10. DEC's website has information about Suffolk County's water quality monitoring program to detect pesticides in groundwaters of Nassau and Suffolk counties: *http://www.dec.state.ny.us/website/dshm/prl/suffolk.pdf*
11. DEC's website has information about statewide pesticide use and sales at the county level: *http://www.dec.state.ny.us/website/dshm/prl/.*
12. DEC's website has information about spill incidents and inactive hazardous waste sites that can be searched at the county and city level: *http://www.dec.state.ny.us/website/der/*

13. The NYS Department of Health's (DOH) website contains more information about Unusual Disease Pattern Investigations: h*ttp://www.health.state.ny.us/nysdoh/cancer/csii/nyscsii.htm*
14. DOH website contains information about the health effects of radon and also shows data for indoor radon measurements taken throughout New York State: *http://www.health.state.ny.us/nysdoh/radon/radonhom.htm.*
15. DOH website has the most recent advisories about the consumption of sportfish and game: *http://www.health.state.ny.us/nysdoh/fish/fish.htm*
16. Suffolk County Water Authority's "Your Water". This website allows you to access water quality reports for the Suffolk County water zones: *http://www.scwa.com/*

V. Integration

A. Methodology

The goal of Integration (Step 3 of the Unusual Disease Patterns Protocol) is to evaluate environmental contaminants and other risk factors that could be related to the elevated breast cancer incidence and make conclusions about their potential importance as risk factors for breast cancer in the CMP area. Conclusions in this step were made using a process recommended by the National Academy of Sciences (NAS, 2001[2]) to help US EPA determine the drinking-water contaminants that would most likely pose the greatest future threat to the safety of drinking water.

In Chapter III, the NYS DOH toxicology team classified environmental contaminants to characterize the likelihood that they are environmental risk factors for breast cancer. In Chapter IV, the NYS DOH environmental exposure team used existing environmental data sets and comparison areas to identify environmental contaminants that are elevated or potentially elevated in the CMP area. In this chapter, NYS DOH staff combined the toxicological and exposure information for each of environmental contaminants to evaluate the likelihood that each contaminant was an important risk factor for breast cancer, or other health effects, in the CMP area.

The narratives allow two specific questions to be answered.

1. Are there environmental contaminants in the CMP area that would warrant further evaluation using environmental investigative techniques (i.e., environment sampling)?
2. Are there environmental contaminants that would warrant further consideration for a possible analytical epidemiological study in the CMP area?

The integration narrative of each contaminant contains a qualitative discussion of the following attributes.

The degree of confidence (*high, low*) that the environmental data set accurately represents exposure of the residents to environmental contaminants in the CMP area during the years important to the start and development of breast cancers reported between 1993 and 1997 (i.e., perhaps 5 - 40 years earlier). This ranking is based largely on an assessment of two aspects of exposure.

[2] National Academy of Sciences (NAS), 2001. Classifying Drinking Water Contaminants for Regulatory Consideration. Washington, D.C.; National Academy Press.

- The degree to which the environmental data set accurately represents the exposure of the residents in the CMP area during the years covered by the environmental data set.

Environmental Data Set	Qualitative Descriptor of Exposure Estimate
provides direct evidence of exposure to a contaminant (e.g., biomonitoring data) or direct evidence (e.g., sampling data) of the presence of a contaminant in an environment medium that humans directly contact (e.g., drinking water, air)	good
provides indirect evidence (e.g., modeling data) of the presence of a contaminant in an environment medium that humans directly contact (e.g., drinking water, air)	fair
provides limited or marginal evidence of the presence of a contaminant in the environment (e.g., production or emissions data, pesticide application data)	poor

- The degree of confidence that the environmental data set accurately represents environmental conditions in the CMP area 5 to 40 years before 1993-1997.

Environmental Data Set	Qualitative Descriptor of Exposure Estimate
provides environmental data collected during the time of cancer initiation and development (5-40years prior to 1993 - 1997)	good
provides environmental data collected in 1987 or later, but can reasonably be assumed to be representative of environmental conditions during the time of cancer initiation and development (5-40years prior to 1993 – 1997).	fair
provides environmental data collected 1987 or later, but it is unlikely or uncertain that the data are representative of environmental conditions during the time of cancer initiation and development (5-40years prior to 1993 - 1997).	poor

In addition, minor considerations are given to other aspects of exposure when appropriate data are available and relevant. These other aspects include.

- The degree to which the exposure associated with the CMP environmental medium is a substantial source of the total exposure to that contaminant.
- The spatial pattern of contamination in the CMP area (e.g., unique to a small area; local, but in several areas; widespread).

Integration also considers the results of a weight-of-evidence analysis that classified each contaminant on its likelihood of being an environmental risk factor for human breast cancer. In other words, its category (*known*, *probable, possible, potential, not classifiable*, and *unlikely*) in the classification scheme for evaluating contaminants as environmental risk factors for human breast cancer.

Finally, Integration evaluates likelihood of health effects (breast cancer and non-cancer effects) at the estimated or measured levels of each contaminant in an environmental medium within the CMP area (e.g., air or drinking water). The likelihood of cancer effects can range from *very low* to *very high* and the likelihood of non-cancer effects can range from *minimal* to *high*. The estimated likelihood of health effects in the CMP area depend on how the estimated or measured environmental levels in the CMP area compare to the environmental levels that are typically used to set health-based guidelines or standards for environmental contaminants.

The Integration Evaluation was conducted in two phases. The first set of contaminants evaluated, which was published in the *Working Draft Report (June 2004)* included: ethylene thiourea in outdoor air, the pesticide 2,4-D, 1,1,1-trichloroethane, 1,1-dichloroethane, carbon tetrachloride and benzene in public water supplies. Those analyses are reprinted in Section *V-B. Results from the Working Draft Report*. The remaining contaminants evaluated (see *Table 56),* which include outdoor air contaminants and pesticides, are discussed in *V-C. Results for Remaining Contaminants.*

B. Results from Working Draft Report

1. Outdoor Air - Ethylene Thiourea (ETU)

The modeled data of ETU for outdoor air in the CMP area suggest that the levels of ETU in the CMP area may be elevated compared to other areas within the state. NYS DOH researchers have lower confidence in the ETU exposure estimates because the modeled estimates may not be very close to the actual ETU air concentrations within the CMP area (see *Section IV 3-d)*. ETU is classified as “a potential risk factor for human breast cancer” (Category 2C, see *Section III-C, Table I5*). A more detailed analysis was done to determine if ETU is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

ETU air concentration estimates were derived from the US EPA Cumulative Exposure Project (CEP) database. NYS DOH researchers evaluated the CEP database for ETU emission sources in New York and surrounding states that may have contributed to the air concentration estimates for the CMP region. The only ETU emission source in NYS included in the CEP database is in western New York. This source would not contribute to the concentration estimates in Suffolk County. A review of adjacent states’ emission sources identified one source in Stratford, Connecticut. This source is an industrial facility involved in the manufacture of rubber products. The sources in New York and Connecticut also were listed as ETU sources in the US EPA’s Toxic Release Inventory. Thus, emissions from the facility in Connecticut are likely to be the sole contributor to the modeled estimates of ETU in the CMP region.

ETU is used in the rubber-making industry and to make certain pesticides (i.e., ethylene-bis-dithiocarbamate fungicides). The general population may be exposed to ETU while using rubber products or pesticides that contain trace amounts of ETU and when eating foods containing pesticide residues. Reliable estimates of ETU exposure from these sources are not available. Thus, we are unable determine the relative contribution that the modeled air levels could contribute to the total ETU exposure for a CMP resident.

The estimated ETU concentration, averaged over the entire CMP area, is 8.95×10^{-8} micrograms per cubic meter (mcg/m^3). This value is approximately 34-times higher than the estimated state average, 18-times higher than the estimated state average excluding New York City, and is about 2.5 times higher than the estimated Suffolk County average *(Table 38).* The highest value modeled in the CMP area is 1.37×10^{-7} mcg/m^3.

Table 38. Comparisons of ETU modeled concentrations

Comparison Region	Modeled average ETU concentration (mcg/m^3)	Ratio CMP to comparison area
CMP	8.95×10^{-8}	1.00
Suffolk County without CMP	3.51×10^{-8}	2.55
New York State	2.64×10^{-9}	33.9
NYS without NYC	4.86×10^{-9}	18.4

A review of the electronic database of the Toxic Release Inventory (TRI) shows that ETU source in Stratford, Connecticut reported ETU releases for 1989 to 1997. Thus, estimates of ETU air concentrations in the CMP area at the time of cancer initiation (perhaps 5-40 earlier than 1993-1997) cannot be estimated using the TRI database.

The estimated amount released in 1990, which was the year used to estimate the ETU levels in the CMP, was the same amount reported from 1990 to 1997. After 1997, the Toxic Release Inventory did not list a ETU release for the facility. The most likely causes of this de-listing are that the amount of ETU released was below the threshold for reporting or that ETU was no longer released. In addition, the half-life of ETU in air is short (estimated at between 0.5 and 4.7 hours (Howard et. al., 1991)), thus, current levels of ETU are likely to be lower than those estimated in 1990.

In conclusion, the degree of confidence that the 1990 ETU data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993-1997 is *low*. This ranking is based on two determinations. (1) The ETU data are considered a *poor* estimator of residential exposure in the CMP area during the year covered by the ETU data set because the ETU estimates are based on modeled data. (2) The ETU data are considered a *poor* estimate of ETU levels in the CMP air during the years important to the start and development of breast cancers reported in 1993-1997 (i.e., perhaps 5-40 years earlier) because their validity for that time period cannot be readily determined.

Toxicity Narrative (Breast Cancer)

The classification of ETU as "a potential risk factor for human breast cancer" (Category 2C) is based on the level of evidence for data on humans (*inadequate*), animals (*inadequate*) and mode-of-action (*limited*). The toxicological information that formed the basis for those determinations is summarized below. The definitions for all toxicological classifications are found in *Table 12.*

One study looked for evidence of thyroid cancer in humans exposed to ETU. This study of 1,929 workers (including 699 women) engaged in the production or manufacture of ETU did not find any evidence of an increased incidence of thyroid cancer or of an increased incidence of cancer in general (NTP, 1992; IARC, 1987). No data on the incidence of breast cancer were found. Thus, human data are classified as inadequate.

Animal studies show that lifetime oral exposure to ETU can cause thyroid, liver, and pituitary gland tumors in mice, and thyroid tumors in rats (NTP, 1992). Lifetime oral exposure to ETU did not cause tumors in one strain of hamsters (Gak et al., 1976). ETU did not cause mammary gland tumors in any of these studies. One study (Belpoggi et al., 2002) of rats fed a diet containing the pesticide mancozeb, which is contaminated with ETU and can be metabolically converted to ETU, found an increase in mammary gland tumors in rats that lived more than 2 years, which is longer than the typical study length for cancer studies. Whether ETU, by itself, would also increase the incidence of mammary tumors if the animals were kept longer (as in the mancozeb study) is not known. Thus, the data on the mammary carcinogenicity of ETU in animals are classified as *inadequate.*

Data on the mode-of-action by which ETU might cause cancer were also evaluated. The mode-of-action data indicate that damage to DNA does not play a major role in ETU-induced carcinogenesis in animals. Instead, the data suggest ETU produces thyroid tumors in animals by interfering with the normal functioning of the thyroid gland, which reduces the amount of circulating thyroid hormone [thyroxin (T_4)] produced by the thyroid gland. The resulting reduction in thyroxin level stimulates an increased secretion, by the pituitary, of thyroid-stimulating hormone (TSH). This increased secretion of TSH, in turn, stimulates the cells of the thyroid gland to proliferate and increases the production of thyroxin (NTP, 1992). The increased cell division and proliferation increases the chance of cancer-causing mutations, potentially leading to the formation of thyroid tumors.

This generally accepted mode-of-action leads to several not-yet-accepted hypotheses about potential ETU-hormonal interactions. For example, preliminary data from human epidemiology suggest, but do not demonstrate, that a combination of hypothyroidism (i.e., chronically low levels of serum thyroxin) and unusually long lifetime exposure to estrogen, might lead to an increased risk of breast cancer (Morabia et al., 1992). This hypothesis, which needs to be confirmed by a larger human study and by studies in animals, could help explain the late-in-life mammary tumors observed in the mancozeb bioassay.

On the other hand, exposure to mancozeb is not the same as exposure to pure ETU. The mancozeb used in these studies was only 85% pure (Belpoggi et al., 2002); the rest was ETU and other, unidentified contaminants. Furthermore, mancozeb is converted in the body to a number of other metabolites that would not be formed from ETU, including (probably) carbon disulfide (US EPA, 2001). Carbon disulfide is capable of cross-linking proteins

(US EPA, 2001), a potential mode-of-action that would be expected to accelerate the formation of any tumors that might be caused by ETU. The mode-of-action data are classified as *limited.*

Risk Narrative

(1) Cancer Effects

Long-term exposure to high levels of ETU caused cancer in laboratory animals. The dose-response data describing the relationship between ETU exposure and the incidence of cancer in animals can be analyzed and expressed as the ETU air concentration that is associated with an estimated excess lifetime human cancer risk of one in one million (1×10^{-6}), assuming continuous exposures. One estimate of this air concentrations is 7.7×10^{-2} mcg/m^3 (see *Table 39),* which is based on cancer of the thyroid.

Table 39. Risk reference values for cancer effects of ethylene thiourea

One in One Million (1×10^{-6}) Risk Level: Air Concentration[1]	Cancer type	Source
7.7×10^{-2} mcg/m^3	thyroid	CA EPA, 1997

[1]Calculated from excess risk level of 1×10^{-6}/ inhalation cancer potency factor (1.3×10^{-5} per mcg/m^3).

This value was chosen because the thyroid is more sensitive than other organs to the toxicity of ETU and because ETU did not induce breast cancer. Thus, it was used as a worst-case surrogate for breast cancer. Using 8.95×10^{-8} mcg/m^3 (the average modeled ETU air concentration in the CMP area) and the one in one million risk level for air (above) indicates that continuous lifetime exposure to ETU in the CMP area would be associated with an excess cancer risk of 1.2×10^{-12}. This risk estimate is about one million times lower than the excess risk (one in one million or 1×10^{-6}) that is generally used to set guidelines or standards. The risks from ETU in air are rated “very low” using the method NYS DOH has used to evaluate potential cancer risks from environmental contaminants *(Appendix V-1).*

(2) Non-Cancer Effects

Exposure to high levels of ETU can cause non-cancer effects in humans and laboratory rats, including impaired thyroid function (Graham et al., 1975; US EPA, 2004). The potential for this and other health effects was evaluated by comparing the estimated ETU air concentration to an air concentration generally used to set standards or guidelines (i.e., the reference concentration, *Table 40*). The reference concentration is an air concentration that is expected to be without an appreciable risk of non-cancer health effects. The estimated ETU air concentration in the CMP area (8.95×10^{-8} mcg/m^3) is only a tiny fraction (0.0000003) of the ETU reference concentration. The non-cancer health risks associated with a ratio below 1 are rated “minimal” on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 40. Risk reference values for non-cancer effects of ethylene thiourea

Reference Dose (RfD)	Reference Concentration[1]	Target organ	Source
8 x10^{-5} mg/kg-day	0.28 mcg/m^3	thyroid	US EPA, 2004

[1]Calculated from (RfD mg/kg-day) x [(70 kg)/(20 m^3/day)] x (1000 mcg/mg).

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on ETU in the air of the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *low confidence* in the likelihood that the ETU air data accurately represent the exposure of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis that classifies ETU as "a potential risk factor for human breast cancer." (3) The risk analysis that indicates that the likelihood of health risks at the modeled ETU air concentrations are estimated to be *very low* for cancer risks and *minimal* for non-cancer risks. These are the lowest possible qualitative descriptors of risk used by the NYS DOH.

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2. Pesticides - 2,4-D (2,4-dichlorophenoxyacetic acid and its salts and esters)

Researchers modified their approach to identifying possibly elevated pesticide exposures to 2,4-D from the Working Draft Report to the Final Report. They modified the approach because 2,4-D application rates were clearly affected by land-use and population density, which vary considerably across Suffolk County. 2,4-D application rates in the CMP area were therefore compared to other parts of Western Suffolk County where land-use and population density are similar (see IV-3. Pesticide Use). As a result of this change, 2,4-D application rates were not considered elevated and the health risks of 2,4-D would not have been evaluated further. However, to provide a complete record of all evaluations conducted, the original Integration Evaluation from the Working Draft Report is provided here.

2,4-D is used for lawn and landscape maintenance and is present in many weed-control products, including those that also contain fertilizer. Between 1997 and 2001, the amounts (in pounds per square mile) of 2,4-D applied by commercial applicators appeared to be higher in the CMP area than in the rest of Suffolk County, and much higher than in the rest of New York State. Thus, the use of 2,4-D by commercial applicators is considered elevated in the CMP area. However, our researchers need to resolve an issue about the reported 2,4-D data.*

2,4-D is classified as “unlikely to be a risk factor for human breast cancer” (Category 4). Nevertheless, a more detailed analysis has been done to determine if 2,4-D is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

As described in the Pesticide Use Section in Part IV of this report, the most comprehensive information on pesticide use in New York is contained in New York State's Pesticide Sales and Use Reporting Database, which was established under New York's Pesticide Reporting Law. The law, which was enacted in 1996, requires commercial pesticide applicators (professional applicators) to report to the NYS DEC, on an annual basis, pesticide use information for each pesticide application they made. The law also requires the sellers of restricted use pesticides to report each sale, private applicators (those who apply restricted use pesticides on their own or their employer’s property) who apply pesticides for agricultural purposes to maintain records of restricted pesticide use, and manufacturers and importers of restricted use pesticides to report sales.

DOH researchers used the commercial application database for the CMP area because it is the only set of data reported to the state that contains information about the locations of pesticide applications. Currently, finalized data are available for the years 1997 through 2001. DOH researchers used ZIP Code level data to evaluate regional differences in pesticide application rates.

These data report product use, not actual environmental levels or human exposures. Such use data are the weakest type of exposure information considered in this evaluation. The time period represented by the data does

not coincide with the time period of onset or development of breast cancer diagnosed between 1993 and 1997. However, 2,4-D has been used for more than 50 years and its recent use may be representative of historic use.

2,4-D is an ingredient in many popular over-the-counter weed control products. Large quantities may be applied by residential and private applicators without any reporting requirements to New York State's Pesticide Sales and Use Reporting Database. Thus, the data are too limited to conclude definitively that, for the years 1997 to 2001, the total amount of 2,4-D applied in the CMP area was greater than amounts applied in other areas, even if the amount applied by commercial applicators was greater.

Typically, 2,4-D is applied to lawns as granules or as a liquid spray. People may be exposed to 2,4-D during its application, and after application, through contact of their skin with treated surfaces (e.g., grass) or surfaces contaminated by spray or dry application. 2,4-D remains in soil for a short time before it is broken down. (Its soil half-life is typically less than seven days.) When 2,4-D is translocated into homes (i.e., tracked into homes on shoes or by household pets), on the other hand, it may persist much longer. In addition to skin contact and (for children) ingestion following hand-to-mouth activity, people can also be exposed by eating produce treated with 2,4-D. Another potential source of exposure is drinking water. However, 2,4-D has not been detected in public or private drinking water of the CMP area monitored by the Suffolk County Department of Health Services.

Limited evidence suggests that indoor exposures to 2,4-D tracked into homes after outdoor use may be a larger source of exposure than dietary intake of 2,4-D. In a study of young children living in homes where 2,4-D was used to treat the lawn, Nishioka et al. (2001) evaluated the exposure from dietary sources (commercial agricultural products) and indoor exposures (i.e., inhalation, ingestion and dermal contact with 2,4-D carried into the home) after outdoor 2,4-D use near the home. For the median exposure estimates, the pre-application exposures from non-dietary indoor and dietary exposure were 0.085 and 1.3 mcg/day, respectively, but the post-application non-dietary indoor and dietary exposures were 1.1 mcg/day and 1.3 mg/day. Thus, after application, indoor exposures increased from 6% of total exposure to 46% of the total exposure. For the maximum exposure estimates, the pre-application exposures from non-dietary indoor and dietary exposure were 0.66 and 1.3 mcg/day, respectively, but the post-application non-dietary indoor and dietary exposures were 7.6 mcg/day and 1.3 mg/day. Thus, indoor exposures increased from 34% to 85% of the total exposure. These estimates are about 1% to 10% the exposure level generally used to set guidelines or standards based on non-cancer health effects (see discussion below).

The exposure of children from outdoor contact with soil, treated plants, or surfaces contaminated by spray or dry application was not evaluated, which was a limitation of the study. However, several observations suggest that, for humans, indoor exposures associated with outdoor use of pesticides, including 2,4-D, are generally greater than outdoor exposures (Wilson et al., 2003).

Data generally indicate an increased persistence of pesticides indoors compared to outdoors and subsequently higher levels of pesticides in house dust compared to outside dirt (Simcox et al., 1995; Wilson et al., 2003). In particular, Nishioka et al. (1996) estimated the 2,4-D could be found in homes for up to 1 year after application, which is much longer than the expected persistence of 2,4-D in the soil. In addition, Wilson et al. (2003) reported

that mean 2,4-D concentration in house floor dust was about 40-times higher than the mean 2,4-D concentration in the play area soils outside the houses.

Not all of the 2,4-D applied to lawns is available for absorption into the human body. The percentage of 2,4-D applied to lawns that has the potential to be knocked or rubbed off plants and onto people (called the dislodgeable residues) has been measured at 0.1% (Nishioka et al., 1996). Also, both the amounts of 2,4-D remaining on treated lawns and of dislodgeable residues can decrease rapidly (within days) after application. These factors limit the amount of 2,4-D that humans can absorb into the body after contact with treated lawns. Most people, including children are likely to spend more time indoors than outdoors (US EPA, 1999, 2002).

In conclusion, the degree of confidence that the 2,4-D data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *low*. This ranking is based on two determinations. (1) 2,4-D use data provide only limited or marginal evidence of an increased presence of 2,4-D in the CMP environment. Thus, the use data are considered a *poor* estimator of human residential exposure during the years covered by the data set. (2) 2,4-D has been used for more than 50 years, and its recent use may be reasonably representative of historic use. Thus, the 2,4-D use data for 1997-2001 are considered a *fair* estimate of 2,4-D use in the CMP area during the 5 to 40 years important to the start and development of breast cancers first reported in 1993-1997.

Toxicity Narrative (Breast Cancer)

The classification of 2,4-D as “unlikely to be a risk factor for human breast cancer” (Category 4) is based on the level of evidence for data on humans (*inadequate)*, animals (*negative*), and mode-of-action (*negative*). The toxicological information on which those determinations were based is summarized below.

2,4-D is one of several chlorophenoxy herbicides. There is an International Register of Workers Exposed to Chlorophenoxy Herbicides, Chlorophenols and Dioxins that includes most workers who have been occupationally exposed to substantial amounts of chlorophenoxy herbicides and their contaminants, which included dioxins. As of 1992, this Register included 22,000 workers, but only 1,000 were women and only 600 of these had been exposed to 2,4-D without simultaneous exposure to other contaminants (Kogevinas et al., 1993). A combined study (Kogevinas et al., 1997) of 36 smaller studies based on the Register data concluded that 2,4-D did not increase the risk of breast cancer. However, the study had a limited ability to detect cancer effects because of the relatively small number of women with only 2,4-D exposures.

In a study with a larger number of women, Schreinemachers (2000) conducted an ecological study to compare the cancer rates of entire populations of certain counties of wheat-producing states that used large quantities of 2,4-D, with the cancer rates of similar populations of counties in which much smaller amounts of 2,4-D were used. This study included 172,000 women from high-use counties and 459,000 women from low-use counties. The data indicate similar mortality rates from breast cancer in the high-use and low-use counties. Thus, the study did not see any relationship between breast cancer rates and 2,4-D use. The results of this study are limited mainly by the study design, which did not allow the scientists to take into account individual differences in exposure within a

community or individual differences in other risk factors that may alter the rate of breast cancer within or between communities.

Thus, the human data are classified as *inadequate*.

Early animal studies, while not indicating that 2,4-D could cause breast cancer, gave conflicting results with regard to certain other forms of cancer (reviewed by Bond et al., 1989; Garabrant and Philbert, 2002). More recent, state-of-the-art two-year carcinogenicity studies in rats and mice showed no evidence of cancer in any tissue (including the mammary gland) in animals fed diets containing 2,4-D, including at least one dose that showed some toxic effects on the animals (Charles et al, 1996a). Furthermore, there was no evidence of 2,4-D induced cancer in any tissue in dogs fed 2,4-D diets for one year. The data in dogs complement the rat and mice data, but must be regarded as somewhat preliminary because a one-year exposure is not long enough, and the highest dose tested was not high enough, to thoroughly test the carcinogenic potential of 2,4-D in dogs. However, these data provide sufficient evidence that 2,4-D does not induce breast cancer in mice or rats and thus, the animal data are classified as *negative*.

Data on the mode-of-action by which 2,4-D might cause cancer were also evaluated. Studies provide consistent evidence that 2,4-D is not capable of damaging DNA, either in bacteria or in mammalian cells, either *in vitro* or *in vivo*. 2,4-D (acid form) is readily excreted by the kidney into the urine, and becomes toxic to the liver and kidney only at high doses that exceed the kidney's ability to secrete it. The chronic studies in mice and rats (described above) provide evidence that, even at mildly toxic doses, 2,4-D still does not cause cancer in any tissue. Common salts and esters of 2,4-D (which are also used in pesticide formulations) are readily converted in the body to 2,4-D acid which is then secreted by the kidney into the urine. Thus the mode-of-action data are consistent with the hypothesis that 2,4-D does not cause cancer and are therefore classified as *negative.*

Risk Narrative

(1) Cancer Effects

The weight-of-evidence analysis supports the classification of 2,4-D as "unlikely to be a risk factor" for breast cancer, which is the most conclusive classification for contaminants that do not appear to be associated with human breast cancer. This classification is based largely on recent animal studies that showed no evidence of cancer in any tissue (including the mammary gland) in animals fed diets containing 2,4-D. It is also based on findings that human data and mode-of-action studies provide little evidence that 2,4-D causes cancer (of any kind) in humans. The lack of positive data on carcinogenicity preclude a risk analysis of the cancer effects of 2,4-D.

(2) Non-Cancer Effects

Some industrial workers exposed to large amounts of 2,4-D and other related chemicals suffered nervous system damage. Exposure to high levels of 2,4-D damages the liver, kidneys and blood of laboratory animals (US EPA, 2004). The potential risk of these and other health effects resulting from environmental exposure to 2,4-D in the CMP area cannot be directly evaluated from the Pesticide Sales and Use Reporting Database. However, the

potential health risks associated with 2,4-D lawn applications (in other geographical areas) was evaluated in a recent paper (Nishioka et al., 2001) which established that 2,4-D can be tracked into homes after lawn applications. Nishioka et al. (2001) estimated that a child's daily 2,4-D dose from 2,4-D tracked into the home was less than 10% of the reference dose, which is the daily dose that is expected to be without an appreciable risk of non-cancer health effects. The non-cancer health risks associated with exposures below the reference dose is rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on the environmental levels of 2,4-D in the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *low confidence* in the likelihood that the 2,4-D use data accurately represent exposures of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997 (i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis that classifies 2,4-D as "an unlikely risk factor for human breast cancer." (3) The risk analysis that indicates the likelihood of non-cancer health risks at indoor exposures (estimated by Nishioka et al., 2001) associated with outdoors 2,4-D application to be *minimal.* This is the lowest possible qualitative descriptor of risk used by the NYS DOH.

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3. Drinking Water Contaminants

Suffolk County regulatory sampling data for each of the public wells in the study area provide a comprehensive basis for assessing levels of contaminants associated with public drinking water. Results from the regulatory data set were compared against a secondary data source from United States Geological Survey, providing an independent confirmation of the types and patterns of contamination. When contaminants were detected as a result of this effort, a third data source from NYS DOH was used to determine whether contaminants were in the distribution system and to determine the likely geographical area where contaminants were detected.

In general, analyses of these data sets indicate that very few contaminants were detected, and only at low levels, in public drinking water in the study area. When compared to other areas in Suffolk County, both median contaminant levels and rates of detection were low. Nevertheless, the level of community concern and the high quality and comprehensive nature of the public drinking water data allowed NYS DOH researchers to complete analyses for specific contaminants by source and distribution areas. The result was the identification of a few isolated areas that experienced historical levels of contamination that would have exceeded current standards of 5 mcg/L in four locations. The four areas assessed are Scott's Beach, Soundview Association, Coram Municipal Office Building and Crystal Brook.

a. 1,1,1-Trichloroethane (1,1,1-TCA)

1,1,1-TCA was detected in drinking water samples collected in all four of the listed areas. 1,1,1-TCA levels were at times detected at Scott's Beach, Soundview Association and the Coram Municipal Office Building at levels that

exceeded the current regulatory standard of 5 mcg/L. 1,1,1-TCA was classified as “not classifiable as risk factor for human breast cancer” (Category 3). However, a more detailed analysis has been done to determine if 1,1,1-TCA is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

Although the levels were not elevated in the CMP area, as compared to other areas of Suffolk County, the regulatory data indicate that exposure to low levels of 1,1,1-TCA were likely in limited areas within the CMP area. The specific areas and the resident populations served within these areas are estimated and presented in *Section IV E-6*.

1,1,1-TCA is an organic liquid with a chloroform-like odor. It is largely used as a solvent for removing grease from machined metal products, in textile processing and dyeing and in aerosols. 1,1,1-TCA is likely to enter the environment by evaporation or in wastewater from its production or use in metal cleaning. It can also enter the environment in leachates and volatile emissions from landfills. 1,1,1-TCA evaporates rapidly from water and soil. It does not bind to soils nor is it broken down by microbial action; so it may leach to ground water. It has little tendency to accumulate in aquatic life (US EPA, 2004; ATSDR, 1995). The specific sources of 1,1,1-TCA that contaminated the public wells in Scott’s Beach, Soundview Association, Coram Municipal Office Building and Crystal Brook were not determined for this report.

The data used in the drinking water supply evaluation described in *Section IV E-6* indicate that the exposure to 1,1,1-TCA could have occurred from 1979 – 1987 (Sound View), 1979 – 1988 (Scott’s Beach), 1977 – 1995 (Crystal Brook), and 1981 – 1987 (Coram Municipal Building). Thus, this environmental data set is one of the few that allow documentation of long-term exposures. Although there are no data on the presence of 1,1,1-TCA in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination pre-dated monitoring for this contaminant.

In conclusion, the degree of confidence that the 1,1,1-TCA data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *high*. This ranking is based on two determinations. (1) The 1,1,TCA data set provides direct evidence of the presence of 1,1,1-TCA in drinking water during the years covered by the data set. Thus, the data are considered a *good* estimate of residential exposures in the CMP area during this time. (2) Since it is known that 1,1,1-TCA was present in the drinking water from 1977 to 1995, and was likely present before then, the 1,1,1-TCA data are considered a *good* estimate of 1,1,1-TCA water concentrations in the CMP area during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5-40 years earlier).

Toxicity Narrative (Breast Cancer)

The classification of 1,1,1-TCA as “not classifiable as a risk factor for human breast cancer” (Category 3) is based on the level of evidence for data on humans (*inadequate*), animals (*negative*), and mode-of-action (*inadequate*). The toxicological information on which those determinations were based is summarized below.

Two small studies that included women have examined the potential association between occupational exposure to 1,1,1-TCA and various types of cancer. A retrospective worker cohort study (Anttila et al., 1995) included 131 women exposed to 1,1,1-TCA between 1975 and 1992, for whom monitoring data for blood levels of 1,1,1-TCA were available. Possible associations were found between exposure to 1,1,1-TCA and multiple myeloma (two cases) and cancers of the nervous system (3 cases). The study did not find an association between exposure to 1,1,1-TCA and breast cancer. The second study (Heineman et al., 1994), a case-control study, found a possible association between exposure to 1,1,1-TCA and brain cancer, but did not gather data on breast cancer. The human data on 1,1,1-TCA are limited by the small number of studies that evaluated the risk of breast cancer, the small sample sizes of the conducted studies, and the failure of the studies to clearly establish 1,1,1-TCA exposures. Thus, the human data are classified as *inadequate*.

Two studies examined the effects of 1,1,1-TCA on laboratory animals. In the first study (Maltoni et al., 1986), rats that were exposed for their lifetimes to diets containing 1,1,1-TCA showed a small increase in the incidence of leukemia and lymphoma, compared to the rats not exposed to 1,1,1-TCA . The rats were carefully examined for various tumors, including mammary tumors, and none were found.

In the second study (Quast et al., 1988), rats and mice were exposed for two years to one of several concentrations of 1,1,1-TCA in air. The animals were carefully examined after death for tumors in various organs. No tumors were found in male or female rats or in male mice. Female mice had benign tumors in a tear gland that humans do not have. The incidence of these tumors increased as the dose of 1,1,1-TCA increased, but the low incidence of these tumors never reached statistical significance. No mammary tumors were detected. Thus, the animal data for mammary tumors are classified as *negative.*

The mode-of-action by which 1,1,1-TCA might cause cancer was also evaluated. Most experimental data indicate that 1,1,1-TCA is not genotoxic (i.e., it does not damage DNA). However, a tiny fraction of the TCA that enters the body in food or in the air is converted to reactive metabolites that, in some test tube experiments, appear to damage DNA and chromosomes and to change normal cells into pre-cancer cells. This has not been shown to occur in an intact animal (ATSDR, 1995; IARC, 1999). Thus, the mode-of-action data are classified as *inadequate.*

Risk Narrative

(1) Cancer Effects

1,1,1-trichloroethane is “not classifiable as a risk factor” for breast cancer in humans. The US EPA and the California Environmental Protection Agency have come to a similar conclusion about the carcinogenic potential of 1,1,1-TCA. 1,1,1-TCA was placed in this category even though the results of animal studies were negative for breast cancer because limited human studies and mode-of-action studies suggest it might have the potential to cause cancer. There are some data supporting its classification as an “unlikely” risk factor for breast cancer and some data supporting its classification as a “potential” risk factor for breast cancer. The lack of positive data on carcinogenicity preclude a risk analysis of the cancer effects of 1,1,1-TCA.

(2) Non-Cancer Effects

Exposure to high levels of 1,1,1-TCA can cause non-cancer effects in humans and animals, primarily on the nervous system, liver and cardiovascular system. The potential for these and other non-cancer health effects was evaluated by comparing the estimated 1,1,1-TCA water concentration in the CMP area to a water concentration that is generally used to set standards or guidelines for contaminants. This concentration corresponds to the reference dose, which is the daily dose of 1,1,1-TCA that is expected to be without an appreciable risk of non-cancer health effects (*Table 41*).

Table 41. Risk reference values for non-cancer effects of 1,1,1-trichloroethane

Reference dose (RfD)		Target organ	Source
mg/kg/day	mcg/L[1]		
0.28	9,800	body weight, nervous system, liver, heart	US EPA, 2003

[1]*Reference dose expressed as a drinking water concentration, calculated from: (RfD in mg/kg-day) x [(70 kg person)/(2 L water/day)] x (1000 mcg/mg).*

1,1,1-TCA was detected in the drinking water wells of Sound View (1979 - 1987), Scott's Beach (1979 - 1988), Crystal Brook (1977 - 1988), and the Coram Municipal Building (1981 - 1987). The average concentrations of 1,1,1-TCA found in samples from these wells were: 6.1 mcg/L (Sound View), 2.9 mcg/L (Scott's Beach), 0.7 mcg/L (Crystal Brook), and 8.3 mcg/L (Coram Municipal Building). The concentrations in individual samples ranged from 1 to 18 mcg/L (Sound View, Scott's Beach and Coram Municipal Building) and from 0.25 to 3 mcg/L (Crystal Brook).

The ratio of the concentration of 1,1,1-TCA in water to the water concentration at the reference dose can be used to characterize the non-cancer health risks from 1,1,1-TCA in drinking water. Based on the average concentrations found in the four areas, the ratios range from 0.00007 to 0.0008. Thus, the doses of 1,1,1-TCA potentially obtained from drinking water are only tiny fractions of the reference dose *(Table 42).* The non-cancer health risks associated with ratios below 1 are rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 42. Qualitative descriptors of potential non-cancer risk associated with past measurements of 1,1,1-TCA in the public water supplies of Sound View, Scott's Beach and Crystal Brook areas and the Coram Municipal Building

Area	Period of known contamination[1]	Average 1,1,1-TCA concentration in drinking water (mcg/L)	Qualitative descriptor of risk[2]
Sound View	1979 – 1987	6.1	minimal (0.0006)
Scott's Beach	1979 – 1988	2.9	minimal (0.0003)
Crystal Brook	1977 – 1995	0.7	minimal (0.00007)
Coram Municipal Building	1981 - 1987	8.3	minimal (0.0008)

[1]*Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected. Although there are no data on the presence of 1,1,1-TCA in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination was present at earlier times.*

[2]*Average 1,1,1-TCA concentration / 1,1,1-TCA reference dose (9,800 mcg/L, Table 41).*

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on 1,1,1-TCA water concentrations in the public water supplies within the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *high confidence* in the likelihood that the 1,1,1-TCA data accurately represent potential exposures of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis that classifies 1,1,1-TCA as "not classifiable as a risk factor for human breast cancer." This class was chosen because while some data support its classification as an "unlikely" environmental risk factor for breast cancer, other data support its classification as a "potential" environmental risk factor for breast cancer. (3) The risk analysis that indicates that the likelihood of non-cancer health risks at average water concentrations found in the public water supplies are estimated to be *minimal.* This is the lowest possible qualitative descriptor of risk used by the NYS DOH.

References

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US EPA (US Environmental Protection Agency. (2004). Consumer Factsheet on: 1,1,1-trichloroethane. Office of Drinking Water. (http://www.epa.gov/safewater/contaminants/dw_contamfs/111-tric.html).

b. 1,1-Dichloroethane (1,1-DCA)

1,1-DCA was detected in drinking water samples collected in all four of the listed areas of the CMP area. At Soundview and Coram Municipal Office Building, 1,1-DCA levels, at times exceeded the current regulatory

standard of 5 mcg/L. 1,1-DCA is classified as a " potential risk factor for human breast cancer"(Category 2C). A more detailed analysis has been done to determine if 1,1-DCA is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

Although the levels were not elevated in the CMP area, as compared to other areas of Suffolk County, Suffolk County regulatory data indicate that exposure to low levels of 1,1-DCA was likely in limited areas within the CMP area. The specific areas and the resident populations served within these areas are estimated and presented in *Section IV-6*.

1,1-DCA is a colorless, oily liquid with a sweet odor. It is used primarily to make other chemicals, to dissolve substances such as paint, varnish, and finish removers, and to remove grease. 1,1-DCA evaporates from water rapidly into air. Sources of 1,1-DCA include releases from industrial processes, as well as the breakdown of 1,1,1-trichloroethane (ATSDR, 1990). 1,1,1-TCA was also present whenever 1,1-DCA was detected in wells in the study area, suggesting that the source of 1,1-DCA in these wells may be either from the breakdown of 1,1,1-TCA (ATSDR, 1990) or from the same source that led to 1,1,1-TCA contamination.

The data used in the drinking water supply evaluation described in *Section IV E-6* indicate that the exposure to 1,1-DCA could have occurred from 1981 – 1987 (Sound View and Coram Municipal Building), 1987 – 1988 (Scott's Beach), and 1987 – 1991 (Crystal Brook). Thus, this environmental data set is one of the few that allow documentation of long-term exposures. Although there are no data on the presence of 1,1-DCA in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination pre-dated monitoring for this contaminant.

In conclusion, the degree of confidence that the 1,1-DCA data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *high*. This ranking is based on two determinations. (1) The 1,1-DCA data set provides direct evidence of the presence of 1,1-DCA in drinking water during the years covered by the dataset. Thus, the data are considered a *good* estimate of residential exposures in the CMP area during this time. (2) Since it is known that 1,1-DCA was present in the drinking water from 1981 to 1991, and was likely present before then, the 1,1-DCA data are considered a *good* estimate of 1,1-DCA water concentrations in the CMP area during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5 - 40 years earlier).

Toxicity Narrative (Breast Cancer)

The classification of 1,1-DCA as "a potential risk factor for human breast cancer" (Category 2C) is based on the level of evidence for data on humans (*inadequate*), animals (*inadequate*), and mode-of-action (*limited*). The toxicological information on which those determinations were based is summarized below.

Information on human carcinogenicity of 1,1,-DCA was not found in the U.S. Environmental Protection Agency's *Integrated Risk Information System database for 1,1-DCA*, *or* the Agency for Toxic Substances and Disease

Registry (ATSDR, 1990) Toxicological Profile for 1,1-Dichloroethane. A search of the electronic database of the National Library of Medicine (PUBMED) did not find any published papers on the human carcinogenicity of 1,1-dichloroethane. Thus, the human data are classified as *inadequate*.

The only long-term carcinogenicity study of 1,1-DCA in rats and mice (NCI, 1978) had serious methodological problems (US EPA, 2004). Most (80%) of the rats in the study got pneumonia, which was probably unrelated to the chemical exposure, and many died before the end of the study. This reduces confidence in the results because sick animals may not respond the same way as healthy animals, and may not live long enough to develop cancer even if exposed to a chemical that causes cancer. Nevertheless, the female rats that survived for at least 52 weeks (21% of those initially in the study) showed a statistically significant dose-related trend for an increased incidence of mammary gland adenocarcinomas. The high-dosed female mice who survived 90 weeks (50% of those initially in the study) did not show any mammary tumors, but did show a slightly increased incidence of benign polyps of the uterus. Male mice showed an increase in liver cancer. Given the methodological problems of the study, the animal data are classified as *inadequate* even though 1,1-DCA showed some potential of causing mammary tumors in female rats.

Data on the mode-of-action by which 1,1-DCA might cause cancer were also evaluated. Several studies have shown that 1,1-DCA is weakly genotoxic in bacteria and to liver cells in culture and is capable of forming DNA adducts. Despite these data obtained *in vitro*, little if any evidence indicate that 1,1-DCA is capable of initiating the carcinogenesis process in an intact animal. However rats in which liver carcinogenesis had already been initiated by another carcinogen (diethyl nitrosamine), and then treated with1,1-DCA, showed an increased rate of formation of colonies of liver cells that are believed to be early markers of carcinogenesis (Story et al., 1986; Milman et al., 1988). These studies suggest that 1,1-DCA may act as a promoter, accelerating the carcinogenesis process. Thus, the mode-of-action data are classified as *limited*.

Risk Narrative

(1) Cancer Effects

One study indicated that long-term exposures to high levels of 1,1-DCA might cause cancer in animals (NCI, 1978), but the methodological problems of the study preclude the conclusion that 1.1-DCA unequivocally caused the cancers. The results of this study were analyzed and expressed as the 1,1-DCA water concentration (6 mcg/L) that is associated with an estimated excess lifetime human cancer risk of one in one million (1×10^{-6}), assuming continuous lifetime exposure *(Table 43).* This estimate was used because it was based on breast cancer, even though the methodological problems of the study (NCI, 1978) weaken confidence in the estimate.

Table 43. Risk reference values for cancer effects of 1,1-dichloroethane

One in One Million (1×10^{-6}) Risk Level Water Concentration[1]	Cancer type	Source
6 mcg/L	breast	CA EPA, 2003

[1]Calculated from the oral cancer potency factor [0.0057 (mg/kg-day)$^{-1}$]

1×10^{-6} Risk Level = [(1×10^{-6}) / 0.0057 (mg/kg-day)$^{-1}$] x [70 kg / (2 L/day)] x 1000 mcg/mg

If a person is exposed for an entire lifetime to the average concentration of 1,1-DCA during the period of known contamination of the wells from three areas of the CMP and at the Coram Municipal Building, the estimated lifetime excess cancer risks from daily exposures would be 7×10^{-7}, 3×10^{-7}, 6×10^{-7} for Sound View, Scott's Beach, and Crystal Brook, respectively, and 4×10^{-7} for the Coram Municipal Building *(Table 44).* These risk estimates are lower than the excess risk level (one in one million or 1×10^{-6}) that is generally used to set guidelines or standards. Other risk estimates are presented in *Table 44*. All the estimated cancer risks are rated "very low" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 44. Qualitative descriptors of potential cancer risk associated with past measurements of 1,1-DCA in the public water supplies of Sound View, Scott's Beach, Crystal Brook, and the Coram Municipal Building.

Area	Period of known contamination[1]	Average 1,1-DCA concentration in drinking water	Qualitative descriptor of excess lifetime cancer risk (risk ratio) for different exposure periods[2]		
		(mcg/L)	70 years (lifetime)	30 years[3]	period of known contamination
Sound View	1981 – 1987	4.2	very low (7×10^{-7})	very low (3×10^{-7})	very low (7×10^{-8})
Scott's Beach	1987 - 1988	1.6	very low (3×10^{-7})	very low (1×10^{-7})	very low (7×10^{-9})
Crystal Brook	1987 - 1991	0.39	very low (6×10^{-8})	very low (3×10^{-8})	very low (4×10^{-9})
Coram Municipal Building	1981 – 1987	2.5	very low (4×10^{-7})	very low (2×10^{-7})	very low (4×10^{-8})

[1]Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected. Although there are no data on the presence of 1,1-DCA in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination was present.

[2]Risk ratio (RR) is calculated from: average 1,1-DCA concentration in drinking water (mcg/L), the one in one million risk level (Table 43) and the proportion of a lifetime that people are exposed (e.g. 70/70 for 70 years, 30/70 for 30 years, 7/70 for 7 years.

RR = (1×10^{-6}) X [1,1-DCA concentration (mcg/L) / one in one million risk level (mcg/L)] x (years exposed/ 70 years).

See Appendix V-1 for qualitative descriptors associated with various risk ratios.

[3]US EPA Exposure Factor Handbook recommended 95th percentile for residence time [i.e., the length of time that people live in one residence (US EPA, 1999)].

(2) Non-Cancer Effects

Exposure to high levels of 1,1-DCA also causes non-cancer effects in animals. 1,1-DCA damages the kidneys and has caused delayed growth in the offspring of animals exposed during pregnancy (ATSDR, 1990). The potential risk of these and other non-cancer health effects was evaluated by comparing the estimated 1,1-DCA water concentration in the CMP area to a water concentration that is generally used to set standards or guidelines for contaminants. This concentration corresponds to the reference dose, which is the daily dose of 1,1-DCA that is expected to be without an appreciable risk of non-cancer health effects (*Table 45*).

Table 45. Risk reference values for non-cancer effects of 1,1-dichloroethane

Reference dose (RfD)		Target organ	Source
mg/kg-day	mcg/L[1]		
0.1	3,500	kidney	US EPA, 1997

[1]*Reference dose expressed as a drinking water concentration, calculated from:*
(RfD in mg/kg-day) x [(70 kg person) / (2 L water/day)] x (1000 mcg/mg).

1,1-DCA was detected in the drinking water wells of Sound View (1981 - 1987), Scott's Beach (1987 - 1988), Crystal Brook (1987 - 1991), and the Coram Municipal Building (1981 - 1987). The average concentrations of 1,1-DCA found in samples from these wells were: 4.2 mcg/L (Sound View), 1.6 mcg/L (Scott's Beach), 0.39 mcg/L (Crystal Brook), and 2.5 mcg/L (Coram Municipal Building) *(Table 46).* The highest concentrations in individual samples were: 8 mcg/L (Sound View), 4 mcg/L (Scott's Beach), 2 mcg/L (Crystal Brook), and 7 mcg/L (Coram Municipal Building).

The non-cancer health risks from 1,1-DCA in drinking water are characterized from the ratio of a CMP water concentration to the water concentration at the reference dose. These ratios for the average concentrations found in the four water supplies range from 0.001 to 0.0001. Thus, the doses of 1,1-DCA potentially obtained from drinking water are only very small fractions of the reference dose *(Table 46).* The non-cancer health risks associated with ratios below 1 are rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 46. Qualitative descriptors of potential non-cancer risk associated with past measurements of 1,1-DCA in the public water supplies of Sound View and the Coram Municipal Building

Area	Period of known contamination[1]	Average 1,1-DCA concentration in drinking water (mcg/L)	Qualitative descriptor of risk[2]
Sound View	1981 – 1987	4.2	minimal (0.001)
Scott's Beach	1987 - 1988	1.6	minimal (0.0005)
Crystal Brook	1987 - 1991	0.39	minimal (0.0001)
Coram Municipal Building	1981 – 1987	2.5	minimal (0.0007)

[1]Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected. Although there are no data on the presence of 1,1-DCA in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination was present at earlier times.

[2]Average 1,1-DCA concentration / 1,1-DCA reference dose (3,500 mcg/L, Table 45).

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on 1,1-DCA water concentrations in the public water supplies within the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *high confidence* in the likelihood that the 1,1-DCA data accurately represent potential exposures of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis that classifies 1,1-DCA as a "*potential* risk factor for human breast cancer." (3) The risk analysis that shows that the likelihood of health risks for 1,1-DCA at water concentrations found in the public water supplies are estimated to be *very low* for cancer risks and *minimal* for non-cancer risks. These are the lowest possible qualitative descriptors of risk used by the NYS DOH.

e. References

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c. Carbon Tetrachloride

Carbon tetrachloride was detected at a higher frequency in drinking water samples collected from the CMP area, compared to samples collected from all other wells in Suffolk County. Within the CMP area, all but two carbon tetrachloride detections were in the Crystal Brook wells. The level of carbon tetrachloride in the Crystal Brook water samples was low. Carbon tetrachloride is classified as “a potential risk factor for human breast cancer” (Category 2C). A more detailed analysis has been done to determine if carbon tetrachloride is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

Although the levels were not elevated in the Crystal Brook area, as compared to other areas of Suffolk County, the Suffolk County data indicate that exposure to low levels of carbon tetrachloride was more likely in the area served by the Crystal Brook wells than elsewhere in Suffolk County. These wells are unique in the study area. The specific area and the resident populations served within these areas are estimated and presented in the *Section IV E-6*.

Carbon tetrachloride is a clear heavy organic liquid with a sweet aromatic odor similar to chloroform. Most of it is used to make chlorofluorocarbon propellants and refrigerants, although this use has been declining steadily. It is also used as dry cleaning agent, a fire extinguisher, and a solvent in many applications, including rubber cement, soaps, and insecticides. Carbon tetrachloride can enter the environment in leachates and volatile emissions from landfills, in wastewater from industries, and from agricultural activities. Carbon tetrachloride evaporates quickly from surface waters and soil. It does not bind to soil and may therefore leach into ground water. It has a low potential to accumulate in aquatic life (US EPA, 2004a).

The data used in the drinking water supply evaluation described in *Section IV E-6* indicate that the exposure to carbon tetrachloride could have occurred from 1977 – 1988 in one public water supply (Crystal Brook). Thus, this environmental data set is one of the few that allow documentation of long-term exposures. Although there are no data on the presence of carbon tetrachloride in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination pre-dated monitoring for this contaminant.

In conclusion, the degree of confidence that the carbon tetrachloride data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *high*. This ranking is based on two determinations. (1) The carbon tetrachloride data set provides direct evidence of the presence of carbon tetrachloride in drinking water during the years covered by the data set. Thus, the data are considered a *good* estimate of residential exposures in the CMP area during this time. (2) Since it is known that carbon tetrachloride was present in the drinking water from 1977 to 1988 and was likely present before then, the carbon tetrachloride data are considered a *good* estimate of carbon tetrachloride water concentrations in the CMP area during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5-40 years earlier).

Toxicity Narrative (Breast Cancer)

The classification of carbon tetrachloride as “a potential risk factor for human breast cancer” (Category 2C) is based on the level of evidence for data on humans (*inadequate*), animals (*inadequate*), and mode-of-action (*strong*). The toxicological information on which those determinations were based is summarized below.

The potential of carbon tetrachloride to increase the risk of breast cancer has been investigated in three occupational studies. In the first study (Blair et al., 2003), 4,049 women who had been employed as dry cleaners (in jobs where carbon tetrachloride was used) for at least one year between 1948 and 1978 were enrolled (based on union records) and then traced for mortality and cause of death until 1993. Exposure was estimated from job title and published monitoring studies for the dry cleaning industry. The observed number of breast cancer deaths (68) was the same as the number expected (based on age-specific mortality rates from the general U.S. population), indicating that (for the total cohort) exposure to dry cleaning solvents had had no effect on the number of women who died of breast cancer. Slightly elevated rates of breast cancer (which were not statistically significant) were found for Black women and for women with estimated medium to high level exposure to dry cleaning solvents (Blair et al., 2003).

Similar results were found in the second study (Blair et al., 1998) in which 1,667 female aircraft maintenance workers who had been employed for at least one year and judged likely to have been exposed to various solvents and other chemicals, were traced for 34 to 38 years for mortality and cause of death. The 18 observed breast cancer deaths in women "ever exposed" to carbon tetrachloride was slightly, but not statistically, elevated compared to the general population of Utah. These studies have several limitations that limit confidence in the findings, including exposure to mixed and various solvents, little or no quantitative assessment of exposure to carbon tetrachloride, a relatively small number of cases, and failure to use an appropriate control group of unexposed workers.

A third study (Cantor et al., 1995) gathered a large amount of data from death certificates that indicated the cause of death to have been breast cancer, including occupational history (industry and occupation), and then developed a method of estimating the probability and level of a number of workplace exposures to various chemicals. The data suggest that women exposed to moderate to high levels of carbon tetrachloride had about a 20% increase in mortality from breast cancer compared to occupational controls. Although the increased risk (based on 1,695 breast cancer cases) was statistically significant, the effect was apparent only when the data were adjusted for socioeconomic status. The methods used in this study are not regarded as capable of yielding definitive results, but only of suggesting directions for further studies. A further limitation of all of these studies is that they are based solely on mortality from breast cancer. An increased incidence of non-lethal breast cancer would not have been detected.

Thus, the human data are classified as *inadequate*.

Animal studies consistently show that carbon tetrachloride causes liver tumors in rats and mice. Most of these studies, however, did not report whether or not carbon tetrachloride also induced mammary tumors. The only

reported induction of breast tumors was in an early study of rats that received repeated injections of large amount of carbon tetrachloride for two years (IARC, 1979). The use of injections, which permits carbon tetrachloride to be distributed to the tissues without first being exposed to metabolism and elimination by either the lungs or liver, makes the results of uncertain relevance to typical human exposures in air or water. Thus, the animal data are classified as *inadequate*.

Data on the mode-of-action by which carbon tetrachloride might cause cancer were also evaluated. When animals and humans are exposed by inhalation, carbon tetrachloride is absorbed into the blood and rapidly distributed to most tissues. Carbon tetrachloride has been found in human milk (Fisher et al., 1997), indicating that it is distributed to human breast. In humans, an enzyme present in breast tissue metabolized carbon tetrachloride into highly reactive metabolites that can damage DNA and which are known to be highly toxic to cells. These metabolites of carbon tetrachloride, not only produce some mutations by themselves, but they also change the structure of the DNA to facilitate the formation of additional mutations by other chemicals. Thus carbon tetrachloride can potentially act as an initiator of carcinogenesis, and also as a promoter, speeding up the carcinogenesis process (IARC,1999). The mode-of-action data suggest a plausible way in which carbon tetrachloride at high levels of exposure could increase the risk of breast cancer; thus the data are classified as *strong*.

Risk Narrative

(1) Cancer Effects

Long-term exposure to high levels of carbon tetrachloride causes cancer in laboratory animals. One estimate of the carbon tetrachloride water concentration that is associated with an estimated excess lifetime human cancer risk of one in one million (1×10^{-6}) is 0.3 mcg/L, assuming continuous exposures (see *Table 47).* This estimate is based on cancer of the liver, for which there is the most evidence of carcinogenicity from carbon tetrachloride. No unit risk has been derived for carbon tetrachloride based on breast cancer because carbon tetrachloride increased the incidence of mammary cancers only in a single study in rats treated by injection (a route of administration of uncertain relevance to humans). Because the liver is the most sensitive organ to carbon tetrachloride, it was used as a worst-case surrogate for breast cancer.

Table 47. Risk reference values for carbon tetrachloride

One in One Million (1×10^{-6}) Risk Level: Water Concentration	Cancer type	Source
0.3 mcg/L	liver	EPA, 2004

If a person is exposed for an entire lifetime to water containing the average concentration of carbon tetrachloride found during the period of known contamination in the wells in the Crystal Brook area, the estimated lifetime excess cancer risk from this daily water exposure would be ten in one million or 1×10^{-5} *(Table 48).* This estimated risk is about 10 times higher than the excess risk level (one in one million or 1×10^{-6}) that is generally used to set guidelines or standards. Risk estimates for shorter periods of exposure are lower and are presented in *Table 48*. All

of these estimated cancer risks are rated “low” on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 48. Qualitative descriptors of potential cancer risk associated with past measurements of carbon tetrachloride in the Crystal Brook public water supply

Period of known contamination[1]	Average carbon tetrachloride concentration in drinking water	Qualitative descriptor of excess lifetime cancer risk (risk ratio) for different exposure periods[2]		
	(mcg/L)	70 years (lifetime)	30 years[3]	period of known contamination
1977 – 1988	2.8	low (1×10^{-5})	low (4×10^{-6})	low (2×10^{-6})

[1]Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected. Although there are no data on the presence of carbon tetrachloride in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination was present at earlier times.

[2]Risk ratio (RR) is calculated from: average carbon tetrachloride concentration in drinking water (mcg/L), the one in one million risk level (Table 48) and the proportion of a lifetime that people are exposed (e.g. 70/70 for 70 years, 30/70 for 30 years, 7/70 for 7 years.

RR = ($1x10^{-6}$) X [carbon tetrachloride concentration (mcg/L) / one in one million risk level (mcg/L)] x (years exposed/ 70 years).

See Appendix V-1 for qualitative descriptors associated with various risk ratios.

[3]US EPA Exposure Factor Handbook recommended 95th percentile for residence time [i.e., the length of time that people live in one residence (US EPA, 1999)].

(2) Non-Cancer Effects

Exposure to high levels of carbon tetrachloride also causes non-cancer effects in humans and/or laboratory animals, including damage to the nervous system, liver and kidney, and damage to the male reproductive system (ATSDR, 1994). The potential risk of these and other non-cancer health effects was evaluated by comparing the estimated carbon tetrachloride water concentration in the CMP area to a water concentration that is generally used to set standards or guidelines for contaminants. This concentration corresponds to the reference dose, which is the daily dose of carbon tetrachloride that is expected to be without an appreciable risk of non-cancer health effects (*Table 49*).

Table 49. Risk reference values for non-cancer effects of carbon tetrachloride

Reference dose (RfD)		Target organ	Source
mg/kg-day	mcg/L[1]		
7×10^{-4}	24	liver	EPA, 2004b

[1]Reference dose expressed as a drinking water concentration, calculated from: (RfD in mg/kg-day) x [(70 kg person) / (2 L water/day)] x (1000 mcg/mg).

Carbon tetrachloride was detected in the drinking water wells of the Crystal Brook area of CMP. The average concentration found in 48 water samples collected between 1977 and 1988 was 2.8 mcg/L and the range of concentrations was 0.5 to 9 mcg/L.

The non-cancer health risks from carbon tetrachloride in drinking water are characterized from the ratio of the Crystal Brook water concentration to the water concentration at the reference dose. This ratio for the average concentration found in the Crystal Brook area is 0.1. Thus, the dose of carbon tetrachloride obtained from drinking water is only about 10% of the reference dose *(Table 50).* The non-cancer health risks associated with a ratio below 1 are rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 50. Qualitative descriptors of potential non-cancer risk associated with past measurements of carbon tetrachloride in the Crystal Brook public water supplies

Period of known contamination[1]	Average carbon tetrachloride concentration in drinking water (mcg/L)	Qualitative descriptor of risk[2]
1977- 1988	2.8	minimal (0.1)

[1]Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected. Although there are no data on the presence of carbon tetrachloride in the affected wells prior to the late 1970s (when monitoring started), it is likely that contamination was present at earlier times.

[2]Average carbon tetrachloride concentration / carbon tetrachloride reference dose (24 mcg/L, Table 49).

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on carbon tetrachloride water concentrations in the public water supplies within the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *high confidence* in the likelihood that the carbon tetrachloride data accurately represent potential exposures of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997 (i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis classifies carbon tetrachloride as a "*potential* risk factor for human breast cancer." (3) The risk analysis that indicates that the likelihood of health risks at water concentrations found in the public water supplies are estimated to be *low* for cancer risks and *minimal* for non-cancer risks. These are the second lowest and lowest, respectively, possible qualitative descriptors of risk used by the NYS DOH.

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d. Benzene

Like carbon tetrachloride, benzene was detected at a higher frequency in drinking water samples collected from the CMP area, compared to samples collected from all other wells in Suffolk County. The highest benzene levels were in the Crystal Brook wells, but the level of benzene in these water samples (detectable only during 1986-1987) was still low. Benzene is classified as "a probable risk factor for human breast cancer" (Category 2A). A more detailed analysis has been done to determine if benzene is likely to be a major risk factor for breast cancer, or other health effects, in the CMP area.

Exposure Narrative

Though not elevated when compared to other areas of Suffolk County, Suffolk County data indicate that that exposure to low levels of benzene were likely in the area served by the Crystal Brook wells. These wells are unique in the study area. The specific area and the resident populations served within these areas were estimated to be the same as for carbon tetrachloride and were estimated and presented in the *Section IV E-6*.

Benzene is a clear, colorless aromatic liquid. It is highly flammable. The greatest use of benzene is as a building block for making plastics, rubber, resins and synthetic fabrics like nylon and polyester. It has been used as a solvent, for example in printing, paints, and dry cleaning. It is also found in gasoline (US EPA, 2004a).

Benzene is released to air primarily from fumes and exhaust connected with its use in gasoline. Other sources are fumes from its production and use in manufacturing. In addition, there are discharges into water from industrial effluents and losses during spills. When benzene is released to soil, it either evaporates very quickly or leaches into groundwater. It is degraded by some microbes in soil and in some ground waters (US EPA, 2004a).

Sampling data indicate that exposure to benzene was limited to, at most, two years. Benzene was only detected in the Crystal Brook system within a single five-month period in 1986 and 1987. Benzene was looked for, but not found, in system water samples collected from the late-1970s (when the regulatory monitoring for benzene began) to 1985. After 1987, benzene was looked for, but not found in system water samples.

In conclusion, the degree of confidence that the benzene data accurately represent exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *high*. This ranking is based on two determinations. (1) The benzene data set provides direct evidence of the presence of benzene in drinking water during the years covered by the data set. Thus, the data are considered a *good* estimate of residential exposures in

the CMP area during this time. (2) The data are considered a *good* estimate of benzene water concentrations in the CMP area during the years important to the start and development of breast cancers reported in 1993-1997 (i.e., perhaps 5 - 40 years earlier) because there was evidence of contamination from 1986 - 1987.

Toxicity Narrative (Breast Cancer)

The classification of benzene as a "probable risk factor for human breast cancer" (Category 2A) is based on the level of evidence for data on humans (*limited*), animals (*sufficient*) and mode-of-action (*strong*). The toxicological information used as the basis for those determinations is summarized below.

The possible association between exposure to benzene and human breast cancer has been investigated for a number of occupational groups in Shanghai, China, where occupational levels of exposure to benzene have been higher than in the United States. Petralia et al. (1998) evaluated the relative incidence of breast cancer for women in various occupations and found that between 1980 and 1984 the incidence was elevated almost two-fold among rubber manufacturing workers (22 cases), over three-fold among scientific research workers (8 cases), and seven-fold among doctors of Chinese medicine (19 cases). Each of these elevated risks were statistically significant. They then examined the association between the likelihood of occupational benzene exposure and the risk of breast cancer. They found that workers in occupations that were exposed to moderate to high levels of benzene, or that had a high probability of exposure to benzene, also had a 30% increased risk of breast cancer compared to the general population of Shanghai (65 cases). This elevated risk level was statistically significant. In contrast, groups exposed to lower levels of benzene, or with a low probability of occupational exposure to benzene, did not have an increased risk of breast cancer.

A recent study of women occupationally exposed to benzene in Western New York (Petralia et al., 1999) found that workers with a long duration or medium-to-high probability of exposure to benzene also had an two-to-three fold elevated risk of pre-menopausal breast cancer, which was statistically significant (236 cases). These data were based on lifetime occupational histories and were adjusted for age, education, and reproductive and medical history. However, the measure of exposure was considered to be somewhat crude. Furthermore, it was not possible to exclude the possibility that other chemical exposures contributed to the risk of breast cancer.

Two worker studies, on the other hand, suggest that exposure to benzene may not increase the mortality rate from breast cancer. The first (Paci et al., 1989) was of 876 women who had been employed in an Italian shoe-manufacturing plant, 51 of whom had died by the end of the study, only 4 of breast cancer. The second study (Yin et al., 1996) involved 36,000 (mostly young) women from 12 cities in China who were exposed to benzene (and other chemicals) in various occupations. At the conclusion of the study only 272 of these women had died, only 8 of breast cancer. These are too few deaths and too few breast cancer cases to form the basis of any conclusions.

Thus, the human data are classified as *limited*.

Benzene causes mammary cancers in female mice, based on data from three different strains that were studied in two different laboratories. The results were unequivocal. The incidence of carcinomas in B6C3F_1 mice exposed to

benzene showed a dose-related increase ($p<0.001$ for the trend and $p<0.001$ for the incidence at the highest dose (Huff et al., 1989)). Similar results were obtained with the other two strains (Maltoni et al., 1989).

The mouse data are supported by data showing that, in one strain of rats (the Sprague-Dawley) benzene (by oral ingestion) caused a small increase in the incidence of mammary cancer of marginal statistical significance ($p<0.10$) (Maltoni et al., 1989; CA EPA, 2002). A similar marginally increased incidence of mammary carcinomas was shown (in both dams and offspring) when benzene was administered by inhalation to pregnant Sprague-Dawley rats, and then to the resulting offspring (Maltoni et al, 1989). Other strains of rats (Wistar and Fischer 344), on the other hand, showed no increased incidence of mammary cancer. Benzene causes cancers of numerous tissues in both mice and rats, most consistently carcinomas of the Zymbal gland in all strains of mice and rats tested (Maltoni et al, 1989; Huff et al., 1989). Benzene's potential to cause cancers of estrogen-responsive tissues is suggested by an increase in the incidence of cancers of the uterus in Fischer rats and of the ovary (as well as the mammary gland) in $B6C3F_1$ mice (Huff et al., 1989).

Based on the induction of mammary cancers in three strains of mice, with supporting data in rats, the animal data are classified as *sufficient.*

Data on the mode-of-action by which benzene might cause cancer were also evaluated. Benzene absorbed by the body is distributed to fatty tissues, particularly those that are well-supplied with blood, such as breast tissue. Benzene is converted to metabolites by enzymes that are present in the liver and the mammary glands of mice, rats, and humans. Some of these metabolites are highly reactive and capable of damaging DNA, or of preventing the repair of DNA damage caused by other cancer causing chemicals (US EPA, 2002). Thus benzene and its metabolites have the potential to act both as initiators and promoters of cancer. The mode-of-action data are classified as *strong.*

Risk Narrative

(1) Cancer Effects

Long-term exposure to high levels of benzene causes cancer in laboratory animals. One estimate of the benzene water concentration that is associated with an estimated excess lifetime human cancer risk of one in one million (1×10^{-6}) is 0.5 mcg/L, assuming continuous exposures (see *Table 51).* This estimate was based on the induction of mammary tumors in female rats.

Table 51. Risk reference value for cancer effects of benzene

One in One Million (1×10^{-6}) Risk Level: Water Concentration[1]	Cancer type	Source
0.5 mcg/L	breast	CA EPA, 2001

[1] *Calculated from the oral cancer potency factor [0.07 $(mg/kg\text{-}day)^{-1}$]:*

1×10^{-6} Risk Level = [(1×10^{-6}) / 0.07 $(mg/kg\text{-}day)^{-1}$] x [70kg / (2 L/day)] x 1000 mcg/mg

If a person is exposed for an entire lifetime to water containing the average concentration of benzene found during the period of known contamination in the wells of the Crystal Brook area, the estimated lifetime excess cancer risk from this daily water exposure would be 8 x 10^{-6} *(Table 52)*. The somewhat higher average concentration (6.3 mcg/L, found between December 1986 and April 1987) corresponds to an estimated lifetime excess cancer risk of 10 x 10^{-6} *(Table 52)*. These estimated risks are about 8 to 10 times higher than the excess risk level (one in one million or 1 x 10^{-6}) that is generally used to set standards or guidelines. Risk estimates for shorter periods of exposure are lower and are presented in *Table 52*. These estimated lifetime cancer risks are rated as "low" (lifetime risks of lifetime or 30-year exposures) or "very low" (lifetime risks of exposure only during the period of documented elevated levels) on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 52. Qualitative descriptors of potential cancer risk associated with past measurements of benzene in the Crystal Brook public water supplies

Period of known contamination[1]	Average benzene concentration in drinking water	Qualitative descriptor of excess lifetime cancer risk (risk ratio) for difference exposure periods[2]		
	(mcg/L)	70 years (lifetime)	30 years[3]	period of known contamination
1986 – 1987	3.9	low (8 x 10^{-6})	low (3 x 10^{-6})	very low (2 x 10^{-7})
December 1986 – April 1987	6.3	low (1 x 10^{-5})	low (5 x 10^{-6})	very low (8 x 10^{-8})

[1]Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected, or if the dates cover less than one year, the first day of the first month to the last day of the last month.

[22]Risk ratio (RR) is calculated from: a verage benzene concentration in drinking water (mcg/L), the one in one million risk level (Table 51) and the proportion of a lifetime that people are exposed (e.g. 70/70 for 70 years, 30/70 for 30 years, 7/70 for 7 years.

RR = (1x10^{-6}) X [benzene concentration (mcg/L) / one in one million risk level (mcg/L)] x (years exposed/ 70 years).

See Appendix V-1 for qualitative descriptors associated with various risk ratios.

[3]US EPA Exposure Factor Handbook recommended 95th percentile for residence time [i.e., the amount of amount of time people live in one residence (US EPA, 1999)].

(2) Non-Cancer Effects

Exposure to high levels of benzene also causes non-cancer effects in humans and animals, including damage to the blood-cell-forming tissues and to the immune and nervous system (ATSDR, 1997). The potential risk of these and other health effects was evaluated by comparing the estimated benzene water concentration in the CMP area to a water concentration that is generally used to set standards or guidelines for contaminants. This concentration corresponds to the reference dose, which is the daily dose of benzene that is expected to be without an appreciable risk of non-cancer health effects (*Table 53*).

Table 53. Risk reference values for non-cancer effects of benzene

Reference dose (RfD)		Target organ	Source
mg/kg-day	mcg/L[1]		
0.004	140	immune system	US EPA, 2004b

[1] *Reference dose expressed as a drinking water concentration, calculated from:*

(RfD in mg/kg-day) x [(70 kg person)/(2 L water/day)] x (1000 mcg/mg).

Benzene was detected in the drinking water wells of the Crystal Brook area of CMP between 1986 and 1987. The average concentration found in 11 water samples collected during this time was 3.9 mcg/L and the range of concentrations was 0.5 to 12 mcg/L. Between December 1986 and April 1987, the average concentration found in six water samples was 6.3 mcg/L and the maximum was also 12 mcg/L (*Table 54).*

The non-cancer health risks from benzene in drinking water are characterized from the ratio of the Crystal Brook water concentration to the water concentration at the reference dose. These ratios for the average concentrations found during 1986 - 1987 and December, 1986 – April, 1987, are 0.03 and 0.04, respectively. Thus, the doses of benzene obtained from drinking water are only about 3% - 4% of the reference dose *(Table 54).* The non-cancer health risks associated with ratios below 1 are rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

Table 54. Qualitative descriptors of potential non-cancer risk associated with past measurements of benzene in the Crystal Brook public water supplies

Period of known contamination[1]	Average benzene concentration in drinking water (mcg/L)	Qualitative descriptor of risk[2]
1986 - 1987	3.9	minimal (0.03)
December 1986 - April 1987	6.3	minimal (0.04)

[1] *Exposure period is from the first day of the year that the contaminant was first detected to the last day of the year that the contaminant was last detected, or if the dates cover less than one year, the first day of the first month to the last day of the last month.*

[2] *Average benzene concentration / benzene reference dose (140 mcg/L, Table 53).*

Conclusions

This integration of the exposure and toxicity data does not support a recommendation for additional follow-up studies on benzene water concentrations in the public water supplies within the CMP area. This conclusion is based on the results of three separate analyses. (1) The exposure analysis that shows *high confidence* in the likelihood that the benzene data accurately represent potential exposures of CMP residents during the years important to the start and development of breast cancers reported in 1993-1997(i.e., perhaps 5 - 40 years earlier). (2) The literature review and analysis that classifies benzene as a "probable risk factor for human breast cancer." (3) The risk analysis that indicates that the likelihood of lifetime health risks associated with two years of exposure to benzene in drinking water at concentrations found in the public water supplies during the period of contamination

are estimated to be *very low* (for cancer risks) and *minimal* (for non-cancer risks). These are the lowest possible qualitative descriptors of risk used by the NYS DOH. A two-year exposure period was used because the monitoring data shows benzene was detected in the drinking water supplies of the Crystal Brook area in 1986 and 1987, but not before or after.

The potential for benzene exposure from other sources was also considered. Benzene is an important commercial commodity that, because of its frequent use, has become widespread in the environment of developed countries. Benzene is commonly found in indoor and outdoor air. In almost all cases, benzene levels inside residences or offices are higher than levels outside. Gasoline and gasoline-related activities are major sources of benzene exposure. Cigarette smoke is another common source of benzene exposure, which accounts for about half of the benzene to which the general population is exposed. For most people, the level of exposure to benzene through air is greater than their exposure through food, beverages, and drinking water. Thus, it is likely that benzene exposures from drinking water in the CMP area contributed a relatively small proportion of the total exposure of CMP residents to benzene during the period of contamination.

The data available at this time do not show benzene air levels in the CMP areas to be elevated compared to other areas. Two databases on benzene in air were reviewed for estimated concentrations of benzene in outdoor air. The US EPA's Cumulative Exposure Project (CEP) contained an error in the benzene data and therefore could not be used in an analysis. The US EPA's National-scale Air Toxics Assessment (NATA) database did not indicate an elevation in benzene levels for the CMP area. Both of these databases are discussed in *Section IV. E-3 Air Quality.*

In summary, the integration of the exposure and toxicity data area does not support a recommendation of additional follow-up studies on benzene in the CMP area.

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C. Results for Remaining Contaminants

1. Air Contaminants

The modeled outdoor air concentration data for 25 air contaminants in the CMP area suggest that their concentrations may be elevated compared to their concentrations in other areas in the state. These comparisons were originally presented in *Environmental Exposure Evaluation* and are presented again in *Table 55*. These estimates were derived from the US EPA's Cumulative Exposure Project (CEP, 1990) and National-scale Air Toxics Assessment (NATA, 1996, see *Appendix IV-2*). The basis and limitations of these estimated air concentrations were discussed in *Section IV. E-3. Air Quality.* One additional contaminant, ozone, was added to the list because of community concerns about the air quality in the CMP area. The Long Island area does not meet the federal air standard for ozone and has been a “non-attainment” area since an ozone standard was first introduced in the early 1970s (see, *Section IV-E.3.b* for more on ozone).

One air contaminant, ethylene thiourea (ETU) was evaluated in the *Working Draft Integration Report (see V-B Results from Working Draft Report).* ETU was chosen because it was the chemical with the highest comparison ratio. However, the integration evaluation showed that the modeled air concentration in the CMP area was substantially lower than potential health-based guidelines for ETU in air. The *Working Draft Report* contained a detailed presentation of the integration process that was used to evaluate the potential cancer and non-cancer health risks from ETU in air. The same process was used to evaluate the potential health risks for each of the remaining air contaminants. The major findings of each step in the process for each contaminant are presented in *Tables 55 — Tables 59.*

Exposure Narrative

These air contaminants were further evaluated because the environmental data suggested that their outdoor air concentrations in the CMP area might be higher than outdoor concentrations in other parts of the state. However, the CMP population also may be exposed to air contaminants from other sources. *Table 55* provides readers with

some background information on each air contaminant selected for integration. It presents common uses and sources for each contaminant along with potential routes of exposure for the general population. This information is not specific to the CMP area and is derived from a variety of sources that are provided as references. It is not intended to be a comprehensive listing of all known information for each contaminant.

Indoor air, for example, is becoming recognized as a primary source of exposure to many environmental contaminants. Attention has been focused on products of incomplete combustion (gas stoves, wood stoves, fireplaces, and tobacco smoke) and on environmental chemicals found in consumer products that are used in the home (Gammon et al., 2003; Rudel et al., 2003; Triche et al., 2005). Other sources of exposure include food, water, pharmaceutical products, and cosmetics. This information might be helpful to those concerned about other sources of exposure to CMP air contaminants. *Table 56* also provides data on the presence or absence of each contaminant in the CMP water supplies. However, the qualitative information in the table is insufficient to estimate an individual's total exposure to any one contaminant or the relative importance of any particular source or pathway of exposure.

This investigation focuses on identifying unusual factors that might be related to why breast cancer incidence was elevated in the CMP communities between 1993 and 1997 compared to other parts of New York State. For the 25 contaminants identified by the CEP or NATA dataset, researchers evaluated the degree of confidence that the 1990 (CEP) or 1996 (NATA) data accurately represents exposure during years important to evaluating elevated breast cancer rates in 1993 - 1997. The same methodologies were used that are described in the *Working Draft Report* (see *V. Integration, A. Methodology*). The confidence ranking in the exposure data for all the contaminants is *low* because they all come from similar databases. This ranking is based on two determinations. (1) Because the air estimates are based on modeled data, the CEP and NATA data are considered only a *fair* estimate of residential exposure in the CMP area during the year covered by the CEP or NATA data sets.
(2) However, during the years important to the start and development of breast cancers (perhaps 5 - 40 years before these cancers were diagnosed in 1993 - 1997), the CEP and NATA data are considered a *poor* estimate of air levels in the CMP air. This is because the validity of these modeled data for that earlier period cannot be readily determined.

Estimates of ozone concentrations in the CMP areas were not based on modeled estimates, but rather on continuous measurements by air monitoring stations in Babylon (western Suffolk County) and in Riverhead (eastern Suffolk County). Monitoring data are preferred to modeled data. The measured concentrations reported by these two stations, on either side of the CMP area, do not differ greatly (NYS DEC, 2004). Moreover, the land use patterns, traffic density, and degree of urbanization, which correlate with ozone levels, are also similar. Thus, it is reasonable to conclude that the ozone concentrations in the CMP area are likely to be similar to the values reported for Babylon and Riverhead. Thus, the monitoring data provide a *good* estimate of the level of ozone present in air of the CMP area during the years covered by the dataset.

In addition, ozone data from the Babylon monitoring station have been reported annually, at least since 1979 (NYS DEC, 1998, 2003) and indicate that ozone levels at that station decreased about 40% between 1979 and 1994 (and

very little between 1994 and 2003). Thus, the ozone data provide monitoring data for 14 – 18 years before the diagnosis of breast cancer in 1993 – 1997. These could be important years for the possible initiation of some of the diagnosed cancers among CMP residents because the latency period between first exposure and cancer could be 5 to 40 years. Overall, the ozone data are considered a *fair* to *good* estimate of air levels in the CMP air during the years important to the start and development of breast cancers reported in 1993 - 1997. Thus, the overall confidence in the exposure estimates during the time of possible cancer initiation is *high*.

Toxicity Narration (Breast Cancer)

Using the same process described in *Part III. Toxicological Evaluation*, each air contaminant[3] was categorized as a risk factor for breast cancer based on an evaluation of the available human data, animal data, and mode-of-action data *(Table 57).* Two chemicals (1,2-dibromoethane and ethylene oxide) are classified as probable risk factors for human breast cancer (Category 2A). One chemical (acrylamide) is classified as a possible risk factor for human breast cancer (Category 2B). Fourteen chemicals are classified as potential risk factors for human breast cancer (Category 2C). Nine chemicals are not classifiable (Category 3). One chemical is classified as an unlikely risk factor for human breast cancer (Category 4). The criteria for these classifications are described in *Tables 12* and *13.*

Risk Narrative

(1) Cancer Effects

Cancer risk estimates could be calculated for 13 CMP air contaminants *(Table 58).* These calculations require experimental results showing long-term exposure to high levels of a chemical caused cancer (any kind) in laboratory animals or humans and such data were not available for certain contaminants in *Table 58.*

The dose-response data describing the relationship between exposure to these contaminants and the incidence of cancer in animals or humans can be used to calculate the air concentration that is associated with an estimated excess lifetime human cancer risk of one in one million (1×10^{-6}), assuming continuous exposure. This is the air concentration that is generally used when environmental standards or guidelines are based solely on cancer effects. The same method was used as described in *Part VB-1. Outdoor Air – Ethylene Thiourea* to calculate the estimated excess lifetime human cancer risks associated with 70 years of exposure to each contaminant at its modeled CMP air concentration.

The estimated cancer risk for 10 of the 13 contaminants is less than one in one million (1×10^{-6}). Cancer risks equal to or less than 1×10^{-6} are classified as "very low" using the method NYS DOH has used to evaluate potential cancer risks from environmental contaminants *(Appendix V-1).* Three chemicals are associated with estimated cancer risks greater than one in one million. The estimated risks for acetaldehyde (3.4×10^{-6}) and cadmium (1.4×10^{-6}) are just above one in one million. These risk levels are classified as "low" (see A*ppendix V-1).* The

[3] Glycol ethers are a family of similar chemicals. The CEP air data (Table 54) are for glycol ethers (unspecified) but we evaluated the toxicity of two specific glycol ethers (Tables 56, 57, 58, and 59).

estimated risk associated with diesel particulate matter is 7.2 x 10^{-4}, and is classified as “moderate” (see *Appendix V-1).*

In all cases, the risk estimates are not based solely on dose-response data for breast cancer. Rather, the risk estimates are based typically on dose-response data for a sensitive site or sites in a sensitive animal species or in humans. For example, for acrylamide, the risk estimate is based on dose-response data for a group of four cancer types, including breast cancer. Thus, the risk estimates in *Table 58* are used as worst case surrogates for breast cancer. For certain chemicals whose risk estimates are based only on respiratory tract tumors (e.g., acetaldehyde, arsenic, beryllium, cadmium, 1,3-dichloropropene), the risk estimates are likely to be over-estimates of the risk of breast cancer. The potency of these chemicals to cause cancer in cells in the respiratory tract is likely to be greater than the potency to cause cancer in cells outside the respiratory tract. Some of the chemicals may react with chemicals in the cells of the respiratory tract and never enter the general circulation within the body. Others may be treated differently by the body once they leave the respiratory tract. This plausibility of a reduced potency for breast cancer is supported by scientific data that provide ample evidence these chemicals cause respiratory tract cancers (*Table 58*) but little, if any evidence, that they cause breast cancer *(Table 57).*

The estimated excess lung cancer risk (7.2 x 10^{-4}) associated with diesel particulate warrants additional discussion about its relevance to the risk of breast cancer. Diesel particulate matter is classified as a potential risk factor for breast cancer based on limited data from human, animal, and mode-of-action studies (see *Table 15*). Most of the evidence comes from studies of diesel exhaust (DE), which contains both diesel particulate matter and diesel exhaust gases. The gas phase is composed of many urban hazardous air pollutants, such as acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde. The particle phase also has many different compounds that can be classified by size or composition. The diesel particulates of greatest health concern are those that are in the categories of fine and ultra fine particles. These fine and ultra fine particles are composed of elemental carbon and adsorbed compounds such as polycyclic aromatic hydrocarbons (PAHs), sulfate, nitrate, and metals.

Several large human occupational studies, as well as experimental animal studies, indicate that inhaled DE (and by inference, diesel particulate matter) causes lung cancer (US EPA, 2002; CA EPA, 2002). An increased risk of breast cancer from the inhalation of diesel exhaust would require the inhaled chemicals/particles to be absorbed from the lungs into blood and transported to breast tissue. Cancer studies provide some evidence that this might occur. Some human studies suggest possible associations between exposure to DE and cancers in tissues outside the respiratory tract (e.g. bladder, gastrointestinal tract, liver, and gall bladder, lymphatic and blood-forming tissues). However, these studies do not demonstrate that the increased risks result solely from increased exposure to DE (US EPA, 2002). In animal studies, likewise, many studies have shown that DE causes lung cancer (US EPA, 2002). Only one study (Iwai et al., 1986) reported tumors (malignant lymphomas in the spleen) outside the respiratory tract (US EPA, 2002).

Additional insight into the potential carcinogenicity of DE and by inference, diesel particulate matter, is obtained by recognizing that polycyclic aromatic hydrocarbons or PAHs are found on diesel particulate matter (Bonner et al., 2005). PAHs are a group of over 100 chemicals that are formed during the incomplete burning of coal, oil, gas,

wood, garbage, or other organic substances, such as tobacco and charbroiled meat. They can also be found in natural substances such as crude oil, coal, coal tar pitch, creosote, and roofing tar. PAHs are released also by forest fires and volcanoes. PAHs are classified as probable breast cancer carcinogens (see *Table 15),* largely based on animal and mode-of-action data. These data suggest the potential for diesel particulate matter to cause breast cancer.

In spite of this evidence, additional studies on diesel particulate matter within the CMP area are not recommended. First, CMP residents do not appear to have an unusual exposure to diesel particulate matter because the modeled estimates for diesel particulate matter for the CMP area are similar to those for the rest of Suffolk County (see *Table 55).*

Secondly, NYS DOH researchers have concerns about the accuracy of the modeled air concentrations for diesel particulate matter. These estimates were based on 1996 data, the first year that US EPA included diesel particulate matter in the National Air Toxics Assessment. They were based on an inventory of diesel sources (e.g., cars, trucks, trains, planes, and farming and construction equipment) and approximation techniques to estimate the contribution of their emissions to air concentrations of diesel particulate matter. Many simplifying assumptions were made to make these estimations and these assumptions reduce confidence in the modeled air concentrations.

Finally, several other scientists are studying the potential effects of air pollution, including diesel exhaust, on human breast cancer and early results are mixed. NYS DOH staff have published a study on the possible association between living near high traffic areas (that is, near sources of diesel particulate matter, PAHs, and ozone) and the incidence of breast cancer on Long Island (Lewis-Michl et al., 1996). The study found no association between increased breast cancer risk and increased traffic volume. More recently, Nie et al. (2005), in a study of breast cancer among western New York women, presented preliminary data at a scientific meeting that linked increased exposure to PAHs from traffic occurring at the time of menarche with an increased risk of premenopausal breast cancer. In contrast, increased exposure at the time of a woman's first childbirth was associated with an increased risk of postmenopausal breast cancer. As part of the same study in western New York, Bonner et al. (2005) examined the relationship between increased exposure at birth to total suspended particulates (used as a surrogate measure of air pollution and PAHs) and the increased risk of breast cancer later in life. This study suggests that exposure in early life to high levels of PAHs may increase the risk of postmenopausal, but not premenopausal, breast cancer. However, Bonner et al. (2005) caution that other confounders related to geography cannot be ruled out as possible factors for the increased risk for postmenopausal cancers. The entire scope of research on diesel particulate matter can be seen by searching the National Library of Medicine’s electronic database of citations from biomedical literature (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=search&DB=pubmed) using the keywords diesel particulate matter.

(2) Non-Cancer Effects

For each of the CMP air contaminants, long-term exposure to high levels can cause non-cancer health effects in laboratory animals or humans. The potential for most of these contaminants to cause non-cancer health effects was

evaluated by comparing each contaminant's estimated CMP air concentration to the air concentration generally used to set standards or guidelines (i.e., the reference concentration). The reference concentration is an air concentration that is expected to be without an appreciable risk of non-cancer health effects. This is the same method used to evaluate non-cancer risks in the *Working Draft Report* (see Section *VB-1. Outdoor Air – Ethylene Thiourea*). This analysis could not be completed for two contaminants (diethanolamine and propionaldehyde) because of insufficient data to determine a reference concentration. However, data that are available suggest that these chemicals are not very potent toxicants (HSDB, 2005). For ozone, the federal 8-hour ozone standard was used because public health agencies have not derived a reference concentration for ozone.

For every contaminant except ozone, the estimated CMP air concentration is less than the reference concentration, and the ratio of the CMP concentration/reference concentration was less than 1 *(see Table 59).* The non-cancer health risks associated with a ratio below 1 are rated "minimal" on a qualitative scale that has been used by the NYS DOH *(Appendix V-1).*

For ozone, the ratio of the estimated CMP ozone level to the ozone 8-hour standard is 1.1. The non-cancer health risks associated with this ratio were not rated using the qualitative scale used for the other contaminants (see *Appendix V-1)* because the ratio, as mentioned above, is based on the ozone 8-hour standard rather than the ozone reference concentration. A ratio greater than 1 indicates that the estimated CMP ozone level is higher than the ozone standard. This result is consistent with the observation that the Long Island area sometimes exceeds the 8-hour ozone standard and has been in a "non-attainment" area since an ozone standard was first introduced in the early 1970s. At concentrations above the 8-hour standard, the NYS DOH recommends limiting strenuous outdoor physical activity to reduce the risk of adverse effects (such as nose and throat irritation, shortness of breath, chest pain, coughing and decreases in lung function). People who may be especially sensitive include the very young and those with pre-existing respiratory problems such as asthma.

This finding does not indicate the need for additional studies in the CMP area because the analysis indicates that CMP residents do not have an unusual exposure to ozone. The air quality in the CMP area is likely to be similar to other parts of Long Island. Many physicians and scientists are already studying the potential health effects of ozone. The scope of research can be seen by searching the National Library of Medicine's electronic database of citations from biomedical literature (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=search&DB=pubmed) using the keyword "ozone." In addition, NYS DEC has on-going efforts to reduce ozone air concentrations in the area and to provide advanced notice to the public so that individuals can change their daily activities to reduce the potential for adverse health effects on days when the 8-hour ozone standard is expected to be exceeded.

Conclusions

The integration of the exposure and toxicity data for CMP air contaminants does not support a recommendation for follow-up studies on these contaminants in the CMP area. Although the reasons for this conclusion for contaminants identified by the CEP and NATA datasets are somewhat different than the reasons for ozone, the conclusion in all cases is based on the results of three separate analyses (*see Table 60).*

The only cancer risk level classified as *moderate* was for diesel particulate matter, and it was based on lung cancer data. As discussed previously, risk estimates based on lung cancer are considered poor surrogates for risk estimates for breast cancer. In addition, CMP residents do not appear to have an unusual exposure to diesel particulate matter because the modeled data are similar to the rest of Suffolk County. Finally, there are concerns about the accuracy of the modeled air concentrations for diesel particulate matter in the CMP area.

Acetaldehyde and arsenic are two contaminants that have a *low* cancer risk *(see Table 58).* These are respiratory carcinogens with *inadequate* evidence for carcinogenic activity in breast tissue in humans or animals *(see Table 57).* The three chemicals that are classified as *probable* or *possible* risk factors for human breast cancer are all determined to be of *very low* risk (for any type of carcinogenic activity) at the air concentrations estimated for the CMP area. With the exceptions indicated, the qualitative descriptors of cancer and non-cancer risk for CEP and NATA contaminants are *very low* or *minimal* (the lowest of the qualitative descriptors of cancer and non-cancer risk used by NYS DOH).

For ozone, the exposure analysis shows *high* confidence in the exposure estimates. However, ozone is *not classifiable* as a risk factor for human breast cancer. The estimated CMP air concentration is slightly higher than the 8-hour ozone standard, which indicates the potential for adverse non-cancer health effects. However, monitoring data indicate that the levels of ozone in the CMP area are similar to the levels in other parts of Long Island; thus, residents are not likely to have an unusual exposure to ozone.

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Table 55. Air Contaminants: Estimated Air Concentrations in the CMP Areas and Comparison Ratios for Three Comparison Areas

Chemical	CMP Concentration estimate (mcg/m^3)	Comparison Ratios		
		CMP/ Suffolk	CMP/NYS without NYC	CMP/ NYS
CEP* or NATA**				
Acetaldehyde*	1.54	1.96	2.89	1.18
Acrylamide*	4.14×10^{-8}	0.97	1.37	0.41
Acrylic acid*	1.02×10^{-3}	1.97	10.96	6.78
Aniline*	1.67×10^{-3}	0.61	1.01	1.53
Arsenic*	3.32×10^{-4}	1.07	1.18	0.46
Beryllium*	2.51×10^{-5}	1.27	1.67	0.84
Cadmium**	1.21×10^{-4}	1.19	0.40	0.30
1,2-Dibromoethane**	5.44×10^{-5}	1.04	4.05	5.37
1,1-Dichloroethene*	1.13×10^{-5}	1.33	0.16	0.24
1,3-Dichloropropene*	5.46×10^{-2}	0.93	1.12	0.46
Diesel particulates**	2.37	0.88	1.32	0.44
Diethanolamine*	2.13×10^{-6}	5.24	0.08	0.06
Dimethyl phthalate*	4.02×10^{-4}	0.74	1.80	3.21
Ethylene oxide**	3.99×10^{-4}	1.50	0.52	0.20
Ethylene thiourea*	8.95×10^{-8}	2.55	18.43	33.88
Glycol ethers*	0.612	0.85	1.10	0.45
Hydrochloric acid*	0.939	1.18	1.35	0.65
Hydrofluoric acid*	6.73×10^{-2}	1.51	2.86	2.81
Methylene diphenyl diisocyanate*	2.51×10^{-4}	1.17	2.90	0.61
Methyl ethyl ketone*	1.44	1.52	2.10	0.94
Methyl *tert*-butyl ether*	0.871	3.44	4.84	2.39
Propionaldehyde*	0.534	2.55	4.33	1.91
1,2,4-Trichlorobenzene*	8.83×10^{-5}	0.68	3.14	4.55
1,1,1-Trichloroethane*	2.92	0.90	1.11	0.70
Trichloroethene*	0.416	0.86	1.12	0.80
NYS DEC Monitoring Data***				
Ozone	No monitoring stations are in the CMP area. Closest monitoring stations are in Babylon and Riverside, NY and reported 95 and 84 parts per billion (ppb). The average of these estimates is 90 ppb.		Not applicable	

**CEP: US EPA Cumulative Exposure Project (Rosenbaum et al., 1998).*

***NATA: National-scale Air Toxics Assessment (US EPA, 1996).*

****Average of 4th highest daily maximum 8-hr average for 2001 – 2003 for monitoring stations. Data from NYS DEC (2003).*

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Table 56. Air Contaminants Selected for Further Investigation: Summary of Uses, Sources, Routes of Exposure and Other Environmental Media Where a Contaminant Was Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
Acetaldehyde	*Uses:* Intermediate in the synthesis of other chemicals, e.g., perfumes, polyester resins, dyes. Food preservative and flavoring agent. Solvent in rubber, tanning, paper industries. *Sources:* Industrial releases and ubiquitous product of incomplete combustion. Residential fireplaces and woodstoves are the two highest emission sources, followed by industrial emissions (including coal refining and waste processing). Additional sources include vehicle exhaust, tobacco smoke, and food (including roasting coffee). *Routes of exposure:* Primarily inhalation; also ingestion and dermal contact; also produced when food and alcohol are metabolized.	None Identified
Acrylamide	*Uses:* Intermediate in production of polyacrylamides, synthetic chemicals, dyes, adhesives, permanent press fabrics. Soil conditioning, ore processing, flocculent for sewage/waste treatment. *Sources*: Industrial releases, soil amendments, treated wastewater used as a drinking source. *Routes of exposure:* Inhalation, ingestion of low levels in contaminated drinking water, ingestion of/dermal contact with conditioned soil.	None Identified
Acrylic acid	*Uses:* Manufacture of plastics, floor polish, paint and other coatings, leather finishings. *Sources:* Industrial releases, consumer products (paints, floor polishes, etc.), some species of algae produce acrylic acid naturally. *Routes of exposure*: May be released during production into wastewater and air, resulting in possible exposure by inhaling or drinking contaminated air or water. Use of consumer products (e.g., polishes, paints or coatings, adhesives, plastics, textiles) may result in exposure by several routes.	None Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
Aniline	*Uses:* Chemical intermediate for synthesizing dyes, agricultural products, polymers, and rubber. Used as solvent and gasoline additive (anti-knock agent). *Sources:* Industrial releases, released from burning plastic and tobacco and from degradation of some organic/biological wastes. These emissions do not persist in air, but are converted to other products. Detected in drinking- and surface-waters. Product of incomplete combustion and biological degradation. Natural product found in small amounts in some foods (e.g., corn, grains, rhubarb, apples, beans, and black tea). *Routes of exposure:* Inhalation, ingestion, or dermal contact of contaminated medium.	None Identified
Arsenic	*Major use:* Wood preservative. *Minor Uses:* Alloys in lead storage batteries; less toxic organic forms used as pesticides. *Historical uses*: medicines and pesticides (on cotton, orchards, potatoes, weedkiller, household rat and ant poison). *Sources:* Arsenic is released from burning coal, oil, wood, municipal waste incinerators, pesticide use. *Routes of exposure:* Dermal absorption through skin contact with contaminated soil/water or during pesticide application; ingestion of contaminated food, water, and soil (more common with children); inhalation from combustion of coal, oil, wood, etc. Inhalation of fumes from copper or lead smelting, treating wood or working with treated wood, or inhalation during pesticide application.	None Identified
Beryllium	*Uses*: Alloys for electrical and electronic parts, construction materials, molds for plastics, x-ray machines, mirrors, nuclear reactors. *Sources:* Industrial releases, released from burning coal, fuel oil, and tobacco; also from some hazardous waste sites. *Routes of exposure*: Inhalation of contaminated air. Exposure from consumer products is believed to be minimal.	None Identified
Cadmium	*Uses*: Batteries, pigments, plastics, metal coatings/alloys. *Sources:* Consumer products (batteries, paints, etc.); industrial releases; found in food and tobacco smoke. *Routes of exposure*: Food ingestion and inhalation of tobacco smoke are major source of exposure for most people. Inhalation while soldering or welding, or during smelting/refining of metals.	None Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
1,2-Dibromoethane	*Current uses*: Control bark beetles and termites in logs, moths in beehives; also, chemical intermediate in synthesis of dyes, resins, waxes, and gums. *Historical uses*: gasoline additive; fumigant for citrus, vegetables, grain, soil and turf (nematodes). *Sources*: Pesticide use, industrial releases. *Routes of exposure:* Inhalation (considered minor because only low levels detected in ambient air); ingestion of contaminated food (prior to 1984 ban as a fumigant) and water (potentially now); inhalation and dermal contact during pesticide application.	Contaminated drinking water is a potential concern. In the CMP area, 1,2-dibromoethane was not detected in private water; detected in only one public water well (1996-2001) at level below NYS drinking water standard.
1,1-Dichloroethene (vinylidene chloride)	*Uses*: Manufacture of plastic wrap for food, PVC plastics, flame-retardant coatings for carpets. Sources: Industrial releases. *Routes of exposure*: Inhalation of air near facilities; ingestion of contaminated water or (possibly) food; dermal contact during manufacturing; contamination of food by plastic wraps was determined by FDA not to pose a health risk.	In the CMP area, 1,1-dichloroethene was not detected in public drinking water systems (historical analyses). It was detected in three samples of private drinking water and all detects were below NYS drinking water standards.
1,3-Dichloropropene (DCP), cis- and trans-	*Major use*: Agricultural pesticide. *Minor uses*: Solvent and chemical intermediate. *Sources:* Pesticide use, industrial releases, very small amounts of DCP are formed during water chlorination. *Routes of exposure*: Inhalation of air near farms or hazardous waste sites; ingestion of contaminated food and water; dermal contact during pesticide application.	In the CMP area, in public drinking water samples there has been no detection of *cis*-DCP. To date, there has been no testing for *trans*-DCP in public drinking water. In private drinking water wells, there has been no detection of *cis*- or *trans*-DCP.
Diesel particulate matter (DPM)	*Components*: Particles (elemental carbon + adsorbed organics, metals, sulfate, nitrate); gas phase: aldehydes, benzene, butadiene, polycyclic aromatic hydrocarbons (PAHs). *Sources*: On-road and non-road diesel engines, including: aircraft, boats, farming, construction, recreational vehicles, lawnmowers, etc. *Routes of exposure:* Primarily inhalation, dermal exposure possible but not as significant.	None Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
Diethanolamine	*Uses:* Cutting oils, soaps, shampoos, cleaners, polishers, cosmetics, pharmaceuticals; chemical intermediate in manufacture of rubber; softening agent; emulsifier for agricultural chemicals. *Sources*: Consumer products (soaps, cosmetics, cleaners, etc.), industrial releases, agricultural chemicals. *Routes of exposure*: Primary route is dermal contact with cosmetics and detergents; some inhalation exposure possible.	None Identified
Dimethyl phthalate (DMP)	*Uses:* Manufacture of flexible plastics such as tubings, PVC bags (including those used for hemodialysis and for intravenous solutions) and rubber-coating agents. Component of insect repellants, pesticides, molding powders, lacquers, solid rocket propellant. *Sources*: Industrial releases, medical products, pesticides/repellants, consumer use of lacquers. *Routes of Exposure:* Ingestion of contaminated food or water; intravenous, from hemodialysis tubing and PVC bags; inhalation of insect repellants, pesticides or air containing DMP; dermal contact with insect repellants.	None Identified
Ethylene oxide	*Major use*: Chemical intermediate in synthesis of ethylene glycol, antifreeze, polyester. *Minor uses*: Fumigant for nuts and spices, sterilant for medical supplies. *Sources*: Industrial releases, car exhaust, tobacco smoke, some foods, medical offices. *Routes of exposure:* Inhalation of car exhaust, tobacco smoke, and of air contaminated by industrial emissions; ingestion of food; contact with medical equipment.	None Identified
Ethylene thiourea (ETU)	*Uses:* Chemical intermediate in synthesis of rubber and fungicides (ethylene-bis-dithiocarbamates). *Sources*: Industrial releases, pesticide use. *Routes of exposure:* Inhalation during production of rubber products or inhalation of pesticides or ingestion of foods contaminated with ETU.	None Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
Glycol ethers (GE)	Glycol Ethers: A group of compounds that includes 2-methoxyethanol (EGME), 2-ethoxyethanol (EGEE), 2-butoxyethanol (EGBE). *Uses:* Solvents for resins, lacquers, paints, varnishes, gum, perfume, dyes and inks, cleaning compounds, liquid soaps, cosmetics, hydraulic fluids. *Sources*: Industrial releases, consumer products (paints, cosmetics, detergents, etc.) *Routes of exposure:* Inhalation and dermal exposure in chemical industries producing these products; or from the use of these products occupationally or in the home.	None Identified
Hydrochloric acid	*Uses:* Multi-purpose industrial and laboratory chemical. Specific uses include production of chlorides, refining ores, cleaning metals, removing scale from boilers, manufacture of fertilizers, dyes, and in photographic, textile and rubber industries. *Sources:* Industrial releases, product from combustion of fuels and refuse incineration. *Routes of exposure:* Inhalation of contaminated air; dermal contact during manufacturing.	None Identified
Hydrofluoric acid (HF)	*Uses:* Production of fluorocarbons, which are used in refrigerants, aerosol sprays, and plastics. HF is also used for etching and polishing glass. *Sources:* Industrial releases, present in tobacco smoke, emissions from coal combustion, dust from weathering rocks and soils, volcanoes. *Routes of exposure:* Inhalation of contaminated air; dermal contact during manufacturing.	None Identified
Methyl ethyl ketone (MEK)	*Uses:* General purpose solvent, particularly for gums, resins, cellulose acetate, cellulose nitrate, paints, adhesives. *Sources:* Released into ambient air from manufacturing waste, drying paints and adhesives, auto exhaust, cigarette smoke, hazardous waste sites. Formed by photooxidation of other air pollutants (butane and other hydrocarbons). Emitted by some plants. Detected in some ground waters and foods. *Routes of exposure:* Inhalation of contaminated air; ingestion of water and food.	None Identified
Methylene diphenyl diisocyanate (MDI)	*Uses:* Production of polyurethane foams. *Sources*: Industrial releases. *Routes of exposure:* Inhalation of ambient air; dermal contact during manufacturing.	None Identified

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where the Contaminant Has Been Identified
Methyl tert-butyl ether (MTBE)	*Uses:* Oxygenating agent in gasoline, until its NYS ban in January 2004; used as a laboratory chemical and in medicine to dissolve gallstones. *Sources*: Industrial releases, medicines. *Routes of exposure: Historically*: Inhalation of gasoline vapors during refueling or from automobile exhaust when MTBE was added to gasoline. *Current:* Ingestion of contaminated ground water; inhalation of vapors in an occupational setting; internal from use as medicine.	MTBE has not been detected in public (community or non-community) drinking water systems. In private water systems, MTBE was detected in 4 historical samples (1971-1996) and 18 recent samples (1996-2001); all results were below NYS drinking water standards.
Propionaldehyde	*Uses:* Synthesis of plastics and rubber chemicals; disinfectant and preservative. *Sources:* Industrial releases, consumer use as disinfectant or preservative, ubiquitous combustion product from burning of wood, gasoline, diesel, polyethylene, tobacco, municipal waste. Found in drinking water and coffee. *Routes of exposure:* Inhalation of ambient or indoor air or tobacco smoke; minor exposure from ingestion, dermal contact.	None Identified
1,2,4-Trichlorobenzene (TCB)	*Uses:* Heat-transfer medium (e.g., transformer oil), degreaser, lubricant, solvent in chemical manufacturing. Insecticide against termites (historical use). *Sources*: Industrial releases, consumer products (degreaser, lubricant, etc.) *Routes of exposure:* Ingestion of contaminated drinking water and food; dermal contact from use of degreasers and lubricants.	None Identified
1,1,1-Trichloroethane (methyl chloroform, or TCA)	*Uses:* Solvent in glues, paints, metal degreasers, spot cleaners, aerosol sprays. *Sources*: Industrial releases, use of consumer products (cleaners, glues, paints, degreasers, etc.). *Routes of exposure:* Inhalation or dermal contact during use of consumer products; ingestion of contaminated drinking water.	TCA has been the most commonly occurring volatile organic contaminant found (at levels at or below NYS drinking water standards) in public and private drinking water supply data for the CMP area. (See *Working Draft,* Chapt. 5, pp.153-157.)
Trichloroethene (TCE)	*Uses:* Degreaser; solvent used for dry cleaning, in chemical manufacture and in many household products (e.g., paint removers, spot removers, adhesives, typewriter correction fluid). *Sources*: Industrial releases, product use (degreasers, adhesives, cleaners, etc.) *Routes of exposure:* Inhalation; dermal contact with products containing TCE; ingestion of contaminated drinking water; inhalation from using contaminated water for bathing or cooking.	In the CMP area, TCE was detected in less than 1% of public community drinking water samples (1971-1996); all detetced samples were below the drinking water standard. TCE was detected in 4% of private drinking water wells (1996-2001) and 6% of private drinking water wells (1971-1996) in the CMP area.

Contaminant	Contaminant Uses, Sources and Routes of Exposure for the General Public	Information on Other Environmental Media in the CMP Area Where a Contaminant Has Been Identified
Ozone	*Use:* Ozone is sometimes used to disinfect water or to "purify" air. *Source of ground level ozone:* Formed by sunlight from oxides of nitrogen (NO_x) and volatile organic compound (VOC) emissions from industrial sources and gasoline and diesel engines. *Source of data:* From air monitors in Babylon and Riverhead (see Table 1) *Routes of exposure:* Inhalation of ambient air or indoor air "purified" with ozone.	None Identified

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- *1,2-Dibromoethane, July 1992 (updated May 25, 2001)*
- *1,1-Dichloroethene, May 1994 (updated May 25, 2001)*
- *1,3-Dichloropropene, September 1992 (updated May 25, 2001)*
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- *Aniline*
- *Diethanolamine*
- *Dimethylphthalate*
- *Ethylene dibromide (dibromoethane)*
- *Glycol ethers*
- *Hydrochloric acid*
- *Hydrofluoric acid*
- *Propionaldehyde*
- *Methylene diphenyl diisocyanate*
- *1,2,4-Trichlorobenzene*

Table 57. Breast Cancer Risk Factor Categories for Air Contaminants.

Chemical	Breast Cancer Risk Factor Category	Data - Level of Evidence*		
		Human	Animals	Mode-of-Action
CEP or NATA				
Acetaldehyde	Potential (2C)	Inadequate	Inadequate	Strong
Acrylamide	Possible (2B)	Inadequate	Limited	Limited
Acrylic acid	Potential (2C)	No Data	Inadequate	Limited
Aniline	Potential (2C)	Inadequate	Negative	Limited
Arsenic	Potential (2C)	Inadequate	Inadequate	Strong
Beryllium	Potential (2C)	Inadequate	Inadequate	Limited
Cadmium	Potential (2C)	Inadequate	Inadequate	Strong
1,2-Dibromoethane	Probable (2A)	Inadequate	Sufficient	Strong
1,1-Dichloroethene	Potential (2C)	Inadequate	Inadequate	Limited
1,3-Dichloropropene	Potential (2C)	Inadequate	Negative	Limited
Diesel particulate matter	Potential (2C)	Inadequate	Inadequate	Limited
Diethanolamine	Potential (2C)	Inadequate	Inadequate	Limited
Dimethyl phthalate	Not Classifiable (3)	Inadequate	Inadequate	Inadequate
Ethylene oxide	Probable (2A)	Limited	Limited	Strong
Ethylene thiourea	Potential (2C)	Inadequate	Inadequate	Limited
Glycol ethers				
Ethylene Glycol Butyl Ether	Not Classifiable (3)	Inadequate	Negative	Inadequate
Ethylene Glycol Methyl Ether	Potential (2C)	Inadequate	Inadequate	Limited
Hydrochloric acid	Not Classifiable (3)	Inadequate	Inadequate	Inadequate
Hydrofluoric acid	Not Classifiable (3)	Inadequate	Inadequate	Inadequate
Methylene diphenyl diisocyanate	Potential (2C)	Inadequate	Negative	Limited
Methyl ethyl ketone	Not Classifiable (3)	Inadequate	Inadequate	Negative
Methyl *tert*-butyl ether	Unlikely (4)	Inadequate	Negative	Negative
Propionaldehyde	Potential (2C)	Inadequate	Inadequate	Limited
1,2,4-Trichlorobenzene	Not Classifiable (3)	No Data	Inadequate	Inadequate
1,1,1-Trichloroethane	Not Classifiable (3)	Inadequate	Inadequate	Inadequate
Trichloroethene	Not Classifiable (3)	Inadequate	Negative	Inadequate
NYS DEC Monitoring Data				
Ozone	Not Classifiable (3)	Inadequate	Negative	Inadequate

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Table 58. Air Contaminants and Estimated Excess Lifetime Cancer Risks at Estimated CMP Air Concentrations

Contaminant	Breast Cancer Risk Factor Category	CMP Air Concentration Estimate (mcg/m^3)	Risk Reference Values (one in one million air concentration as mcg/m^3)[a]	Target Organ(s) (species)	Estimated Lifetime Excess Cancer Risk[b]
CEP or NATA					
Acetaldehyde	Potential	1.54	0.45 (US EPA, 2005)	nose (rat)	3.4×10^{-6}
Acrylamide	Possible	4.14×10^{-8}	7.7×10^{-4} (US EPA, 2005)	CNS, breast, thymus, uterus (rat)	5.4×10^{-11}
Acrylic acid	Potential*	1.02×10^{-3}	IARC (1999) determined it was not classifiable as to its carcinogenicity to humans; data to calculate a cancer potency factor data are not available		
Aniline	Potential	1.67×10^{-3}	0.63 (CA EPA, 2002)	spleen (rat)	2.6×10^{-9}
Arsenic	Potential	3.32×10^{-4}	2.3×10^{-4} (US EPA, 2005)	lung (human)	1.4×10^{-6}
Beryllium	Potential	2.51×10^{-5}	4.2×10^{-4} (US EPA, 2005)	lung (human)	6.0×10^{-8}
Cadmium	Potential	1.21×10^{-4}	5.6×10^{-4} (US EPA, 2005)	lung (human)	2.2×10^{-7}
1,2-Dibromoethane	Probable	5.44×10^{-5}	1.7×10^{-3} (US EPA, 2005)	nose, hemangiosarcoma, mesothelioma (rat)	3.2×10^{-8}
1,1-Dichloroethene	Potential*	1.13×10^{-5}	US EPA (2005) concluded that the weight of evidence is not sufficient to justify deriving a cancer potency factor		
1,3-Dichloropropene	Potential	5.46×10^{-2}	0.25 (US EPA, 2005)	respiratory (mouse)	2.2×10^{-7}
Diesel particulate matter	Potential	2.37	0.0033 (CA EPA, 2002)	lung (human)	7.2×10^{-4}
Diethanolamine	Potential*	2.13×10^{-6}	IARC (2000) determined it was not classifiable as to its carcinogenicity to humans; data to calculate a cancer potency factor data are not available		
Dimethyl phthalate	Not Classifiable	4.02×10^{-4}	US EPA (2005) determined that it was not classifiable as to its human carcinogenicity; data to calculate a cancer potency factor data are not available		
Ethylene oxide	Probable	3.99×10^{-4}	0.011 (CA EPA, 2002)	leukemia (rat)	3.6×10^{-8}
Ethylene thiourea	Potential	8.95×10^{-8}	7.7×10^{-2} (CA EPA, 2002)	thyroid (rat)	1.2×10^{-12}
Glycol ethers		0.612			
Ethylene Glycol Butyl Ether	Not Classifiable		US EPA (2005) determined that its human carcinogenic potential cannot be determined at this time;		
Ethylene Glycol Methyl Ether	Potential*		US EPA (2005) did not evaluate its carcinogenic potential; data to calculate cancer potency factor are not available		
Hydrochloric acid	Not Classifiable	0.939	IARC (1992) determined it was not classifiable as to its carcinogenicity to humans; data to calculate a cancer potency factor data are not available		
Hydrofluoric acid	Not Classifiable	6.73×10^{-2}	Published literature on its carcinogenicity in animals or humans were not found; data to calculate a cancer potency factor were not found		
Methylene diphenyl diisocyanate	Potential*	2.51×10^{-4}	US EPA (2005) determined that the human carcinogenic potential cannot be determined; data to calculate a cancer potency factor are not available		

Contaminant	Breast Cancer Risk Factor Category	CMP Air Concentration Estimate (mcg/m^3)	Risk Reference Values (one in one million air concentration as mcg/m^3)[a]	Target Organ(s) (species)	Estimated Lifetime Excess Cancer Risk[b]
Methyl ethyl ketone	Not Classifiable	1.44	US EPA (2005) determined that data inadequate to classify its human carcinogenic potential; data to calculate a cancer potency factor are not available		
Methyl *tert*-butyl ether	Unlikely	0.871	3.8 (CA EPA, 2002)	kidney, testes, leukemia/lymphoma (rat)	2.3×10^{-7}
Propionaldehyde	Potential*	0.534	Published literature on its carcinogenicity in animals or humans were not found; data to calculate a cancer potency factor were not found		
1,2,4-Trichlorobenzene	Not Classifiable	8.83×10^{-5}	US EPA (2005) determined that it was not classifiable as to its human carcinogenicity; data to calculate a cancer potency factor are not available,		
1,1,1-Trichloroethane	Not Classifiable	2.92	US EPA (2005) determined that it was not classifiable as to its human carcinogenicity; data to calculate a cancer potency factor are not available		
Trichloroethene	Not Classifiable	0.416	0.5 (CA EPA, 2002)	liver, lung, lymphoma (mouse)	8.0×10^{-7}
NYS DEC Monitoring Data					
Ozone	Not Classifiable	Monitoring stations in Babylon and Riverside, NY reported 95 and 84 ppb, the average of these estimates is 90 ppb.	In 2-year and lifetime inhalation studies (NTP, 1994), there was no evidence of carcinogenic activity in rats and weak evidence of carcinogenic activity mice (lung); however, these data have not been used to calculate a cancer potency agency by any public health agency		

**These chemicals were identified as potential risk factor for breast cancer based on limited data from mode-of-action studies. The animal and human evidence on breast cancer for these chemicals were either absent, inadequate, or negative.*

[a]This is the air concentration associated with an estimated excess lifetime human cancer risk of one in one million. It is calculated as (1×10^{-6})/(Unit Risk $(mcg/m^3)^{-1}$), where the unit risk is taken from US EPA or CA EPA.

[b]Cancer Risk = ((Air Concentration) / (one in one million risk level)) x (1×10^{-6}). Cancer risks less than one in one million (1×10^{-6}) are rated "very low" using the method NYS DOH has used to evaluate potential cancer risks from environmental contaminants (Appendix V-1).

Table 58 References

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Table 59. Air Contaminants and Estimated Ratios for Non-Cancer Effects at Estimated CMP Air Concentrations

Contaminant	CMP Air Concentration Estimate (mcg/m^3)	Risk Reference Value (RfC, as mcg/m^3)[a]	Target Organ (species)	CMP Air Concentration / RfC[b]
CEP or NATA				
Acetaldehyde	1.54	9 (US EPA, 2005)	olfactory (rat)	0.17
Acrylamide	4.14×10^{-8}	0.7(US EPA, 2005)	nerve (rat)	5.9×10^{-8}
Acrylic acid	1.02×10^{-3}	1 (US EPA, 2005)	olfactory (mouse)	1.0×10^{-3}
Aniline	1.67×10^{-3}	1 (US EPA, 2005)	blood, spleen (rat)	1.7×10^{-3}
Arsenic	3.32×10^{-4}	1.1 (US EPA, 2005)	lung (human)	3.0×10^{-4}
Beryllium	2.51×10^{-5}	0.02 (US EPA, 2005)	lung (human)	1.2×10^{-3}
Cadmium	1.21×10^{-4}	0.02 (NYS DOH, 1990)	kidney (human)	6.0×10^{-3}
1,2-Dibromoethane	5.44×10^{-5}	9 (US EPA, 2005)	nose (mouse)	6.0×10^{-6}
1,1-Dichloroethene	1.13×10^{-5}	200 (US EPA, 2005)	liver (rat)	5.6×10^{-8}
1,3-dichloropropene	5.46×10^{-2}	20 (US EPA, 2005)	respiratory (mouse)	2.7×10^{-3}
Diesel particulate matter	2.37	5 (US EPA, 2005)	lung (rat)	0.47
Diethanolamine	2.13×10^{-6}	Data are limited, and agencies have not derived a reference concentration (ATSDR, 2005; CA EPA, 2005; EPA, 2005)		
Dimethyl phthalate	4.02×10^{-4}	37,000 (US EPA, 2004)	kidney (assumed)	1.1×10^{-8}
Ethylene oxide	3.99×10^{-4}	165 (ATSDR, 1990, 2005)[c]	kidney (mouse)	2.4×10^{-6}
Ethylene thiourea	8.95×10^{-8}	0.28 (US EPA, 2005)	thyroid (rat)	3.2×10^{-7}
Glycol Ethers	0.612			
Ethylene glycol butyl ether		*13,000 (US EPA, 2005)*	*blood (rat)*	*4.7×10^{-5}*
Ethylene glycol methyl ether		*20 (US EPA, 2005)*	*testes (rabbit)*	*0.031*
Hydrochloric acid	0.939	20 (US EPA, 2005)	nasal tract (rat)	0.047
Hydrofluoric acid	6.73×10^{-2}	16.7 (ATSDR, 2003,2005)[d]	respiratory (human)	4.0×10^{-3}
Methylene diphenyl diisocyanate	2.51×10^{-4}	0.6 (US EPA, 2005)	olfactory (rat)	4.2×10^{-4}
Methyl ethyl ketone	1.44	5,000 (US EPA, 2005)	developmental (mice)	2.9×10^{-4}
Methyl t-butyl ether (MTBE)	0.871	3,000 (US EPA, 2005)	liver, kidney (rat)	2.9×10^{-4}
Propionaldehyde	0.534	Data are limited, and agencies have not derived a reference concentration (ATSDR, 2005; CA EPA, 2005; EPA, 2005)		
1,2,4-Trichlorobenzene	8.83×10^{-5}	3.7 (US EPA, 2004)	adrenal, developmental (rat)	2.4×10^{-5}

Contaminant	CMP Air Concentration Estimate (mcg/m^3)	Risk Reference Value (RfC, as mcg/m^3)[a]	Target Organ (species)	CMP Air Concentration / RfC[b]
1,1,1-Trichloroethane	2.92	2,300 (US EPA, 2004)	nervous system (gerbil)	1.3×10^{-3}
Trichloroethene	0.416	40 (US EPA, 2001)	central nervous system (human)	0.010
NYS DEC Monitoring Data				
Ozone	no monitoring stations in CMP area, monitoring stations in Babylon and Riverside, NY reported 95 and 84 ppb, the average of these estimates is 90 ppb	80 ppb[e]	respiratory tract (human)	1.1[f]

[a] *RfC = reference concentration.*

[b] *The health risk at ratios below 1 are rate "minimal" on a qualitative scale that has been used by the NYS DOH (Appendix V-1).*

[c] *0.09 ppm = 165 mcg/m^3 = intermediate minimal risk level (ATSDR, 2005)*

[d] *0.02 ppm = 16.7 mcg/m^3 = acute minimal risk level (ATSDR, 2003)*

[e] *This is federal 8-hour standard for ozone (US EPA, 1997). A RfC has not been determined.*

[f] *The ratio was calculated using the ozone 8-hour standard rather than a RfC (i.e., ratio = monitored ozone concentration/ozone 8-hour standard.*

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Table 60. Integration Summary: None of the Air Contaminants Are Recommended for Further Study

Contaminant	EXPOSURE ANALYSIS			TOXICITY ANALYSIS	RISK ANALYSIS	
	Degree of Confidence That Environmental Data Set Accurately Represents Exposure of CMP Residents		Exposure Estimate - Confidence	Classification As Environ Risk Factor for Human Breast Cancer (*Table 57*)	Likelihood of Health Effects at Estimated CMP Air Concentrations	
	During Years Covered by Dataset	During the Years Covering the Possible Start and Development Of Cancer			Cancer (*Table 58*)	Non-Cancer *(Table 59)*
CEP Or NATA						
Acetaldehyde	Fair	Poor	Low	Potential	Low	Minimal
Acrylamide	Fair	Poor	Low	Possible	Very Low	Minimal
Acrylic Acid	Fair	Poor	Low	Potential	Inadequate data	Minimal
Aniline	Fair	Poor	Low	Potential	Very Low	Minimal
Arsenic	Fair	Poor	Low	Potential	Low	Minimal
Beryllium	Fair	Poor	Low	Potential	Very Low	Minimal
Cadmium	Fair	Poor	Low	Potential	Very Low	Minimal
1,2-Dibromoethane	Fair	Poor	Low	Probable	Very Low	Minimal
1,1-Dichloroethene	Fair	Poor	Low	Potential	Inadequate data	Minimal
1,3-Dichloropropene	Fair	Poor	Low	Potential	Very Low	Minimal
Diesel Particulate Matter	Fair	Poor	Low	Potential	Moderate	Minimal
Diethanolamine	Fair	Poor	Low	Potential	Inadequate data	
Dimethyl Phthalate	Fair	Poor	Low	Not Classifiable	Inadequate data	Minimal
Ethylene Oxide	Fair	Poor	Low	Probable	Very Low	Minimal
Ethylene Thiourea	Fair	Poor	Low	Potential	Very Low	Minimal
Glycol Ethers						
Ethylene Glycol Butyl Ether	Fair	Poor	Low	Potential	Inadequate data	Minimal
Ethylene Glycol Methyl Ether	Fair	Poor	Low	Not Classifiable	Inadequate data	Minimal
Hydrochloric Acid	Fair	Poor	Low	Not Classifiable	Inadequate data	Minimal
Hydrofluoric Acid	Fair	Poor	Low	Not Classifiable	Inadequate data	Minimal
Methylene Diphenyl Diisocyanate	Fair	Poor	Low	Potential	Inadequate data	Minimal
Methyl Ethyl Ketone	Fair	Poor	Low	Not Classifiable	Inadequate data	Minimal

Contaminant	EXPOSURE ANALYSIS			TOXICITY ANALYSIS	RISK ANALYSIS	
	Degree of Confidence That Environmental Data Set Accurately Represents Exposure of CMP Residents		Exposure Estimate - Confidence	Classification As Environ Risk Factor for Human Breast Cancer (*Table 57*)	Likelihood of Health Effects at Estimated CMP Air Concentrations	
	During Years Covered by Dataset	During the Years Covering the Possible Start and Development Of Cancer			Cancer (*Table 58*)	Non-Cancer *(Table 59)*
Methyl Tert-Butyl Ether	Fair	Poor	Low	Unlikely	Very Low	Minimal
Propionaldehyde	Fair	Poor	Low	Potential	Inadequate Data	
1,2,4-Trichlorobenzene	Fair	Poor	Low	Not Classifiable	Inadequate Data	Minimal
1,1,1-Trichloroethane	Fair	Poor	Low	Not Classifiable	Inadequate Data	Minimal
Trichloroethene	Fair	Poor	Low	Not Classifiable	Very Low	Minimal
NYS DEC Monitoring Data						
Ozone	Good	Good	High	Not Classifiable	Inadequate Data	The estimated CMP air concentration exceeds the federal 8-hour ozone standard

2. Pesticide Use

The estimated loading rates (pounds of pesticide applied per square mile) for four pesticides (2,4-D, carbaryl, dicamba, and mecoprop) were higher in the CMP area than in the rest of Suffolk County. These comparisons were originally presented and their limitations discussed in Environmental Exposure Evaluation and are presented again in *Table 61.*

Table 61. Estimated pesticides loading for the CMP area and Suffolk County (excluding the CMP area)

Pesticides	Loading Rate (pounds/square mile		Loading Rate Ratio (CMP/Suffolk County excluding CMP)
	CMP	Suffolk County excluding CMP	
2,4-D*	80	40	2
Dicamba	8	4	2
Mecoprop (MCPP)	30	12	2.5
Carbaryl	140	100	1.4

2,4-D was evaluated in the Working Draft Integration Report (see *Section VB-2*).

Exposure Narrative

Personal exposures to pesticides cannot be reliably estimated from the New York State Pesticide Sales and Use Reporting Database, which was used to obtain the pesticide loading rates in *Table 61.* This is the same problem discussed in the analysis of the potential health risk associated with exposure to 2,4-D. However in that case, Nishioka et al. (2001) estimated the residential exposures of children to 2,4-D after it was applied to their lawns, so that estimate was used to evaluate the potential health risks from long-term exposure to 2,4-D (see *Section VB-2*). The results suggested a “minimal” non-cancer risk. Indoor residential exposures to pesticides has become recognized as a potential source of long-term exposure to pesticides used outdoors and indoors (e.g., Obendorf et al., 2005). This is because pesticides may be more persistence indoors than outdoors because the degradation processes are slower indoors.

State Health researchers were unable to find any scientific data that could be used to make similar estimates for dicamba, mecoprop, and carbaryl. In addition, they choose not to use the 2,4-D dose estimates as surrogates for these pesticides because the relationship between application rates outside and residues levels inside a home depend on many factors that vary greatly among pesticides, including the application rate, environmental half-lives, volatility, and other factors. Thus, similar estimates of indoor residential exposures to these pesticides was not possible.

The degree of confidence that the loading rates for dicamba, mecoprop, and carbaryl accurately represent the potential for CMP residents to have higher exposures than other residents of Suffolk County during years important to evaluating elevated breast cancer rates in 1993 - 1997 is *low*. This ranking is based on two determinations.

(1) The data provide only limited or marginal evidence of an increased presence of these pesticides in the CMP environment. Thus, the use data are considered a *poor* estimator of human residential exposure during the years covered by the data set.

(2) These pesticides have been registered for use in the United States for about 40 years, and their recent use may be reasonably representative of historic use.

Thus, the pesticide use data for 1997-2001 are considered *fair* estimates of dicamba, mecoprop, and carbaryl use in the CMP area during the 5 to 40 years important to the start and development of breast cancers first reported in 1993-1997.

Toxicity Narration (Breast Cancer)

Using the same process described in *Part III. Toxicological Evaluation*, each pesticide was categorized as a risk factor for breast cancer based on an evaluation of the available human data, animal data, and mode-of-action data *(Table 62)*. Dicamba, mecoprop, and carbaryl are all *not classifiable (Category 3).* The criteria for this and other classifications are described in *Tables 12* and *13*.

Risk Narrative

Risk narratives could not be completed because exposure estimates could not be made.

Conclusions

Because exposures could not be estimated due of lack of data, State Health researchers could not evaluate breast cancer risk. However, toxicity data on these pesticides do not identify them as suspected risk factors for breast cancer and this reduced researchers' concerns over the inability to quantify exposure. The limited data on these pesticides suggest the need for further animal or mode-of-action studies. Additional human studies might be warranted if animal or mode-of-action data were positive for breast cancer, and a large, highly exposed population was available for study.

Existing government programs focus on minimizing the potential health risks of applying these and other pesticides. The US EPA and NYS DEC currently register pesticides including dicamba, mecroprop, and carbaryl for use. As part of that process, potential health and environmental risks associated with their use is routinely evaluated using current risk assessment methods—carbaryl was recently evaluated and dicamba, mecroprop will be re-evaluated. These reviews minimize the risks to applicators and others because practices/uses that lead to unreasonable risks will be cancelled or mitigated.

Mechanisms also exist in New York State to track and investigate pesticide poisonings. The NYS DOH maintained Pesticide Poisoning Registry requires physicians and health care facilities to report confirmed or suspected pesticide poisonings within 48 hours. It also requires clinical laboratories to report abnormally depressed cholinesterase levels and abnormally elevated tissue levels of pesticides within 48 hours. The registry monitors both the acute and chronic effects of pesticide exposure, and investigates occurrences of pesticide poisoning. It may perform environmental monitoring to determine the source and circumstances of exposure. More information on the program can be found at http://www.health.state.ny.us/nysdoh/pest/brochure.htm.

Because there is limited data on dicamba, mecoprop, and carbaryl and because programs exist to minimize possible health effects associated with pesticide use, this analysis does not support a recommendation for additional follow-up studies in the CMP area.

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Table 62. Breast Cancer Risk Factor Categories for Pesticides

Pesticide	Breast Cancer Risk Factor Category	Data - Level of Evidence		
		Human	Animals	Mode-of-Action
2,4-D	unlikely (4)	Inadequate	Negative	Negative
Dicamba	not classifiable (3)	Inadequate	Inadequate	Inadequate
Mecoprop (MCPP)	not classifiable (3)	Inadequate	Inadequate	Inadequate
Carbaryl (Sevin)	not classifiable (3)	Inadequate	Negative	Inadequate

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D. Evaluating the Potential Health Risks of Exposures to More than One Contaminant

People are sometimes exposed to mixtures of chemicals (for example, gasoline, which contains benzene and many other chemicals), rather than to a single chemical (for example, benzene). Evaluating the health risks of exposure to chemical mixtures is more difficult than evaluating the health risks from a single chemical. This difficulty stems from several reasons:

- Mixtures may contain many chemicals, each of which has it own toxicological effects.
- The identity and concentration of all the chemicals in the mixture may not be known because of limitations in identification and measurement techniques
- Toxicological information is often limited or not available for many or most of the chemicals of a mixture
- Mixtures of chemicals may cause different effects or have different potencies to cause effects than those of single chemicals.

Scientists believe that there are four possible toxicological results from exposures to a mixture of chemicals.

(1) **No effect.** The combined toxicological effects of exposures to two or more chemicals are no different than the effects of each chemical separately. Each chemical has its own toxicity but does not alter the toxicity of the other chemicals in the mixture. This is most likely true when different chemicals affect different organs by different toxicological modes-of-action. In arithmetic terms, this result can be represented by 2 for one effect and 2 for another effect.

(2) **Additivity.** The combined toxicological effects of exposures to two or more chemicals are equal to the sum of the individual effects of each chemical. (2 and 2 = 4).

(3) **Antagonism.** The combined toxicological effects of exposures to two or more chemicals are less than expected from additivity. (2 and 2 < 4).

(4) **Synergism.** The combined toxicological effects of exposures to two or more chemicals are greater than expected from additivity. (2 and 2 > 4).

Data on the toxicological effects of most mixtures are limited or unavailable. In addition, data to support one of the four results for a particular mixture of chemicals are generally not available. Even when data are available, their usefulness is limited by other considerations. For example, often it is unknown whether the results vary with dose levels. Therefore, experimental results at high doses may not be applicable at lower environmental doses. Also, the mixtures tested in experiments (e.g., gasoline at the gas station) may not be the same as the mixtures in the environment (e.g., gasoline that has leaked into the ground and has been “weathered” by environmental forces). Thus, methods to evaluate the potential risks from exposure to chemical mixtures are based typically more on assumptions and judgements about chemicals rather than on data obtained for actual mixtures. Consequently, these methods are generally used as screening to identify areas or situations where additional mixture-specific research and analysis may be productive and informative.

1. Estimated Total Excess Cancer Risk from CMP Contaminants

The assumption of additivity is the recommended approach for the assessment of cancer risks from chemical mixtures (ATSDR, 2001; NRC, 1989; US EPA, 1986, 1989, 2000, 2003). It is possible that the carcinogenic chemicals might show antagonistic or synergistic interactions, but it is unlikely that these types of interactions are present at low doses (e.g., NRC, 1989), such as those modeled or measured in the CMP area.

The combined calculated excess cancer risk to CMP residents from all the CMP contaminants was estimated in this analysis. This was done by adding up the estimated excess cancer risks for each contaminant, which were presented and discussed in *Sections V-B* and *V-C.* The analysis only included the 16 contaminants (13 air and 3 water) that had sufficient scientific data to calculate cancer risk *(Table 62)*. Risk estimates for the other contaminants could not be calculated because the cancer data on the contaminants were not available or were inadequate to support the calculation of a carcinogenic potency factor.

In this evaluation, the estimated total excess cancer risk is not specific to one type of cancer, but is the excess risk for any type of cancer. Three of the 16 risk estimates (acrylamide, benzene, and 1,1-dichloroethane) were based on breast cancer data. The other 13 were based on other types of cancer.

Based on the summation of the total excess cancer risk estimates, almost 99% of the estimated risk is from diesel particulate matter. The total estimated excess cancer risk from the contaminants in air and water is 7.3×10^{-4}. The estimated excess cancer risk from diesel particulate matter alone is 7.2×10^{-4}. The estimated total excess cancer risk from 16 contaminants is essentially the same because adding many very small risks to one larger risk does not change the total risk by much. The results of this screening indicate that further evaluation of cancer risks from combined exposures is not warranted.

2. Estimated Combined Non-Cancer Risks from CMP Contaminants

An assumption of additivity is also the recommended approach for the assessment of the non-cancer risks from mixtures (ATSDR, 2001; US EPA, 1986, 1989, 2000, 2003). This assumption is most likely to be true only when chemicals in the mixture cause the same effect on the same organ by the same toxicological mode-of-action (ATSDR, 2001; US EPA, 2000). However, mode-of-action data are so rare that it generally assumed that chemicals that affect the same target organ or organ system share the same mode-of-action. This assumption may lead to an overestimation of the non-cancer risks from mixtures if the contaminants do not share a common mode-of-action for target organ or organ systems. In this report, the organ(s) associated with the reference concentrations or dose were identified as the target organ(s) of the chemical.

The combined calculated non-cancer risk to CMP residents was estimated for each organ or organ system that is a target for more than one CMP contaminant by adding together the ratios calculated by dividing the CMP air or water concentration by the reference concentration or reference dose *(Table 63).* These ratios were presented and discussed in *Sections B* and *C* of this chapter. A ratio of 1 is the ratio generally used when environmental agencies propose air guidelines based solely on the non-cancer effects of a chemical *(see Appendix V-1).* The sum of

individual ratios is the combined ratio. A minor modification of ATSDR (2001) mixture guidance policy was used to interpret the likelihood of interactions among CMP contaminants if people were exposed simultaneously to more than one contaminant.

ATSDR (2001) guidance policy suggests that if the individual ratios for a target organ/system are all less than 0.1, any interactions (including additivity) between the chemicals in the mixture are unlikely. This is the case for the potential effects of CMP contaminants on kidney, nervous system, developmental effects, and blood *(Table 63).* It also suggests that if only one contaminant in the mixture has an organ-specific ratio of 0.1 or greater, then the interactions between the contaminant and other contaminants that affect the same organ are also unlikely. This is the case for the liver *(Table 63).* Carbon tetrachloride has a ratio of 0.12, but the three other liver toxicants have substantially lower ratios, keeping the combined ratio at 0.12.

In addition, the guidance policy suggests a potential for interactions among chemicals with the same target organ/system when more than one individual ratio is greater than 0.1 and the combined ratio is greater than 1. This is the case for CMP contaminants that share the respiratory tract as a target organ system *(Table 64).* Acetaldehyde (0.17), diesel particulate matter (0.47), and ozone (1.1) have ratios greater than 0.1 and the combined ratio is 1.7. However, a closer examinations of the toxicological data on these three contaminants suggest that the likelihood of substantial interactions is low.

Although all three contaminant share the same target organ system (the respiratory tract), the target organ for acetaldehyde is the nose (nasal passage), the target organ for diesel particulate matter are the lungs, and the target organs for ozone include the nose and lungs. If we re-calculate hazard indices for nose and the lung to reflect a more careful identification of target organs, the combined hazard indices for ozone and acetaldehyde (1.3) and ozone and diesel particulate matter (1.6).

Consideration of two other factors also reduces concerns about the likelihood of interaction among these three contaminants. (1) The estimated CMP concentrations of acetaldehyde and diesel particulate matter are much lower than air concentrations known to cause effects in animals or estimated to cause effects in humans. (2) It is unlikely that diesel particulate matter and ozone have the same mode of action for lung toxicity.

The estimated CMP air concentration (1.54 mcg/m^3) for acetaldehyde (used to calculate a hazard index of 0.17) is about 84,000-times lower than the lowest concentration known to cause damage to cells lining the nasal passage of rats. It is also about 11,000-times lower that an estimate of the lowest concentration expected to cause damage in humans. Similarly, the estimated CMP concentration (2.37 mcg/m^3) for diesel particulate matter (used to calculate a hazard index of 0.47) is about 400-times lower than the lowest concentration known to cause lung damage in rats or expected to cause lung damage in humans. The concentrations for acetaldehyde and diesel particulate matter are so low that they wouldn't contribute to the risk of respiratory health effects from ozone (hazard index of 1.1) on certain hot weather days when the 8-hour ozone standard is exceeded on Long Island.

In addition, the use of the combined index is likely to be valid when the contaminants share a mode of action for effects on a target organ. Ozone and acetaldehyde are highly reactive contaminants and may share a common

mode of action, but acetaldehyde at low concentrations affects primarily the nose, and not the lungs. Ozone and diesel particulate matter share a common target organ (lungs), but are likely to cause toxic effects via different modes of action.

Collectively, the screening analysis of the toxicity and exposure data indicates that it is unlikely that exposure to mixtures of CMP contaminants would increase significantly the potential for non-cancer kidney, nervous system, blood, liver, or developmental effects (*Table 64).* A more detailed analysis indicates that it is unlikely that mixtures of ozone, diesel particulate matter, acetaldehyde, and other contaminants would increase significantly the risk of respiratory effects.

3. Recommendations

These analyses do not support a recommendation of additional studies of mixtures within the CMP area. They indicate that it unlikely that exposure to mixtures of CMP contaminants would increase significantly the potential for cancer or non-cancer health risks above the potentials associated with individual contaminants. In addition, the data on the quality of the CMP air show it is similar to the air of other parts of Long Island. The potential health effects of mixture of air contaminants are being studied. Papers on the potential effects of air pollution on human health can be found by searching the National Library of Medicine's electronic database of citations from biomedical literature (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=search&DB=pubmed) using the words "air pollution."

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Table 63. Estimated Total Excess Cancer Risks from CMP Contaminants at Estimated Air and Water Concentrations

Contaminant	Tumor Site(s)	Estimated Lifetime Excess Cancer Risk
Air*		
Acetaldehyde	nose (rat)	3.4×10^{-6}
Acrylamide	nervous system or breast or thymus or uterus (rat)	5.4×10^{-11}
Aniline	spleen (rat)	2.6×10^{-9}
Arsenic	lung (human)	1.4×10^{-6}
Beryllium	lung (human)	6.0×10^{-8}
Cadmium	lung (human)	2.2×10^{-7}
1,2-Dibromoethane	nose, and hemangiosarcoma and mesothelioma (rat)	3.2×10^{-8}
1,3-Dichloropropene	respiratory (mouse)	2.2×10^{-7}
Diesel particulate matter	lung (human)	7.2×10^{-4}
Ethylene oxide	leukemia (rat)	3.6×10^{-8}
Ethylene thiourea	thyroid (rat)	1.2×10^{-12}
Methyl *tert*-butyl ether	average of kidney, testes, and leukemia/lymphoma (rat)	2.3×10^{-7}
Trichloroethene	average of liver, lung, and lymphoma (mouse)	8.0×10^{-7}
Water		
Benzene**	breast	2.2×10^{-7}
Carbon tetrachloride***	liver	4.0×10^{-6}
1,1-Dichloroethane***	breast	3.0×10^{-7}
Total (air and water)		7.3×10^{-4}

**Duration of exposure = 70 years, assumes a person lives in the CMP area all their life.*

***Duration of exposure =period of known contamination*

****Duration of exposure = 30 years, US EPA (1999) Exposure Factor Handbook recommended 95th percentile for residence time (i.e., the length of time that people live in one residence (US EPA, 1999)).*

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Table 64. Individual and Total Ratios for CMP Contaminants That Had the Same Target Organ or Organ Systems for Non-Cancer Effects

Contaminant	Source	CMP Air Concentration Estimate Reference Concentration*
Kidney		
Cadmium	outdoor air	0.0060
Dimethyl phthalate	outdoor air	0.000000011
Ethylene oxide	outdoor air	0.0000024
1,1-Dichloroethane	drinking water	0.0012
Methyl *tert*-butyl ether	outdoor air	0.00029
Total		**0.0075**
Nervous System		
Acrylamide	outdoor air	0.000000059
1,1,1-Trichloroethane	outdoor air	0.0013
Trichloroethene	outdoor air	0.010
Total		**0.011**
Developmental Effects		
Methyl ethyl ketone	outdoor air	0.00029
1,2,4-Trichlorobenzene	outdoor air	0.000024
Total		**0.00031**
Blood		
Aniline	outdoor air	0.0017
Glycol Ether:Ethylene glycol butyl ether	outdoor air	0.000047
Total		**0.0017**
Liver		
Carbon tetrachloride	drinking water	0.12
Methyl *tert*-butyl ether	outdoor air	0.00029
1,1-Dichloroethene	outdoor air	0.000000056
1,1,1-Trichloroethane	drinking water	0.00085
Total		**0.12**
Respiratory Tract (nose, nasal passage, lungs)		
Acetaldehyde	outdoor air	0.17
Acrylic acid	outdoor air	0.0010
Arsenic	outdoor air	0.00030
Beryllium	outdoor air	0.0012
1,2-Dibromoethane	outdoor air	0.0000060
1,3-Dichloropropene	outdoor air	0.0027
Diesel particulate matter	outdoor air	0.47
Hydrochloric acid	outdoor air	0.047
Hydrofluoric acid	outdoor air	0.0040
Methylene diphenyl diisocyanate	outdoor air	0.00042
Ozone	outdoor air	1.1
Total		**1.8**

**Ratios less than 1 indicate that the likelihood of non-cancer health risks at the modeled air concentrations are estimated to be* very low*. This category is the lowest of the qualitative descriptors of non-cancer risk used by the NYS DOH (see Appendix V-1).*

E. Uncertainties in the Estimates of the Potential for Cancer and Non-Cancer Health Risks

1. Uncertainties in Toxicity Data Used to Make Health Risk Estimates

The calculated estimates of the excess lifetime human cancer risks or the percentage of the reference dose or reference concentration associated with exposure to the various contaminants in air and water are based on a number of health-protective assumptions. The estimates are frequently based on extrapolating the observations of health effects in animals exposed to high doses for their lifetimes to humans exposed to much lower doses. Health-protective, but scientifically reasonable, choices are made to bridge data gaps in the extrapolations. Health-protective choices are those that more often than not lead to an overestimation of risk for most people. Below are some examples of these choices.

- The assumption that effects that occur at high doses may also occur at lower doses, but generally less frequently or less severely.
- The assumption those chemicals that cause cancer or other effects in animals also cause the same effects in humans.
- The choice to use the most sensitive effects in the most sensitive species in the risk analysis.

This last point is particularly important for the breast cancer risk analysis for those carcinogenic chemicals where dose-response data on the induction of breast cancer in animals or humans are absent (for example, ETU, carbon tetrachloride, and diesel particulate matter). For these chemicals, the cancer risk analysis was based on the most sensitive cancer induced in animals/humans exposed by the chemical.

As a result of all these factors, the health risk estimates are typically considered "worst case" estimates; that is, the true risks at the estimated exposure levels are likely to be lower than the estimates and unlikely to be higher than the estimates.

However, certain other factors could lead to an underestimate of risk under certain circumstances.

- Humans may be more sensitive to certain chemicals than the most sensitive animal species.
- The completeness and quality of the toxicity databases for chemicals vary greatly. In other words, we know much more about some chemicals than other chemicals. Although this uncertainty is considered when evaluating human health risks, incomplete or poor quality data may result in an underestimation of risk.

2. Uncertainties in Exposure Data Used to Make Health Risk Estimates

The data selected for the environmental exposure evaluations were not specifically developed for use in health risk estimates. In every case, it is uncertain whether the data accurately estimate contaminant levels in the CMP area. It also is uncertain if human contact occurred, let alone if it occurred during a time period relevant to the induction and development of breast cancer or other chronic health effects.

The data used to evaluate air contaminants were obtained from modeling estimates derived by US EPA. Sampling was not done to determine if the modeled estimates were close to the actual levels in the ambient air. In fact, some of the modeled estimates are air levels that could not be measured accurately using standard measurement methods. Additionally, the modeled estimates represent only outdoor concentrations over recent one-year periods and may not reflect exposures during years critical to the development of breast cancer or other chronic health effects.

The data set used to characterize pesticides was the most limited. In fact, it was not possible to use the loading data (pounds/gallons of pesticides applied) from the New York State Pesticides Sales and Use Database to estimate human exposures.

The data used to evaluate contaminants in public water supplies were based on limited sampling frequencies at a few points in the distribution system. The samples were collected for other purposes, such as evaluating regulatory compliance and responses to address specific contamination issues, not to estimate human exposures. As a result, they are not accurate representations of average exposures in the CMP area over long periods. It is unlikely, for example, that the average level of 1,1,1-TCA in a public water supply used in the calculation of health risk is the average long-term exposure level for all people served by the water supply, let alone people served by other water supplies or on private wells. On the other hand, the measured concentrations were in water samples collected during years relevant to the development of breast cancer or other chronic health effects.

Even if we assume a degree of confidence in the human relevance of the toxicity and exposure data used to calculate health risk estimates, other factors important to their interpretation should be considered. Risk estimates were based on standard exposure parameters commonly used to develop guidelines and standards for contaminants in air and water. These parameters include a breathing rate of 20 cubic meters per person per day, a water consumption rate of 2 liters per person per day, a body weight of 70 kg, and continuous (air) or daily (water) long-term exposures. These parameters won't hold for every individual, family members or community sharing some common exposure. The exposures scenario for air contaminants (exposure to the same level of contamination every day for 70 years for cancer or 30 years for non-cancer effects) is unlikely for most people, who, if exposed, are more likely to be exposed for part of the day and for part of their lifetime.

For example, the epidemiological evaluation considered length of residence in the CMP area and was able to estimate the year of first residence in the CMP area for 72% of the women in the CMP area diagnosed with breast cancer between 1993 and 1997. Of these, 78% had lived at their address at the time of breast cancer diagnosis for 5 years or more and 62% had lived there for 10 years or more. This shows that the assumption that all the CMP residents lived in the same residence for their entire life is false.

Similarly, two of the exposure scenarios for water contaminants (people within the CMP area served by a public water supply were exposed to the same level of contamination every day for 30 years or 70 years) are also unlikely given our knowledge about the groundwater contamination on Long Island.

When faced with uncertainties, risk assessors often make health-protective choices. These choices were made in both the toxicity and exposure assessments of this report. Using these health-protective assumptions, the evaluation suggests that none of the contaminants in air or water are likely to be related to the elevated breast cancer incidence among women in the CMP area. It also suggests that except for ozone none of the contaminants are likely to be related to non-cancer health effects in the CMP area.

Appendix V-1. NYS DOH procedure for evaluating potential health risks for contaminants of concern

To evaluate the potential health risks from contaminants of concern associated with environmental exposures throughout New York State, but particularly with exposures associated with hazardous waste sites, the New York State Department of Health assessed the risks for cancer and noncancer health effects.

Increased cancer risks are estimated by using site-specific information on exposure levels for the contaminant of concern and interpreting them using cancer potency estimates derived for that contaminant by the US EPA or, in some cases, by the NYS DOH or other authoritative bodies (e.g. the California Environmental Protection Agency. The following qualitative ranking of cancer risk estimates, developed by the NYS DOH, is then used to rank the risk from very low to very high. For example, if the qualitative descriptor was "low", then the excess lifetime cancer risk from that exposure is in the range of greater than one per million to less than one per ten thousand. Other qualitative descriptors are listed below:

Excess Lifetime Cancer Risk

Risk Ratio	Qualitative Descriptor
equal to or less than one per million	very low
greater than one per million to less than one per ten thousand	low
one per ten thousand to less than one per thousand	moderate
one per thousand to less than one per ten	high
equal to or greater than one per ten	very high

An estimated increased excess lifetime cancer risk is not a specific estimate of expected cancers. Rather, it is a plausible upper bound estimate of the probability that a person may develop cancer sometime in his or her lifetime following exposure to that contaminant.

There is insufficient knowledge of cancer mechanisms to decide if there exists a level of exposure to a cancer-causing agent below which there is no risk of developing cancer, namely, a threshold level. Therefore, every exposure, no matter how low, to a cancer-causing compound is assumed to be associated with some increased risk. As the dose of a carcinogen decreases, the chance of developing cancer decreases, but each exposure is accompanied by some increased risk.

There is general consensus among the scientific and regulatory communities on what level of estimated excess cancer risk is acceptable. An increased lifetime cancer risk of one in one million or less is generally not considered a significant public health concern.

For noncarcinogenic (i.e., non-cancer) health risks, the contaminant intake was estimated using standard exposure assumptions consistent with recommendations of the US EPA (1999). This dose was then compared to a risk reference dose (estimated daily intake of a chemical that is likely to be without an appreciable risk of health effects) developed by the US EPA, ATSDR and/or NYS DOH. The resulting ratio was then compared to the following qualitative scale of health risk:

Qualitative Descriptions for
Noncarcinogenic Health Risks

Ratio of Estimated Contaminant Dose to Risk Reference Dose*	Qualitative Descriptor
equal to or less than one	minimal
greater than one to five times	low
greater than five to ten times	moderate
greater than ten times the	high

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*Also ratio of estimate air level to reference concentration

Noncarcinogenic effects unlike carcinogenic effects are believed to have a threshold, that is, a dose below which adverse effects will not occur. As a result, the current practice is to identify, usually from animal toxicology experiments, a no-observed-effect-level (NOEL). This is the experimental exposure level in animals at which no adverse toxic effect is observed. The NOEL is then divided by an uncertainty factor to yield the risk reference dose. The uncertainty factor is a number that reflects the degree of uncertainty that exists when experimental animal data are extrapolated to the general human population. The magnitude of the uncertainty factor takes into consideration various factors such as sensitive subpopulations (for example, children or the elderly), extrapolation from animals to humans, and the incompleteness of available data. Thus, the risk reference dose is not expected to cause health effects because it is selected to be much lower than dosages that do not cause adverse health effects in laboratory animals.

The measure used to describe the potential for noncancer health effects to occur in an individual is expressed as a ratio of estimated contaminant intake to the risk reference dose. A ratio equal to or less than one is generally not considered a significant public health concern. If exposure to the contaminant exceeds the risk reference dose, there may be concern for potential noncancer health effects because the margin of protection is less than that afforded by the reference dose. As a rule, the greater the ratio of the estimated contaminant intake to the risk reference dose, the greater the level of concern. This level of concern depends upon an evaluation of a number of factors such as the actual potential for exposure, background exposure, and the strength of the toxicologic data.

VI. Summary and Conclusions

A. Epidemiologic evaluation

The epidemiologic evaluation confirmed that the excess in breast cancer incidence in the CMP area is not likely due to features of disease detection or reporting in the area, or characteristics of the analysis such as population estimation (including underestimation of the population at risk due to seasonal residents). The breast cancer excess has persisted in the years following the original analysis. Examination of the characteristics of cases has identified no population subgroups disproportionately affected, and there is no evidence of any unusual breast cancer cell types.

Further evaluation of the sociodemographic characteristics shows that the CMP area has several characteristics associated with a higher risk of breast cancer. These include a higher percentage of people identified as white and higher income and education levels. There also is a higher proportion of people employed in education and health care, which have been associated with higher breast cancer incidence in studies that did not control for reproductive factors.

A statistical model was constructed to see how much of the excess in breast cancer incidence could be related to variations in racial composition, income, and educational levels. These factors are not believed to affect breast cancer risk directly, but are correlated with reproductive and lifestyle risk factors for breast cancer such as age at first childbirth and alcohol consumption that are not as easily measured. In the statistical model, the magnitude of the breast cancer excess in the CMP area is reduced and is no longer statistically significantly different from the rest of New York State.

B. Toxicologic Evaluation

The system developed by state health researchers to classify substances as risk factors for breast cancer was successfully implemented for the first time during the CMP Investigation. This system was used to generate a list of substances for research and investigation purposes in New York State and additional substances may be evaluated if evidence exists of unusual exposures in areas with elevated breast cancer.

C. Environmental Exposure Evaluation

State health researchers examined a large of amount of existing information about environmental contaminants and other potential exposures in the CMP area. They evaluated air quality, pesticide use, in-home radon, hazardous waste sites, industrial sites, public and private drinking water and electromagnetic fields in addition to data from a number of state environmental quality databases, such as spills, waste water discharge permits, fishing advisories,

etc. The results showed that the levels of contaminants and other possible environmental exposures in the CMP area were similar to or lower than the rest of New York State for the vast majority of those evaluated.

D. Integration (Health Risk Evaluation)

The potential human health risks from exposure to 31 individual contaminants were evaluated based on evidence of possible exposure to elevated levels in the CMP area. The majority of these were air contaminants that were found at slightly higher levels than other areas of the state. The evaluation suggests that none of the contaminants are likely to be related to the elevated breast cancer incidence among women in the CMP areas. It also suggests that except for ozone, none of the contaminants are likely to be related to non-cancer health effects in the CMP area. Ozone levels in the CMP area as well as the rest of Long Island sometimes exceed the 8-hour ozone standard. When the standard is expected to be exceeded, the NYS DOH recommends limiting strenuous outdoor physical activity to reduce the risk of adverse effects (such as nose and throat irritation, shortness of breath, chest pain, coughing and decreases in lung function). People who may be especially sensitive include the very young and those with pre-existing respiratory problems such as asthma. State health researchers also evaluated the potential risks from exposure to mixtures of CMP contaminants and found it is unlikely that they would increase significantly the potential for non-cancer or cancer effects over those associated with individual contaminants if people were actually exposed to all the contaminants at once and continuously.

Because no unusual factors related to breast cancer incidence or other health effects were found in the CMP area, NYS DOH recommends surveillance for this area, consistent with other statetwide activities (see below for details).

1. NYS DOH will provide ZIP-Code level cancer data for breast, colorectal, lung and prostate cancer periodically for New York State.
2. NYS DOH will identify and assess potential exposures throughout the state through routine environmental health activities and take action to reduce those exposures when necessary.
3. NYS DOH will continue to provide public health education about health outcomes and environmental exposures in New York communities. The agency will respond to individual and public health inquiries recognizing the scientific limitations in answering these questions.
4. As resources allow, NYS DOH will design and carry out studies of highly exposed populations that have been identified by biological or environmental monitoring.
5. NYS DOH will explore the feasibility and usefulness of environmental health surveillance and tracking for different health outcomes and exposures throughout the state.
6. NYS DOH will re-evaluate the Unusual Disease Pattern Protocol based on its first trial in the CMP area to determine its usefulness in conducting follow-up investigations for cancer and other health outcomes in New York State. This evaluation will consider the use of other methods including basic research into the biology of cancer and the mechanisms of carcinogens, and studies of highly exposed populations. It will also consider the likelihood that these methods will further knowledge about the role of the environment in disease occurrence.

Appendix VI-1. Alphabetical Index of Public Environmental Health Concerns

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