Health Consultation

Evaluation of Cancer Incidence in Census Tracts of Attleboro and Norton, Bristol County, Massachusetts: 1982-2002

Shpack Landfill
MAD 980503973

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I. SUMMARY

At the request of residents from the city of Attleboro and the town of Norton, the Massachusetts Department of Public Health (MDPH), Center for Environmental Health's (CEH) Community Assessment Program (CAP) conducted an evaluation of cancer incidence within these two communities. Residents' concerns were focused mainly on suspected increases of cancer in neighborhoods near the Shpack Landfill. The Shpack Landfill, located on the border between Norton and Attleboro, comprises approximately 9 acres and was designated a National Priorities List (NPL) Superfund site in 1986. The Shpack Landfill operated from approximately 1946 until 1968, receiving domestic, industrial, and low-level radioactive waste.

The CAP evaluated the incidence of 13 different types of cancer within Attleboro and Norton and their respective census tracts for the 21-year period of 1982–2002. To evaluate patterns or trends over time, cancer incidence rates were calculated for four time periods: 1982–1987, 1988–1993, 1994–1999, and 2000–2002. The 13 cancer types selected for this evaluation were based upon cancer types that were elevated in an earlier MDPH report entitled *Phase I: Evaluation of Cancer Incidence in Attleboro and Norton, MA, 1994–1998* and those cancer types associated with environmental contaminants detected at the Shpack Landfill. In addition, based upon residents' concerns, a review of all cancer diagnoses within about a 1-mile radius of the Shpack Landfill was conducted.

Of the 13 cancer types evaluated in the city of Attleboro and the town of Norton during the four time periods, the majority occurred approximately at or near expected rates, based on the statewide rates of cancer and the populations of Attleboro and Norton. The exceptions included statistically significant elevations in the incidence of lung and bronchus cancer among females in Attleboro during 1988–1993 and among males in Attleboro during 1994–1999; thyroid cancer among males in Attleboro during 1988–1993; liver cancer among males in Attleboro during 2000–2002; and, bladder cancer among females in Attleboro during 2000–2002. In addition, some census tracts demonstrated statistically significant elevations in the incidence of breast cancer, Hodgkin's disease, brain and central nervous system cancer, and lung and bronchus cancer. Although particular cancer types may have been elevated in one of the four time periods, these elevations were not persistent over time.

In addition to evaluating time trends, the geographic distribution of residence at diagnosis for those individuals diagnosed with cancer in Attleboro and Norton was evaluated using mapping software, to determine if any atypical spatial patterns existed. With two exceptions, review of the geographic distribution of cancer for the years 1982–2002 did not reveal any unusual spatial patterns or concentrations of cases at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents of these communities. When the two exceptions were examined more closely, the geographic distributions appeared to follow closely the population density of the areas; in addition, the areas were approximately 1.5 and 3.0 miles, respectively, from the Shpack Landfill which means that the Landfill was not likely to have played a role in these cancers. Analysis of risk factor information (for example, age, gender, smoking history, and occupation) for individuals diagnosed with cancer suggested that the trends observed in Attleboro and Norton are similar to those seen in the general population. The analysis also suggested that smoking likely played some role in the incidence of some cancer types in these two communities.

An additional review of the Massachusetts Cancer Registry data for residents of Attleboro and Norton living within about a 1-mile radius of the Shpack Landfill did not reveal any unusual patterns with respect to any one cancer type or geographic or temporal patterns. However, when the environmental data reviews are complete, these data can be further evaluated.

Based on criteria established by ATSDR, the Shpack Landfill would be classified as posing an Indeterminate Public Health Hazard pending further analysis of relevant environmental data. Opportunities for exposure to the Shpack Landfill will be characterized in the Public Health Assessment (PHA) as a separate report by the MDPH CEH's Environmental Toxicology Program. The PHA will include the results of this cancer incidence evaluation in the context of environmental exposure pathways identified in the PHA.

II. BACKGROUND AND STATEMENT OF ISSUES

In March 2002, residents of the town of Norton and city of Attleboro contacted the Massachusetts Department of Public Health (MDPH) Center for Environmental Health (CEH) with concerns about suspected increases of cancer in neighborhoods near the Shpack Landfill.

The Shpack Landfill is located on the border between Norton and Attleboro and covers a 9- acre area (Figure 1). It operated from approximately 1946 until 1968, receiving domestic, industrial, and low-level radioactive waste. The site was first designated for remedial action under the United States Department of Energy's Formerly Utilized Sites Remedial Action Program (FUSRAP) in 1981. In 1986, the United States Environmental Protection Agency (USEPA) added the site to the National Priorities List (NPL) under the federal Superfund Program. Since the late 1980s and early 1990s, extensive investigations of environmental media (i.e., soil, surface water, and groundwater) have been performed at the Shpack Landfill. Numerous reports have been written that summarize the type and extent of contamination associated with the site. The Shpack Landfill is adjacent to the Attleboro Landfill Inc., a 55-acre site of which approximately 3 acres are a part of the Shpack Landfill Superfund Site. The Shpack Landfill Superfund Site, therefore, consists of the Shpack Landfill (the 6 acres in Norton) and the 3 acres of the Attleboro Landfill (in Attleboro) and will be referred to hereinafter as the *Shpack Landfill*.

In July 1993, the Bureau of Environmental Health Assessment (BEHA) within the MDPH issued a report on the Shpack Landfill entitled *Site Review and Update* (MDPH 1993). In this document, BEHA reported the following possible human exposure pathways (identified initially in its 1989 Preliminary Health Assessment):

- Dermal absorption or ingestion of contaminants in soil, sediments, groundwater, and surface water
- Exposure to gamma radioactivity in the ambient air at the Shpack Landfill
- Dermal exposure to beta/gamma emissions near ground surface level at the Shpack Landfill

In June 2002, the Community Assessment Program (CAP), a division within the CEH, released a report entitled *Phase I: Evaluation of Cancer Incidence in Attleboro and Norton, MA, 1994–1998* (MDPH 2002; Appendix A). In this report, the CAP reviewed available cancer incidence data from the Massachusetts Cancer Registry (MCR) *City and Town Supplement* for 23 different cancer types for Attleboro and Norton (MCR 2001). For both Norton and Attleboro, the majority of cancer types occurred approximately at or below expected rates for the 5-year period

1994–1998. However, in Attleboro, city-wide incidence rates for six cancer types were elevated among males and females combined compared to statewide rates for these cancers; the cancer types included colorectal cancer, Hodgkin's disease, laryngeal cancer, melanoma, multiple myeloma, and pancreatic cancer. The differences between the numbers of observed and expected cases were not statistically significant. In Norton, town-wide elevations were observed in the incidence of lung and bronchus cancer and pancreatic cancer. However, neither of these elevations was statistically significant.

Upon examining gender-specific incidence rates in Attleboro, two cancer types were statistically significantly elevated during 1994–1998 in the 2002 Phase I CAP report (Appendix A). Males in Attleboro experienced a statistically significant elevation of Hodgkin's disease, while the incidence of liver cancer was statistically significantly elevated among females during the 5-year period. In Norton, females experienced slightly elevated rates of lung and bronchus cancer and pancreatic cancer during 1994–1998, but the elevations were not statistically significant.

In an earlier report issued by the MDPH in July 2001 entitled *Evaluation of Female Lung Cancer Incidence and Radon Exposure in Attleboro, MA 1982-1994* (MDPH 2001), the MDPH reported that female lung cancer incidence occurred statistically significantly less often than expected during 1982-1986 and statistically significantly more often than expected during 1987-1994. In addition to an evaluation of cancer incidence data, this report also included a radon survey in which the radon concentrations measured in the homes (or former homes) of female lung cancer cases was compared to the concentrations measured in a group of randomly selected homes in the city. Although the median radon concentration in both the case and control homes was below the USEPA's recommended remediation level of 4 picocuries per liter, the median radon concentration in the case homes (2.4 picocuries per liter) was higher than the median concentration measured in the randomly selected control homes (1.9 picocuries per liter).

While the 2002 CAP Phase I investigation evaluated cancer incidence for the communities of Attleboro and Norton as a whole, this health consultation examines the pattern of cancer in smaller geographic areas of Norton and Attleboro (that is, in census tracts). This investigation focuses in on particular census tracts in close proximity to the Shpack Landfill and assesses

whether any unusual patterns in cancer incidence might suggest that environmental factors played a role in cancer incidence.

In September 2004, the USEPA issued a Record of Decision (ROD) that presents the selected remedial actions to be undertaken at the site (USEPA 2004). In a separate report, the Environmental Toxicology Program within the CEH will evaluate environmental data for the Shpack Landfill in a PHA; this assessment will evaluate the potential for exposure and any public health hazards posed by the site. In addition, the PHA will include the results of this cancer incidence analysis in the context of environmental exposure pathways identified in the PHA.

III. OBJECTIVES

This report evaluates the incidence of 13 different cancer types for census tracts within the city of Attleboro and the town of Norton over the 21-year period of 1982–2002. The 21-year period is the period for which the most recent and complete cancer incidence data were available from the MCR at the writing of this report. The 13 cancer types selected for this evaluation are based upon cancer types that were elevated in the CAP Phase I investigation and/or cancer types associated with environmental contaminants detected at the Shpack Landfill. In addition, based upon residents' concerns, a review of all cancer diagnoses within about a 1-mile radius of the Shpack Landfill was conducted.

A census tract is a geographic subdivision of a city or town designated by the United States Census Bureau. Because age group and gender specific population information is necessary to calculate incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated. The city of Attleboro is divided into eight census tracts (CTs) (Figure 1). The town of Norton is divided into two CTs. According to the 2000 U.S. Census, 42,068 individuals live in the city of Attleboro, an area of 28.28 square miles (USDOC 2000). Census data from 2000 indicates a population of 18,036 in Norton, an area of 29.82 square miles (USDOC 2000). The Shpack Landfill is located in Attleboro CT 6317 and Norton CT 6112. Attleboro CT 6317 covers an area of approximately 7.5 square miles and has a total population

of 6,261 (USDOC 2000). Norton CT 6112 comprises an area of approximately 14 square miles and has a total population of 8,846.

Descriptive epidemiological analyses such as this can be useful in identifying cancer patterns in a geographic context, assessing if a common cause or etiology is possible, and serving to identify areas where further public health investigations or actions may be warranted. This descriptive analysis of cancer incidence data alone cannot be used to establish a causal link between a particular risk factor (either environmental or nonenvironmental) and the development of cancer. In addition, this analysis cannot determine the cause of any one individual's cancer diagnosis. The purpose of this evaluation is to report on the patterns of cancer in the census tracts of Norton and Attleboro and to determine whether such patterns are unusual. The specific objectives of this investigation follow:

- To evaluate the incidence of 13 cancer types [Hodgkin's disease, leukemia, non-Hodgkin's lymphoma (NHL) and multiple myeloma, as well as cancers of the bladder, bone, brain and central nervous system (CNS), breast, kidney, liver, lung, pancreas and thyroid] in the census tracts of Attleboro and Norton and specifically in neighborhoods near the Shpack Landfill located in Attleboro CT 6317 and Norton CT 6112 to determine if cancer is occurring more or less often than expected;

- To examine qualitatively the occurrence of cancer in neighborhoods of Norton and Attleboro within an approximate one-mile radius of the Shpack Landfill;

- To review available information from the MCR on risk factors for individuals diagnosed with cancer in Norton and Attleboro;

- To discuss the results in the context of the available medical literature on the 13 types of cancer evaluated; and

- To determine whether the spatial patterns of cancer diagnoses in these two communities are unusual.

IV. Methods for Analyzing Cancer Incidence

A. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–2002 were obtained for the communities of Attleboro and Norton from the MCR, a division of the MDPH Center for Health Information, Statistics, Research and Evaluation (CHISRE). The MCR is a population-based surveillance system that began collecting information in 1982 on Massachusetts residents diagnosed with cancer in the state. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111s.111B). The 21-year period, 1982–2002, constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this report.

To further address community concerns, a qualitative evaluation of all cancer types diagnosed from 1982 to the present among residents of Norton and Attleboro living within about a 1-mile radius of the Shpack Landfill was also conducted using MCR data[1]. The MCR utilizes an ongoing surveillance system that collects reports on a daily basis. Therefore, it is possible to review case reports for more recent years to qualitatively evaluate cancer patterns in a given area. However, because the data for recent years (i.e., 2003 to the present) are not complete, they cannot be used to calculate more recent incidence rates. In addition, the CEH evaluated reports provided by current and former residents of the area regarding individuals in the two communities diagnosed with cancer.

Thirteen cancer types were evaluated in this investigation, including cancers of the bladder, brain and central nervous system (CNS), breast, bone, kidney, liver, lung and bronchus, pancreas, and thyroid as well as Hodgkin's disease, leukemia, multiple myeloma, and non-Hodgkin's lymphoma (NHL). [Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. See Appendix B for the incidence coding definitions used in this report.] These cancer types were selected for evaluation based on

[1] The cancer incidence data in this report are drawn from data entered on MCR computer files before February 8, 2006. The numbers presented in this report may differ slightly from those published in previous or future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

environmental contaminants detected at the Shpack Landfill and/or elevations that were observed at the city/town level in a preliminary analysis of cancer rates in Attleboro and Norton (Appendix A). All diagnoses reported to the MCR as primary cancers among residents of Attleboro or Norton for the 13 cancer types were included in the analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis.

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Epidemiologic studies have revealed that different types of cancer are individual diseases with separate causes, risk factors, characteristics and patterns of survival (Berg 1996). Cancers are classified by the location in the body where the disease originated (the primary site) and the tissue or cell type of the cancer (histology). Therefore, each of the cancer types reviewed in this report was evaluated separately. Cancers that occur as the result of the metastasis or the spread of a primary site cancer to another location in the body are not considered as separate cancers and therefore were not included in this analysis.

It should be noted that the MCR research file may contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer diagnosed in an individual by another health-care provider. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital/diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than 2 months after the initial diagnosis (MCR 1996). Therefore, duplicate reports of individuals diagnosed with cancer were removed from the analyses whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases.

B. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer cases occurred in Attleboro and Norton, cancer incidence data were tabulated by gender according to six age groups to compare the observed number of cancer cases to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for each of the 13 primary cancer types for each town as a whole as well as for each census tract (CT) within each town. Specifically, SIRs were calculated for four time periods, 1982–1987, 1988–1993, 1994–1999, and 2000–2002, to evaluate patterns or trends in cancer incidence over time.

To calculate SIRs, it is necessary to obtain accurate community population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 United States census data for each CT in Attleboro and Norton (USDOC 1980, 1990, 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990, 1996, and 2001). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the 10-year interval between each census.[2]

Because accurate age group and gender specific population data are required to calculate SIRs, the CT is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a county as defined by the U.S. Census Bureau. Census tracts usually contain between 2,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (USDOC 1990).

According to the latest United States Census, the city of Attleboro is subdivided into eight census tracts (i.e., CTs 6311 – 6318) and the town of Norton is subdivided into two census tracts (i.e., CTs 6111 and 6112) (USDOC 2000). However, two census tracts in Attleboro (i.e., CTs 6312 and 6315) experienced significant boundary and population changes between the 1980 United States Census and the 1990 United States Census. Therefore, in order to calculate accurate cancer incidence rates in this area of Attleboro over time, population and cancer data for these

[2] Using slightly different population estimates or statistical methodologies, such as grouping ages differently or rounding off numbers at different points during calculations, may produce results slightly different from those published in this report.

CTs were combined and SIRs were calculated as if they were one census tract. The town boundaries and census tract locations for Attleboro and Norton are illustrated in Figure 1. As described previously, the Shpack Landfill is located on the border of Attleboro CT 6317 and Norton CT 6112.

C. Interpretation of a Standardized Incidence Ratio (SIR)

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as a larger comparison population designated as "normal" or average. Usually, the state as a whole is selected to be the comparison population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates.

Specifically, an SIR is the ratio of the observed number of cancer cases in an area to the expected number of cases multiplied by 100. The population structure of each town is adjusted to the statewide incidence rate to calculate the number of expected cancer cases. The SIR is a comparison of the number of cases in the specific area (i.e., city/town or census tract) to the statewide rate. Comparisons of SIRs between towns or census tracts are not possible because each community has different population characteristics.

An SIR of 100 indicates that the number of cancer cases observed in the population being evaluated is equal to the number of cancer cases expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer cases occurred than were expected, and an SIR less than 100 indicates that fewer cancer cases occurred than were expected. Accordingly, an SIR of 150 is interpreted as 50% more cancer cases than the expected number; an SIR of 90 indicates 10% fewer cancer cases than expected.

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on four expected cases and six observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more

stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed for a particular cancer type.

D. Calculation of the 95% Confidence Interval

To help interpret or measure the stability of an SIR, the statistical significance of each SIR was assessed by calculating a 95% confidence interval (95% CI) to determine if the observed number of cases is "significantly different" from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). Specifically, a 95% CI is the range of estimated SIR values that have a 95% probability of including the true SIR for the population. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or "normal" population. "Significantly different" means there is less than a 5% chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer cases.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105–130), there is a statistically significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer cases is statistically significantly lower than expected. If the confidence interval range includes 100, the true SIR may be 100. In this case, it cannot be determined with certainty that the difference between the observed and expected number of cases reflects a real increase or decrease in cancer incidence or is the result of chance. It is important to note that statistical significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be lower or higher than the calculated SIR. This would indicate an unstable statistic. Due to the instability of incidence rates based on small

numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

E. Evaluation of Cancer Risk Factor Information

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, smoking history and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to lung and bronchus, bladder, pancreatic and kidney cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for cancers that were statistically significantly elevated in Attleboro and Norton as well as for individual census tracts. However, information about personal risk factors such as family history, hormonal events, diet, and similar factors that may also influence the development of cancer is not collected by the MCR, and therefore, it was not possible to consider their role in this investigation.

F. Determination of Geographic Distribution of Cancer Cases

In addition to the calculation of SIRs, address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2005). This allowed assignment of census tract location for each case as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level within a census tract (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cases in Attleboro and Norton. This evaluation included consideration of the population density of each community and its variability within each community through the use of GIS-generated population density overlays. In instances where the address information from the MCR was incomplete, that is, did not include specific streets or

street numbers, efforts were made to research those cases using telephone books and city residential lists issued within 2 years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. [Note: MDPH is bound by state and federal patient privacy and research laws not to reveal the name or any other identifying information of an individual diagnosed with cancer and reported to the MCR.]

V. Results of Cancer Incidence Analysis

The following sections present cancer incidence data for Attleboro and Norton as well as for each of their census tracts during the 21-year time period, 1982–2002. The census tract-specific analyses help in understanding whether the incidence of cancers observed city- or town-wide may be explained by an increase or decrease in a particular geographic area of the city or town. To evaluate possible trends over time, these data were analyzed by four time periods, 1982–1987, 1988–1993, 1994–1999, and 2000–2002. Tables 1a through 13d summarize cancer incidence data for Attleboro while Tables 14a through 26d summarize cancer incidence data for Norton. SIRs were not calculated for some cancer types in some time periods and/or census tracts due to the small number of observed cases (less than five). It is standard CHISRE policy not to calculate rates with fewer than five observed cases. However, the expected number of cases was calculated for each time period and census tract, and the observed and expected numbers of cases were compared to determine whether excess numbers of cancer cases were occurring.

The incidence of the 13 cancer types evaluated in Attleboro for the four time periods of 1982–1987, 1988–1993, 1994–1999, and 2000–2002 is discussed below.

A. Cancer Incidence in Attleboro

1. Bladder Cancer (Tables 1a – 1d)

For the city of Attleboro as a whole, the incidence of bladder cancer was lower than expected for three of the four time periods: 1982–1987, 1988–1993, and 1994–1999. For 2000–2002, the number of observed diagnoses among males and females combined was greater than the number

expected (22 diagnoses observed versus 17.2 expected); this increase was due to a statistically significant elevation in females (11 diagnoses observed versus 5.1 expected; SIR = 216, 95% CI = 108-386). In males during this period, the number of observed bladder cancer diagnoses was slightly less than expected (11 diagnoses observed versus 12.1 expected).

Generally, within the CTs of Attleboro, bladder cancer occurred at or near expected rates (i.e., within one or two cases of the expected number) during each of the four time periods. Residents of two CTs experienced increased rates of bladder cancer during different time periods. In CT 6317 during 1988–1993, eight diagnoses were observed in males and females combined compared with approximately five diagnoses expected; this elevation was not statistically significant. In CT 6313 during 2000–2002, seven diagnoses were observed in males and females combined compared to approximately two diagnoses expected; this elevation was statistically significant (SIR = 351, 95% CI = 141-724).

In Attleboro CT 6317, where part of the Shpack Landfill is located, bladder cancer occurred about as expected during three of the four time periods: 1982–1987, 1994–1999, and 2000–2002. As stated earlier, the incidence of bladder cancer was elevated among males and females during 1988–1993 but this increase was not statistically significant (eight diagnoses observed versus 5.2 expected). In the subsequent time period, 1994–1999, the incidence of bladder cancer in this CT was lower than expected (2 diagnoses observed versus 6.3 expected).

2. Bone Cancer (Tables 2a – 2d)

For the city of Attleboro as a whole and for Attleboro's individual CTs, bone cancer generally occurred at or near expected rates for the four time periods evaluated.

3. Brain and Central Nervous System Cancer (Tables 3a – 3d)

For the city of Attleboro as a whole, the incidence of brain and CNS cancer was lower than expected for three of the four time periods: 1982–1987, 1988–1993, and 1994–1999. For 2000–2002, the number of observed diagnoses among males was greater than expected (10 diagnoses observed versus 5.0 expected; SIR = 200, 95% CI = 96-368); however, this elevation was not statistically significant.

Within all of the CTs of Attleboro, brain and CNS cancer occurred at or near expected rates (i.e., within one or two diagnoses of the expected number) during each of the four time periods.

4. Breast Cancer (Tables 4a – 4d)

With the exception of the 1988–1993 period, the incidence of breast cancer in Attleboro as a whole occurred about as expected or less than expected. For 1982–1987, 137 diagnoses were observed compared to approximately 136 expected. For 1994–1999 and 2000–2002, fewer diagnoses of breast cancer occurred town-wide than expected. For 1988–1993, however, 186 diagnoses were observed compared to approximately 164 expected; this elevation was not statistically significant.

With two exceptions, breast cancer occurred in Attleboro CTs at or near the expected rates (within one or two diagnoses of the expected number) for three of the four time periods evaluated. During two of the four time periods, 1982–1987 and 1988–1993, three CTs (6312 & 6315 and 6314) experienced more diagnoses of breast cancer than expected. These elevations were not statistically significant. During the subsequent two time periods evaluated, breast cancer occurred in these three census tracts at or near expected rates (i.e., within one or two diagnoses of the expected number). For 1988–1993, when the number of city-wide diagnoses exceeded the number expected, five of the eight CTs (6312, 6314, 6315, 6316, and 6317) experienced a higher incidence of breast cancer than expected. Except for CT 6317, the differences between the number of observed diagnoses and the number expected were not statistically significant.

In CT 6317, where the Shpack Landfill is located, breast cancer occurred slightly less often than expected (19 diagnoses observed versus 20.1 expected) during the 1982–1987 time period. However, during 1988–1993, the incidence of breast cancer was statistically significantly elevated in this CT (38 observed versus 23.7 expected; SIR = 160, 95% CI = 113-220). In the subsequent two time periods, breast cancer occurred less often than expected in CT 6317. During 1994–1999, 20 diagnoses were observed compared to approximately 30.2 expected and, during 2000–2002, 14 diagnoses were observed compared to approximately 17 expected. In CT 6317, although the SIR for breast cancer was significantly elevated during 1988–1993, a

consistent trend over time was not seen, with the incidence being as expected or less than expected during the other three time periods evaluated.

5. Hodgkin's Disease (Tables 5a – 5d)

For the city of Attleboro as a whole, the incidence of Hodgkin's disease was about as expected (i.e., within one or two diagnoses of the expected number) for two of the four time periods evaluated (1982–1987 and 2000–2002) and higher than expected for the middle two time periods evaluated (1988–1993 and 1994–1999). During 1988–1993, 12 diagnoses were observed city-wide compared to approximately nine cases expected. During 1994–1999, 13 diagnoses were observed city-wide compared to approximately nine diagnoses expected. Neither elevation was statistically significant.

Within the CTs of Attleboro, Hodgkin's disease occurred at or near expected rates (i.e., within one to two diagnoses of the expected number) for three of the four time periods. During 1988–1993, in CTs 6312 & 6315, six diagnoses were observed compared to approximately two expected; this elevation was statistically significant (SIR = 320, 95% CI = 117-696).

In CT 6317, where part of the Shpack Landfill is located, Hodgkin's disease occurred about as expected during the four time periods evaluated. Approximately one diagnosis of Hodgkin's disease would be expected in CT 6317 during each time period; the number of observed diagnoses in this CT during the four time periods ranged from none during 1982–1987 to two in each of the last two time periods.

6. Kidney Cancer (Tables 6a – 6d)

Analysis of city-wide kidney cancer incidence in Attleboro over time revealed that kidney cancer occurred less often than expected during 1982–1987 (10 diagnoses observed versus 16.8 expected), more often than expected during 1988–1993 (26 diagnoses observed versus 23.8 expected), less often than expected during 1994–1999 (20 diagnoses observed versus 27.8 expected), and more often than expected during 2000–2002 (21 diagnoses observed versus 17.0 expected). None of the differences between the number of observed versus the number of expected diagnoses were statistically significant.

Generally, within the CTs of Attleboro, kidney cancer occurred at or near expected rates (i.e., within one or two cases of the expected number) during each of the four time periods. There were two exceptions. In CTs 6312 & 6315 during 1988–1993, nine diagnoses were observed in males and females combined compared with approximately six diagnoses expected; this elevation was not statistically significant. In the other three time periods, kidney cancer occurred at or below expected rates in these two CTs. In CT 6311 during 1994–1999, fewer diagnoses of kidney cancer occurred than expected (3 diagnoses observed versus 5.7 expected); this difference was not statistically significant.

In CT 3617, where part of the Shpack Landfill is located, kidney cancer occurred either as expected or less often than expected over the four time periods evaluated.

7. Leukemia (Tables 7a – 7d)

For the city of Attleboro as a whole, the incidence of leukemia was lower than expected for three of the four time periods: 1982–1987, 1994–1999, and 2000–2002. During 1988–1993, the number of observed diagnoses among males and females combined exceeded the number expected (21 diagnoses observed versus 18.3 expected); however, this increase was not statistically significant.

Within the CTs of Attleboro, leukemia generally occurred at or near expected rates during each of the four time periods with one exception. In CTs 6312 & 6315 during 1982–1987, one diagnosis was observed compared to approximately four expected; this decrease was not statistically significant. In CT 6317, where part of the Shpack Landfill is located, leukemia occurred as expected or less often expected during all four time periods.

8. Liver Cancer (Tables 8a – 8d)

Analysis of city-wide liver cancer incidence in Attleboro over time revealed that liver cancer occurred less often than expected during 1982–1987 (1 diagnosis observed versus 3.6 expected), more often than expected during 1988–1993 (8 diagnoses observed versus 4.9 expected), about as expected during 1994–1999 (7 diagnoses observed versus 7.5 expected), and more often than expected during 2000–2002 (11 diagnoses observed versus 6.1 expected). With the exception of the latest time period, the differences between the number of liver cancer diagnoses observed and

the number expected were not statistically significant. For 2000–2002, a statistically significant elevation in liver cancer in Attleboro males occurred with ten diagnoses observed compared to approximately five diagnoses expected (SIR = 217, 95% CI = 104-399).

Within the CTs of Attleboro, liver cancer generally occurred at or near expected rates during each of the four time periods, with one exception. In CTs 6312 & 6315 during 2000–2002, four diagnoses of liver cancer were observed compared to approximately one diagnosis expected; in the three earlier time periods, liver cancer occurred in these two CTs at or below expected rates. In CT 6317, where part of the Shpack Landfill is located, liver cancer occurred about as expected throughout the four time periods evaluated.

9. Lung and Bronchus Cancer (Tables 9a – 9d)

Analysis of city-wide lung and bronchus cancer rates over time revealed no consistent trends. Among males and females combined, lung and bronchus cancer occurred less often than expected during 1982–1987 (119 diagnoses observed versus 129.2 expected), more often than expected during 1988–1993 (163 diagnoses observed versus 142.4 expected), slightly less often than expected during 1994–1999 (161 diagnoses observed versus 163.4 expected), and more often than expected during 2000–2002 (99 diagnoses observed versus 91.5 expected). None of these differences for males and females combined were statistically significant. Among males, incidence rates were higher than expected during 1982–1987 (88 diagnoses observed versus 78.3 expected), lower than expected during 1988–1993 (78 diagnoses observed versus 83.2 expected), higher than expected during 1994–1999 (104 diagnoses observed versus 83.9 expected), and higher than expected during 2000–2002 (52 diagnoses observed versus 46.2 expected). The difference between the number of lung and bronchus cancer diagnoses observed in males and the number expected was statistically significant only during the 1994–1999 time period (SIR = 124, 95% CI = 101-150). Among females, incidence rates were statistically significantly lower than expected during 1982–1987 (31 observed versus 50.1 expected; SIR = 61, 95% CI = 41-86), statistically significantly elevated during 1988–1993 (85 diagnoses observed versus 59.2 expected; SIR = 144, 95% CI = 115-178), statistically significantly lower than expected during 1994–1999 (57 diagnoses observed versus 79.4 expected; SIR = 72, 95% CI = 54-93), and about as expected during 2000–2002 (47 diagnoses observed versus 45.2 expected).

Within most of the CTs in Attleboro, the incidence of lung and bronchus cancer was higher than expected during at least one of the four time periods evaluated. However, for most of the CTs, when the incidence of lung and bronchus cancer was examined over time, no consistent trends were noticed. In CT 6311, lung and bronchus cancer occurred near or below expected rates for 1982–1987 and 2000–2002. During 1988–1993, however, statistically significantly fewer diagnoses occurred in males in this CT than expected (9 diagnoses observed versus 17.7 expected; SIR = 51, 95% CI = 23-97) while during 1994–1999 more diagnoses occurred in males than expected (26 diagnoses observed versus 18.5 expected), although the elevation was not statistically significant. During 1988-1993, the number of observed diagnoses among females in CT 6311 was statistically significantly greater than expected (21 diagnoses observed versus 11.2 expected; SIR = 187, 95% CI = 116-286) while during 1994-1999 fewer diagnoses occurred in females than expected (12 diagnoses observed versus 15.8 expected). In CT 6314, the number of observed diagnoses was greater than expected for the first three time periods, with statistically significant elevations in females during 1988–1993 (10 diagnoses observed versus 3.0 expected) and in males during 1994–1999 (10 diagnoses observed versus 4.2 expected). In the latest time period, however, the incidence of lung and bronchus cancer was about as expected in this CT for males and females. In CT 6318, the incidence of lung and bronchus cancer in males was elevated consistently across the four time periods, although the elevations were not statistically significant. In CT 6318 females, the incidence of lung and bronchus cancer was lower than expected in 1982–1987 , significantly higher than expected in 1988–1993 (20 diagnoses observed versus 9.5 expected; SIR = 210, 95% CI = 128-324), lower than expected in 1994–1999, and higher than expected in 2000–2002 (12 diagnoses observed versus 7.3 epxected), although not statistically significantly higher.

With one exception, the incidence of lung and bronchus cancer was about as expected (within one or two cases of the expected number) or lower than expected among residents of CT 6317, where the Shpack Landfill is located, during each of the four time periods. The one exception occurred among males in CT 6317 during 1982–1987 when 15 diagnoses were observed compared to approximately 11 expected; the difference was not statistically significant.

10. Multiple Myeloma (Tables 10a – 10d)

For the city of Attleboro as a whole, the incidence of multiple myeloma was about as expected (i.e., within one or two diagnoses of the expected number) for three of the four time periods: 1982–1987, 1988–1993, and 2000–2002. For 1994–1999, however, more males were diagnosed with multiple myeloma than expected (10 diagnoses observed versus 5.3 expected); this elevation was not statistically significant.

Within the CTs of Attleboro (including CT 6317, where part of the Shpack Site is located), multiple myeloma generally occurred at or near expected rates during each of the four time periods, with one exception. In CT 6313 during 1994–1999, four diagnoses were observed compared to approximately one expected.

11. Non-Hodgkin's Lymphoma (Tables 11a – 11d)

For the city of Attleboro as a whole, during the four time periods evaluated, non-Hodgkin's lymphoma (NHL) occurred at or near expected rates (i.e., within one or two diagnoses of the expected number) with two exceptions. During 1988–1993, more females were diagnosed with NHL than expected (21 diagnoses observed versus 17.4 expected). Similarly, during 1994–1999, the incidence of NHL in females was elevated (24 diagnoses observed versus 21.4 expected). Neither elevation was statistically significant.

Generally, within the CTs of Attleboro, NHL occurred at or near expected rates during each of the four time periods. The only exception was during 1988–1993 when more females in CT 6313 were diagnosed with NHL than expected (6 diagnoses observed versus 2.4 expected) and fewer males and females combined in CTs 6312 & 6315 were diagnosed during this period (3 diagnoses observed versus 8.1 expected); neither difference was statistically significant. In CT 6317, where part of the Shpack Landfill is located, NHL occurred at or near expected rates during each of the four time periods.

12. Pancreatic Cancer (Tables 12a – 12d)

For the city of Attleboro as a whole, the incidence of pancreatic cancer was about as expected or lower than expected for the four time periods evaluated, with one exception. During 1994–1999,

more females city-wide were diagnosed with pancreatic cancer than expected (19 diagnoses observed versus 13.2 expected); the elevation was not statistically significant.

Within the CTs of Attleboro, pancreatic cancer generally occurred at or near expected rates with the exception of one CT during one of the four time periods. During 1994–1999, six females in CT 6318 were diagnosed with pancreatic cancer compared to approximately two expected; the elevation was not statistically significant. Pancreatic cancer occurred about as expected within CT 6317, the location of the Shpack Landfill, during all four time periods evaluated.

13. Thyroid Cancer (Tables 13a – 13d)

For the city of Attleboro as a whole, thyroid cancer occurred at or near expected rates (i.e., within one or two cases of the expected number) with a few exceptions. During 1982–1987, fewer diagnoses of thyroid cancer occurred city-wide than expected (3 diagnoses observed versus 7.2 expected). For the following time period, 1988–1993, a statistically significant elevation occurred in males (7 diagnoses observed versus 2.6 expected; SIR = 266, 95% CI = 107-548). During 1994–1999, thyroid cancer occurred about as expected city-wide and then during 2000–2002, an elevation occurred in males and females combined (15 diagnoses observed versus 12.1 expected) but it was not statistically significant.

Within the CTs of Attleboro, thyroid cancer generally occurred at or near expected rates during each of the time periods with a few exceptions. Three different CTs (6311, 6312 & 6315, and 6314) each experienced three additional diagnoses of thyroid cancer over what was expected, each during a different time period. None of these elevations were statistically significant. In CT 6317, the location of the Shpack Landfill, thyroid cancer occurred about as expected during all four time periods.

The incidence of the 13 cancer types evaluated in Norton for the four time periods of 1982–1987, 1988–1993, 1994–1999, and 2000–2002 is discussed below.

B. Cancer Incidence in Norton

1. Bladder Cancer (Tables 14a – 14d)

For the town of Norton as a whole, the incidence of bladder cancer was lower than expected or as expected for all four time periods evaluated. The differences between the number of observed diagnoses of bladder cancer compared to the number expected for 1982–1987, 1988–1993, 1994–1999, and 2000–2002 are as follows, respectively: 3 diagnoses observed versus 10.2 expected, 7 diagnoses observed versus 10.4 expected, 10 diagnoses observed versus 11.4 expected, and 5 diagnoses observed versus 5.3 expected. None of these differences were statistically significant.

Within the CTs of Norton, bladder cancer occurred at or slightly below expected rates during each of the four time periods. In CT 6112, where part of the Shpack Landfill is located, with two exceptions, the number of observed diagnoses of bladder cancer was less than the number expected over the four time periods. The two exceptions were in males during 1994-1999 and females during 2000–2002 when the number of observed diagnoses equaled the number expected.

2. Bone cancer (Tables 15a – 15d)

Two individuals were diagnosed with bone cancer in the town of Norton during the 21-year time period of 1982–2002 compared to approximately three diagnoses expected. These two individuals were residents of CT 6112, one male and one female. One diagnosis occurred during the earliest time period (1982–1987) while the second diagnosis occurred more than six years later during the latest time period (2000–2002).

3. Brain and Central Nervous System Cancer (Tables 16a – 16d)

For the town of Norton as a whole, the incidence of brain and CNS cancer was lower than expected or as expected for three of the four time periods: 1982–1987, 1988–1993, and 1994–1999. For the latest time period of 2000–2002, however, the number of observed diagnoses of brain and CNS cancer was greater than expected (7 diagnoses observed versus 3.5 expected). This elevation was due to an elevation in CT 6112, where part of the Shpack Landfill is located.

In CT 6112, five diagnoses of brain and CNS cancer were observed in males compared to one expected (SIR = 507, 95% CI = 163-1,182); this elevation was statistically significant. In the earlier time periods, the incidence of brain and CNS cancer in CT 6112 males was about as expected in 1982–1987 (1 diagnosis observed versus 1.3 expected), 1988–1993 (1 diagnosis observed versus 1.6 expected), and 1994–1999 (1 diagnosis observed versus 1.7 expected).

4. Breast Cancer (Tables 17a – 17d)

For the town of Norton as a whole, the incidence of breast cancer was lower than expected for three of the four time periods: 1982–1987, 1994–1999, and 2000–2002. For 1982–1987, 32 diagnoses occurred in females compared to approximately 38 expected. For 1994–1999, 54 diagnoses occurred compared to approximately 61 expected. For 2000–2002, 22 diagnoses of breast cancer occurred in females compared to approximately 35 expected. For 1988–1993, breast cancer incidence among females occurred near the expected rate with 52 diagnoses reported compared to approximately 49 diagnoses expected. One diagnosis of breast cancer occurred in a male over the 21-year period of 1982–2002. In CT 6112, where part of the Shpack Landfill is located, the incidence of breast cancer was either lower than expected or about as expected for the four time periods evaluated.

5. Hodgkin's Disease (Tables 18a – 18d)

For the town of Norton as a whole, the incidence of Hodgkin's disease was either lower than expected or as expected for the four time periods evaluated. No diagnoses of Hodgkin's disease occurred during the first time period evaluated. During 1988–1993, the number of observed diagnoses equaled the number expected (three diagnoses). During 1994–1999, one diagnosis of Hodgkin's disease was reported compared to approximately four expected. During 2000–2002, the number of observed diagnoses equaled the number expected (two diagnoses). In CT 6112, where part of the Shpack Landfill is located, two diagnoses of Hodgkin's disease occurred over the 21-year period compared to approximately six diagnoses expected.

6. Kidney Cancer (Tables 19a – 19d)

For the town of Norton as a whole, kidney cancer occurred at or near expected rates (i.e., within one or two diagnoses of the expected number) for three of the four time periods: 1982–1987,

1994–1999, and 2000–2002. For 1988–1993, however, 11 diagnoses were observed compared to approximately seven expected; the elevation was not statistically significant. In CT 6112, where part of the Shpack Landfill is located, kidney cancer occurred about as expected during the four time periods evaluated.

7. Leukemia (Tables 20a – 20d)

For the town of Norton as a whole, the incidence of leukemia was lower than expected for three of the four time periods: 1988–1993, 1994–1999, and 2000–2002. During 1982–1987, leukemia occurred as expected, with five diagnoses observed and approximately five diagnoses expected. In CT 6112, where part of the Shpack Landfill is located, the number of observed diagnoses of leukemia was lower than expected for the four time periods. In CT 6112, five leukemia diagnoses were reported over the 21-year period compared to approximately 13 diagnoses expected.

8. Liver Cancer (Tables 21a – 21d)

For the town of Norton as a whole, and for its two CTs, liver cancer occurred at or near expected rates (i.e., within one diagnosis of the expected number) for the four time periods evaluated. For CT 6112, where part of the Shpack Landfill is located, two diagnoses of liver occurred over the 21-year period compared to approximately four diagnoses expected.

9. Lung and Bronchus Cancer (Tables 22a – 22d)

Analysis of town-wide lung and bronchus cancer rates over time revealed no consistent trends. Among males and females combined, lung and bronchus cancer occurred less often than expected during 1982–1987 (32 diagnoses observed versus 35.4 expected), more often than expected during 1988–1993 (45 diagnoses observed versus 41.2 expected), more often than expected during 1994–1999 (59 diagnoses observed versus 49.5 expected), and about as expected during 2000–2002 (29 diagnoses observed versus 28.9 expected). None of these differences for males and females combined were statistically significant. During 1988–1993, an elevation occurred among males (33 diagnoses observed versus 24.5 expected) although it was not statistically significant. In the next time period, the incidence of lung and bronchus cancer among males was as expected (26 diagnoses observed versus 26.0 expected) and in the last time

period, the incidence among males was higher than expected but not statistically significantly higher (19 diagnoses observed versus 15.0 expected). During 1994–1999, the second time period an elevation among males and females combined was observed, the elevation was due to one among females (33 diagnoses observed versus 23.5 expected) and was of borderline statistical significance. In the two time periods before and after 1994–1999, the incidence of lung and bronchus cancer among females was lower than expected.

In CT 6112, where part of the Shpack Landfill is located, the incidence of lung and bronchus cancer was as expected for 1982–1987, higher than expected for 1988–1993 in males, lower than expected for 1994–1999, and as expected for 2000–2002. None of these differences were statistically significant. In CT 6111, the incidence of lung and bronchus cancer was lower than expected for three of the four time periods: 1982–1987, 1988–1993, and 2000–2002. During 1994–1999, however, a statistically significant elevation occurred among males and females combined (38 diagnoses observed versus 22.9 expected; SIR = 166, 95% CI = 117-227) and among females in this CT (21 diagnoses observed versus 10.8 expected; SIR = 194, 95% CI = 120-297).

10. Multiple Myeloma (Tables 23a – 23d)

For the town of Norton as a whole and for its two CTs, multiple myeloma occurred at or near expected rates (i.e., within one diagnosis of the expected number) during each of the four time periods. For CT 6112, where part of the Shpack Landfill is located, the incidence of multiple myeloma was as expected for all four time periods.

11. Non-Hodgkin's Lymphoma (Tables 24a – 24d)

For the town of Norton as a whole, the incidence of NHL was about as expected for three of the four time periods: 1982–1987, 1994–1999, and 2000–2002. For 1988–1993, the number of observed diagnoses among males and females combined was greater than the number expected (14 diagnoses observed versus 10.9 expected). This elevation was due to a slight increase among both males and females and was not statistically significant.

Within the two CTs in Norton, NHL occurred about as expected with one exception. In CT 6112, during 1988–1993, 11 diagnoses of NHL were reported among males and females while

approximately six diagnoses were expected; the elevation was of borderline statistical significance (SIR = 194, 95% CI = 97-347). An additional three diagnoses occurred among males and an additional two diagnoses occurred among females. In this CT, where part of the Shpack Landfill is located, the incidence of NHL was lower than expected during the other three time periods evaluated.

12. Pancreatic Cancer (Tables 25a – 25b)

For the town of Norton as a whole, the incidence of pancreatic cancer was about as expected (i.e., within one or two diagnoses of the expected number) for two of the four time periods: 1982–1987 and 2000–2002. During the middle two time periods, more diagnoses of pancreatic cancer occurred in females in Norton than expected. During 1988–1993, seven diagnoses occurred town-wide among females in Norton compared to approximately three expected; the elevation was not statistically significant. During 1994–1999, seven diagnoses occurred in females town-wide compared to approximately four diagnoses expected; this elevation was not statistically significant.

Generally, within the two CTs in Norton, pancreatic cancer occurred at or near expected rates (that is, the incidence was slightly more or less than expected) across all four time periods. The one exception that occurred was in CT 6112 in 1988–1993 when six diagnoses occurred among males and females combined when approximately three diagnoses were expected. The elevation was not statistically significant.

13. Thyroid Cancer (Tables 26a – 26b)

For the town of Norton as a whole, the incidence of thyroid cancer was lower than expected for three of the four time periods: 1982–1987, 1988–1993, and 1994–1999. During 2000–2002, thyroid cancer occurred more often than expected among females with seven diagnoses observed compared to approximately four diagnoses expected; the difference was not statistically significant.

Within the two CTs in Norton, thyroid cancer occurred at or near expected rates during each of the four time periods.

VI. Review of Cancer Risk Factor Information

As previously mentioned, cancer is not one disease but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (the time between exposure and development of disease), characteristics, and trends in survival. Available information from the MCR related to age, gender, and residence, as well as other factors related to the development of cancer such as smoking and occupation, was reviewed for those cancer types that had statistically significant elevations in incidence in Attleboro, Norton, or individual census tracts during one or more time periods evaluated. Those cancer types included bladder cancer, brain & CNS cancer, breast cancer, Hodgkin's disease, liver cancer, lung and bronchus cancer, and thyroid cancer. Information for each of these cancer types was compared to known or established incidence trends to assess whether any unexpected patterns existed among these cases. For detailed information regarding risk factors associated with all cancer types evaluated in this report, please refer to Appendix C.

Age and gender are risk factors in many types of cancers. A review of age-group specific SIRs by census tract was not possible because of the small numbers of cases in each group (i.e., less than five). However, where there was a statistically significant elevation of cancer cases, the distribution of the age at which individuals were diagnosed was qualitatively reviewed.

Tobacco use is also a known or suspected causal risk factor in several types of cancer, including bladder and lung and bronchus cancers. The smoking history of individuals diagnosed with these types of cancers in Attleboro and Norton was reviewed for those census tracts in which statistically significant elevations in incidence were observed.

The staging of cancer categorizes the extent of the disease and its spread at the time of diagnosis. The distribution of stage of disease at diagnosis for females diagnosed with breast cancer was evaluated for the one CT in Attleboro where the rate was statistically significantly elevated compared to that for the state of Massachusetts.

Breast cancer survival is strongly correlated with an early stage at diagnosis. An evaluation of staging patterns can be used to evaluate the level of screening in a particular area. Communities

in which a large portion of the women are receiving appropriate breast cancer screening (mammography and clinical breast exams) are expected to have a greater number of women diagnosed with earlier stage disease. For this analysis, stage of disease was divided into four categories: local, regional, distant, and unknown. Local stage refers to a diagnosis in which the tumor is invasive but the cancer is confined to the organ of origin. Regional refers to a tumor that has spread beyond the organ of origin, including spread to adjacent tissues, organs, or lymph nodes. Distant stage cancer has metastasized or spread to organs other than those adjacent to the organ of origin, to distant lymph nodes or to both. Some cases are reported to the MCR with an unknown stage, meaning that at the time of reporting the tumor had not yet been staged.

In some studies, an association has been found between specific occupations and an increase in the incidence of bladder cancer, brain and CNS cancer, breast cancer, liver cancer, and lung and bronchus cancer. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types in census tracts with statistically significant elevations, to determine the role that occupational factors may have played in the development of cancers in these areas. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. Further, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

Finally, histologic (cell type) distribution was reviewed for diagnoses of brain and CNS cancer, Hodgkin's disease, and lung and bronchus cancer for census tracts that experienced statistically significantly elevated incidence rates compared to the state. Patterns of disease were compared to known or established incidence trends to assess whether any unexpected patterns exist in these areas.

A. Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 63,210 people in the United States in 2005 (ACS 2005a). Males are three times more likely to develop bladder cancer than females. The risk of bladder cancer increases with age and the mean age at diagnosis is 68-69 years. The greatest risk factor for bladder cancer is cigarette smoking, with smokers being

more than twice as likely to develop bladder cancer as nonsmokers. Studies have also revealed a number of occupations that are associated with bladder cancer. Exposure to certain chemicals in the workplace account for approximately 20-25% of all bladder cancers diagnosed among men in the United States. Transitional cell carcinoma is the most common type of bladder cancer, causing 90% of cases or more in the United States. In the United States, 3-5% of cases are squamous cell carcinoma, 2% or fewer are adenocarcinoma, and 1% or fewer are rhabdomyosarcoma (ACS 2005a).

1) Attleboro, 2000–2002

During 2000–2002, females in Attleboro experienced a statistically significant elevation in the incidence of bladder cancer (11 diagnoses observed versus 5.1 expected; SIR = 216, 95% CI = 108-386) (Table 1d). In the previous time periods, bladder cancer in females occurred less often than expected during 1982–1987, about as expected during 1988–1993, and less often than expected during 1994–1999. The average age at diagnosis for females diagnosed with bladder cancer during 2000–2002 was 74 years old and all of the individuals were age 50 or older at the time of their diagnosis. For the five females for which some occupational information was provided to the MCR, none reported jobs in which occupational exposures possibly associated with the development of bladder cancer would have been likely. Occupational information was unknown for six of the 11 females with bladder cancer. Ten of the 11 females with bladder cancer were diagnosed with transitional cell carcinoma, the most common type of bladder cancer. One of the females had squamous cell carcinoma, a type of bladder cancer found in 3-5% of cases nationally. Of the 11 females, three were reported to the MCR as current or former smokers at the time of diagnosis, five were nonsmokers, and the smoking history of three females was unknown.

2) Attleboro CT 6313, 2000–2002

Bladder cancer was statistically significantly elevated in CT 6313 during 2000–2002 with seven cases observed in males and females combined compared to two diagnoses expected (SIR = 351, 95% CI = 141-724) (Table 1d). Four diagnoses were reported in males and three in females; approximately one diagnosis was expected for males and one for females. In the previous three time periods, fewer cases of bladder cancer occurred than expected in this census tract. The

average age at diagnosis for the seven cases of bladder cancer diagnosed during 2000–2002 was 76 years old and all of the individuals were age 50 or older at the time of their diagnosis. Of the five individuals with some occupational information reported to the MCR, none of the individuals reported jobs in which occupational exposures possibly associated with the development of bladder cancer would have been likely. All seven individuals were diagnosed with transitional cell carcinoma, the most common type of bladder cancer. While the smoking history of two of the seven individuals was unknown, three of the seven were reported to the MCR as current or former smokers at the time of their diagnosis and two of the seven were nonsmokers.

B. Brain and Central Nervous System Cancer

The American Cancer Society estimates that 18, 500 Americans (10,620 men and 7,880 women) will be diagnosed with primary brain cancer (including cancers of the CNS, or spinal cord) in 2005 (ACS 2005). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to 75. Incidence rates are higher in males than females for all types of brain cancer. In adults, the most frequent types of brain tumors are astrocytic tumors, mainly astrocytomas and glioblastoma multiforme. Despite numerous scientific investigations, the causes of brain cancer are still largely unknown. However, a few risk factors have been identified. The most well-established risk factor is exposure to ionizing radiation (e.g., from radiation therapy to the head and neck). In addition, rare cases of brain cancer run in some families. Some types have also been associated with certain rare genetic disorders, such as neurofibromatosis type 1, von Hippel-Lindau disease, and Li-Fraumeni syndrome. Environmental exposures, such as vinyl chloride, aspartame (a sugar substitute), and electromagnetic fields, have been suggested as risk factors for brain cancer, but the evidence to support these associations is inconsistent.

1) Norton CT 6112, 2000–2002

During 2000–2002, the incidence of brain and CNS cancer was statistically significantly elevated in Norton CT 6112, primarily due to an elevation in males (5 diagnoses observed versus 1.0 expected; SIR = 507, 95% CI = 163-1,182). During the earlier three time periods evaluated, brain and CNS cancer occurred about as expected in males in CT 6112.

The average age at diagnosis for males diagnosed with brain and CNS cancer during 2000–2002 was 66 years and four out of five of those diagnosed were age 50 or older at diagnosis. This is consistent with what would be expected based on the epidemiologic literature. Of the five males in CT 6112, four were diagnosed with a glioma-type of brain cancer, one of the most common forms of adult brain cancer. The fifth individual had an unclassified malignant neoplasm of the brain. None of these individuals reported jobs in which occupational exposures possibly associated with the development of brain cancer would have been likely.

C. Breast Cancer

Breast cancer is the most frequently diagnosed cancer among females in the United States and in Massachusetts. An estimated 211,240 new invasive cases of breast cancer were expected to occur among United States females in 2005 while about 1,690 cases were expected among males (ACS 2005b). The risk of breast cancer also increases with age. About 77% of females with breast cancer are over the age of 50 when diagnosed. Females in their 40s account for about 18% of cases and females in their 30s and younger account for about 5% of cases (ACS 2005b). Occupational exposures associated with increased risk for breast cancer have not been clearly identified. However, experimental data suggest that exposure to certain organic solvents and other chemicals [such as benzene, trichloropropane, vinyl chloride, and polycyclic aromatic hydrocarbons (PAHs)] cause breast tumors in animals and thus may contribute to such tumors in humans (Goldberg and Labreche 1996). Other occupational and environmental exposures that have been suggested to increase the risk for breast cancer include polychlorinated biphenyls (PCBs), chlorinated hydrocarbon pesticides (DDT and DDE), and other endocrine-disrupting chemicals (ACS 2005b).

Age, gender, occupation, and stage at diagnosis were reviewed for individuals diagnosed with breast cancer in Attleboro during 1988–1993 in CT 6317 because this was the only CT where a statistically significant elevation in breast cancer occurred during any of the four time periods evaluated.

1) Attleboro CT 6317, 1988–1993

During 1988–1993, females of CT 6317 (where part of the Shpack Landfill is located) experienced a statistically significant elevation in the incidence of breast cancer (38 diagnoses observed versus 23.5 expected; SIR = 162, 95% CI = 114-222). This cancer type occurred less often than expected in this census tract during the previous time period (1982–1987) as well as the two time periods that followed (1994–1999 and 2000–2002). The average age at diagnosis for females diagnosed with breast cancer during 1988–1993 was 62 years and 82% of those diagnosed were age 50 or older at diagnosis. This is comparable to the national experience. None of these individuals reported jobs in which occupational exposures possibly associated with the development of breast cancer would have been likely.

In CT 6317, between 1988–1993, approximately 66% of invasive breast cancer cases reported were local tumors, approximately 26% were regional, approximately 5% were distant, and 3% were of an unknown stage. This is very similar to the distribution observed statewide during this time period (62% local, 28% regional, 5% distant, and 5% unknown), however more women in CT 6317 were diagnosed in the earliest stage in comparison to the state as a whole.

D. Hodgkin's Disease

Hodgkin's disease is a form of cancer that involves the lymphatic system. It is more common among males than females. Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 and 40 (typically ages 25-30) and the second peak occurs in adults aged 55 years and above. However, about 10% to 15% of cases are diagnosed in children 16 years of age or younger (ACS 2005c).

Hodgkin's disease has four major histological subtypes: lymphocytic predominance (LP), nodular sclerosis (NS), mixed cellularity (MC), and lymphocyte depletion (LD) (Mueller 1996). NS Hodgkin's disease is the predominant histology in the young adult age group (ages 15-39), while MC Hodgkin's disease is relatively more frequent in children and older adults (Jarrett and MacKenzie 1999).

The association between Epstein-Barr virus (EBV) and Hodgkin's disease is now well established (Mueller 1996; Jarrett and MacKenzie 1999; Weiss 2000). EBV is a herpes virus and has a widespread distribution throughout the world with more than 80 % of healthy adults infected by the third decade of life (Jarrett and MacKenzie 1999). Primary infection is usually asymptomatic but when infection is delayed until adolescence, as is frequent in developed countries, EBV causes infectious mononucleosis in about 50 % of cases (Jarrett and MacKenzie 1999). The association of Hodgkin's disease and infectious mononucleosis appears primarily among young adult patients (Jarrett and MacKenzie 1999, Hjalgrim et al. 2000).

The clinical and cellular features of Hodgkin's disease suggest a chronic infectious process is associated with Hodgkin's disease, making this cancer an exception from what is generally known of cancer (Mueller 1996). Besides age, gender, and infection with EBV, no other major risk factors for Hodgkin's disease have been established. Occupational exposures to woodworkers and workers in the chemical industry have been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller 1996).

Age, gender, histology and occupation were reviewed for individuals diagnosed with Hodgkin's disease in Attleboro CTs 6312 & 6315 (combined).

1) Attleboro CT 6312 & 6315, 1988–1993

Residents of CTs 6312 & 6315 (combined) experienced a statistically significant elevation in the incidence of Hodgkin's disease during 1988–1993. (As mentioned previously, these two CTs experienced significant boundary and population changes between the 1980 United States Census and the 1990 United States Census. Therefore, in order to calculate accurate cancer incidence rates in this area of Attleboro over time, population and cancer data for these CTs were combined and SIRs were calculated as if they were one CT.) Of the six individuals diagnosed, four were male and two were female. The average age at diagnosis was 34.5 years (age range = 11-77). One individual diagnosed with Hodgkin's disease during 1988–1993 was a child, three were young adults (between the ages of 15-39), and two were older adults. Two of the three

young adults were diagnosed with the nodular sclerosis sub-type of Hodgkin's disease (the predominant histology in young adults) while the third young adult was diagnosed with a less common sub-type of Hodgkin's disease. The child and one of the two older adults were diagnosed with the lymphocyte depletion sub-type, while the second older adult was diagnosed with the lymphocytic predominance sub-type. None of the adults reported employment in an industry in which occupational exposures associated with the disease would have been likely. It should also be noted that, during the other three time periods evaluated, the observed number of diagnoses of Hodgkin's disease among residents of CTs 6312 & 6315 was closer to the number expected (3 observed versus approximately 2 expected).

E. Liver Cancer

An estimated 17,550 people in the United States (12,130 men and 5,420 women) will be diagnosed with liver cancer in 2005, accounting for approximately 1% of all new diagnoses of cancer (ACS 2005d). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Men are at least two to three times more likely to develop liver cancer than women (Yu et al. 2000). Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn 1996). While chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS 2005d), epidemiologic and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, vinyl chloride (a known human carcinogen used in the manufacturing of some plastics) and thorium dioxide (used in the past for certain x-ray tests) are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2005d; London and McGlynn 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. Exposure to arsenic has also been associated with an increased risk of liver cancer (ATSDR 2000).

1) Attleboro, 2000–2002

During 2000–2002, males in Attleboro experienced a statistically significant elevation in the incidence of liver cancer (10 diagnoses observed versus 4.6 expected; SIR = 217, 95% CI = 104-399). In the preceding time periods, liver cancer among males in Attleboro occurred less often

than expected during 1982–1987, more often than expected during 1988–1993 (although not statistically significantly elevated), and less often than expected during 1994–1999. The average age at diagnosis for males diagnosed with liver cancer during 2000–2002 was 69 years old and 90% of those diagnosed were age 50 or older at diagnosis. The majority (70%) of the liver cancers in Attleboro males during this period were hepatocellular carcinomas. This is consistent with the epidemiological literature which reports that about 75% of all liver cancers are of this histology. Four of the ten males worked in occupations with the potential for chemical exposures on the job, although specific job duty information that could identify the types of chemicals used on the job and further define exposure potential was not available.

F. Lung and Bronchus Cancer

According to the epidemiological literature, the incidence of lung cancer increases sharply with age peaking at about age 60 to 70. Only 2% of lung cancers occur before the age of 40. In addition, lung cancer is generally observed more often among males than females (Blot and Fraumeni 1996, MCR 2002).

More than 80% of all lung cancers are thought to be caused directly by smoking cigarettes or by exposure to second hand smoke, or environmental tobacco smoke (ACS 2005e). An increase in cigarette smoking among females has produced lung cancer incidence rates that more closely resemble those experienced by males. The risk of developing lung cancer depends on the intensity of one's smoking habits (e.g., duration of habit, amount smoked, tar yield of cigarette, and filter type). Smoking cessation decreases the elevated risk by about 50%; however, former smokers still carry a greater risk of developing lung cancer than those who have never smoked.

Several occupational exposures have been identified as playing a role in the development of lung cancer. For example, workplace exposure to asbestos is an established risk factor for this disease. Underground miners exposed to radon and uranium are also at an increased risk for developing lung cancer. Other occupations potentially associated with this cancer include chemical workers, talc miners and millers, paper and pulp workers, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers. In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing

radiation, and fuels such as gasoline are also occupational risk factors for lung cancer. Occupational exposure to these compounds in conjunction with cigarette smoking can dramatically increase the risk of developing lung cancer (Blot and Fraumeni 1996).

Lung cancer is divided into two main types: small cell lung cancer and non-small cell lung cancer. Non-small cell lung cancer is further sub-divided into three types: adenocarcinoma, squamous cell carcinoma, and large-cell undifferentiated carcinoma. The different types of lung cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung cancers are adenocarcinomas, 25-30% are squamous cell carcinomas, 20% are small cell cancers, and 10-15% of cases are large cell carcinomas (ACS 2005e). Rates in Massachusetts are very similar to those seen nationally.

Age, gender, smoking history, occupation, and histology distribution were reviewed for those individuals diagnosed with lung and bronchus cancer in Attleboro and Norton, with a particular focus on those census tracts that experienced statistically significant elevations in the incidence of this cancer type.

1). Attleboro, 1988–1993

Although lung and bronchus cancer occurred slightly below expected rates among males in Attleboro during 1988–1993, females experienced a city-wide statistically significant elevation during this time period (85 diagnoses observed versus 59.2 expected; SIR = 144, 95% CI = 115-178). The majority of females (89%) were age 50 or older at the time of diagnosis and the average age at diagnosis was 66. Review of age group-specific SIRs revealed that the overall elevation was not the result of increased incidence among females in any one age group. Rather, increases were noted across all age groups.

For females with a known smoking history, the majority (approximately 88%, n = 53) of individuals diagnosed with lung and bronchus cancer during 1988–1993 reported being current or former smokers at the time of diagnosis. This is consistent with trends observed among all females in the state diagnosed with lung and bronchus cancer (during 1988–1993) and who had a known smoking status at the time of diagnosis (approximately 89% were current or former

smokers). Smoking history was unknown for 29% (n = 25) of lung cancer cases diagnosed among females in Attleboro during this time period.

None of the 85 females indicated jobs in which exposures to chemicals would have been likely. However, occupation was unknown or reported as "retired" or "at home" for more than half (58%, n = 49) of these individuals.

Among the diagnoses with specified histology type (n = 66), 40% were adenocarcinomas, 24% were squamous cell carcinomas, 21% were small cell carcinomas, and 15% were large cell carcinomas. This pattern is consistent with established trends in lung and bronchus cancer incidence in the general population.

As stated earlier, the MDPH conducted a radon survey in Attleboro as part of its July 2001 investigation (MDPH 2001). Results of the radon survey in Attleboro showed that radon levels measured both in the homes or former homes of female lung cancer cases and in the randomly selected households were below the USEPA recommended remediation level of 4 picocuries per liter. However, the median radon concentration measured in case homes (2.4 picocuries per liter) was higher than the median radon concentration measured in the randomly selected control homes (1.9 picocuries per liter).

2). Attleboro, 1994–1999

Although the overall rate of lung and bronchus cancer was as expected in Attleboro during 1994–1999, males experienced a statistically significant elevation in the incidence of this cancer type (104 diagnoses observed versus 83.9 expected; SIR = 124, 95% CI = 101-150). Among the 104 males diagnosed, 94% (n = 98) were age 50 or older at the time of diagnosis and the average age at diagnosis was 67 years.

For males with a known smoking history, the majority (approximately 89%, n = 73) reported being current or former smokers at the time of diagnosis. Approximately 95% of males statewide with a known smoking status who were diagnosed with lung and bronchus cancer during this time period reported being current or former smokers at the time of diagnosis.

Smoking history was unknown for 21% (n = 22) of lung and bronchus cancer cases diagnosed among males in Attleboro during 1994–1999.

Most of the males diagnosed did not report jobs associated with an increased risk of lung and bronchus cancer. However, occupational exposures may have been possible for about 6% of the males diagnosed with lung and bronchus cancer in Attleboro during this time period (e.g., for those individuals employed in the construction and metalworking industries). Occupation was unknown or reported as "retired" for 21% (n = 22) of the individuals.

Review of the distribution of diagnoses by histology type revealed that there were more diagnoses of squamous cell carcinoma and large cell carcinoma and fewer diagnoses of adenocarcinoma in this population of males than expected based on national and statewide incidence trends. Specifically, of the diagnoses with a specific histology classification (n = 81), 26% were adenocarcinomas, 35% were squamous cell carcinomas, 21% were small cell carcinomas, and 18% were large cell carcinomas.

3). Attleboro CT 6311, 1988–1993

Females in CT 6311 experienced a statistically significant elevation in the incidence of lung and bronchus cancer during 1988–1993 (21 diagnoses observed versus 11.2 expected; SIR = 187, 95% CI = 116-286). The average age at diagnosis was 64 years. Review of age group-specific SIRs indicated that the observed elevation was not the result of increased incidence among females in any one age group.

Among females in this CT with a known smoking history, approximately 91% (n = 10) were current or former smokers at the time of diagnosis compared to approximately 89% of females in the state as a whole. One female was a nonsmoker and smoking history was unknown for the remaining ten individuals (48%).

None of these females reported working in occupations thought to be associated with an increased risk of lung and bronchus cancer. However, occupation was unknown or reported as "homemaker" for 62% (n = 13) of these individuals.

The distribution of diagnoses by histology type was as follows: 43% of the 21 diagnoses were adenocarcinomas, 19% were squamous cell carcinomas, 29% were small cell carcinomas, and 9% were large cell carcinomas). The percentages of adenocarcinomas and large cell carcinomas were close to expected while fewer squamous cell and more small cell carcinomas occurred than expected.

It should also be noted that lung cancer among females occurred less often than expected in this census tract during 1982–1987, 1994–1999, and 2000–2002.

As stated earlier, the MDPH conducted a radon survey in Attleboro as part of its July 2001 investigation (MDPH 2001). Radon levels were evaluated in CT 6311, in addition to the city as a whole, because of the statistically significant elevation in female lung cancer incidence during 1987-1994 (one of two time periods evaluated) in CT 6311. For this census tract, the median radon concentration in the homes or former homes of cases was 1.7 picocuries per liter compared to a median radon concentration in the randomly selected homes of 2.9 picocuries per liter.

4). Attleboro CT 6314, 1988–1993

Statistically significant elevations in lung and bronchus cancer incidence were noted among males and females combined and among females when evaluated separately in Attleboro CT 6314 during 1988–1993. Among the 17 males and females, the average age at diagnosis in this census tract was 66 years. Among the ten females diagnosed, the average age at diagnosis was 70 years. Review of age group-specific SIRs indicated that males ages 45-64 were diagnosed with lung and bronchus cancer more often than expected. Slight elevations in incidence were noted among females in most age groups (i.e., 45-64, 65-74, 75-84, and 85+).

Of the residents of CT 6314 with a known smoking history, approximately 80% (n = 12) reported being current or former smokers at the time of diagnosis and three were nonsmokers. Smoking history was unknown for the remaining two individuals. Among residents of Massachusetts diagnosed during the same time period with lung and bronchus cancer and with a known smoking history, approximately 93% were current or former smokers at the time of diagnosis and 7% reported being nonsmokers.

Among the females in this CT with a known smoking history, 78% (n=7) were current or former smokers at the time of diagnosis and two were nonsmokers. One female diagnosed with lung and bronchus cancer during 1988–1993 had an unknown smoking history. Approximately 89% of females in the state were current or former smokers at the time of diagnosis while 11% were nonsmokers.

The majority of individuals diagnosed with lung and bronchus cancer in CT 6314 were not reported as being employed in occupations in which exposures to chemical compounds associated with the development of lung and bronchus cancer were likely. However, two males reported occupations in which exposures may have been possible. Occupation was unknown or reported as "homemaker" for 29% (n = 5) of the 17 individuals and 40% (n = 4) of the ten females.

The histology distribution among individuals diagnosed with lung and bronchus cancer in CT 6314 during 1988–1993 differed from that seen in the general population. Specifically, among the 14 cases with specified histology, 7% were diagnosed as adenocarcinomas, 29% were squamous cell carcinomas, 36% were small cell carcinomas, and 28% were large cell carcinomas. Among the eight females with specified histology, 13% of the cases were adenocarcinomas, 25% were squamous cell carcinomas, 37% were small cell carcinomas, and 25% were large cell carcinomas. The most prevalent histology seen was that of small cell carcinoma. Of those individuals diagnosed with small cell carcinoma, which is the type of lung and bronchus cancer most strongly associated with smoking, all were reported to the MCR as either current or former smokers at the time of diagnosis.

5). Attleboro CT 6314, 1994–1999

Males in Attleboro CT 6314 experienced a statistically significant elevation in the incidence of lung and bronchus cancer during 1994–1999 (10 diagnoses observed versus 4.2 expected; SIR = 237, 95% CI = 113-436). The average age at diagnosis for these individuals was 66 years. The observed elevation was primarily due to an increase in the number of diagnoses among males in age groups 45-64 and 65-74.

Of the ten males diagnosed with lung and bronchus cancer, 100% of those with a known smoking status were current or former smokers at the time of diagnosis. Statewide, 95% of males with lung and bronchus cancer were current or former smokers at the time of diagnosis while five percent were nonsmokers at the time of diagnosis. Smoking history was unknown for two males (20%) in this CT compared to 19% statewide.

Among the ten males diagnosed with lung and bronchus cancer in CT 6314, occupation was listed as "retired" for one person and the remaining nine individuals reported jobs in which exposures associated with the development of lung and bronchus cancer would have been unlikely.

The distribution of cases by histology type among males in this CT was as follows: one adenocarcinoma, four squamous cell carcinomas, one small cell carcinoma, and two large cell carcinomas. The histology type for two males diagnosed with lung and bronchus cancer in this census tract was not specified in the MCR.

6). Attleboro CT 6318, 1988–1993

Statistically significant elevations in the incidence of lung and bronchus cancer were observed among males and females combined and among females when evaluated separately in Attleboro CT 6318 during 1988–1993. Overall, 35 individuals were diagnosed with this cancer type at an average age of 68 years. Of the 20 females who were diagnosed, the average age at diagnosis was 69 years. The elevation observed among females was primarily due to an increase in the number of diagnoses among females in older age groups (i.e., 65-74 and 75-84) while males in age groups 45-64, 65-74, and 75-84 were diagnosed more often than expected.

Of the 35 individuals diagnosed with lung and bronchus cancer, 100% of those with a known smoking status were current or former smokers at the time of diagnosis (versus approximately 93% for the state as a whole). Smoking history was unknown for ten individuals in this CT.

The majority of these individuals were not reported as being employed in occupations in which exposures to chemical compounds associated with the development of lung and bronchus cancer were likely. However, two males reported occupations in which exposures contributing to their

diagnosis may have been possible. Occupation was listed as unknown, retired, or at home for 51% (n = 18) of the 35 individuals and 75% (n = 15) of the 20 females.

Adenocarcinomas represented a smaller proportion of diagnoses than expected in this area of the city while squamous cell carcinomas and, to a lesser extent, large cell carcinomas represented larger proportions of diagnoses for those cases that had a specific histology type (n = 25). Specifically, 20% of these diagnoses were adenocarcinomas, 48% were squamous cell carcinomas, 16% were small cell carcinomas, and 16% were large cell carcinomas. Among the 15 females with a specified histology type, 33% of the cases were adenocarcinomas, 47% were squamous cell carcinomas, 7% were small cell carcinomas, and 13% were large cell carcinomas.

7). Norton CT 6111, 1994–1999

During 1994–1999, residents of Norton CT 6111 experienced a statistically significant elevation in the incidence of lung and bronchus cancer (38 diagnoses observed versus 22.9 expected; SIR = 166, 95% CI = 117-227). When evaluated separately by gender, a statistically significant elevation was also observed among females (21 females diagnosed versus 10.8 expected; SIR = 194, 95% CI = 120-297). The average age at diagnosis for males and females combined was 67 years and 89% (n = 34) of those diagnosed were over the age of 50 at the time of diagnosis. Among the 21 females diagnosed with lung and bronchus cancer, the average age of diagnosis was 65 years and 90% (n = 19) were over the age of 50 at the time of diagnosis. Review of age group-specific SIRs suggests that the observed elevations were primarily due to increased incidence among males aged 65-74 and 75-84 and among females aged 45-64 and 65-74.

Of the individuals in this CT with a known smoking history, all but one individual (or approximately 97%, n = 29) reported being current or former smokers at the time of diagnosis (versus 92% in this time period statewide). Smoking history was unknown for eight (21%) of the individuals in CT 6111 diagnosed with lung and bronchus cancer compared to 19% statewide during this period.

Among females in this CT with a known smoking history, approximately 95% (n = 19) were current or former smokers compared to 89% statewide. One female was a nonsmoker, and smoking history was unknown for the remaining female diagnosed with lung and bronchus

cancer. Females in this CT of Norton with a known smoking history had a higher percentage of current or former smokers when compared with females in the state as a whole diagnosed with this cancer type during 1994–1999 who had a known smoking history.

The majority of individuals did not report working in jobs where exposures that could have contributed to their disease would have been likely. However, three males reported working in the construction industry. Overall, 34% (n = 13) had occupation listed as unknown, retired, or at home. Among females, 29% (n = 6) had occupation listed as unknown, retired, or at home.

In general, review of the distribution of diagnoses by histology type revealed patterns similar to those observed in the general population. Specifically, of the 31 individuals for whom a specific histology type was available, 39% were diagnosed with adenocarcinomas, 23% with squamous cell carcinomas, 22% with small cell carcinomas, and 16% with large cell carcinomas. Among females with specified histology type (n = 19), 47% of the cases were adenocarcinomas, 10% were squamous cell carcinomas, 32% were small cell carcinomas, and 11% were large cell carcinomas.

G. Thyroid Cancer

The thyroid is one of the least cancer-prone organs in the body, representing less than 1% of all cancers occurring among males in the United States and 2.4% among United States females (ACS 2005f). Thyroid cancer is primarily associated with external x-ray treatments of benign medical conditions in childhood or exposure to external radiation (e.g., from atomic bomb fallout exposures). Gender and age also play roles in the development of this disease. Specifically, thyroid cancers occur more often in females than males and, although they can be diagnosed at any age, are generally found in people between the ages of 30 and 50 years (ACS 2005f). Males in the city of Attleboro experienced a statistically significant elevation in the incidence of thyroid cancer during 1988–1993 (7 diagnoses observed versus 2.6 expected; SIR = 266, 95% CI = 107-548).

1). Attleboro, 1988–1993

During 1988–1993, seven males in the city of Attleboro were diagnosed with thyroid cancer compared to 2.6 expected. The average age at diagnosis for these individuals was 59, which is

slightly older than expected for this cancer type. The increase in the incidence of thyroid cancer among males during this time period cannot be explained by an increase in any one area (i.e., census tract) of the city. No males in Attleboro were diagnosed with thyroid cancer during 1982–1987. In the two most recent time periods, 1994–1999 and 2000–2002, five cases of thyroid cancer occurred in males in both periods compared to four and three cases expected, respectively.

VII. Analysis of Geographic Distribution of Cancer Incidence

In addition to determining census tract-specific incidence rates for each cancer type, a qualitative evaluation of the point pattern of cancer diagnoses was conducted. Place of residence at the time of diagnosis was mapped for each individual diagnosed with the cancer types evaluated in this report to assess any possible geographic concentrations of cases in relation to each other or in relation to a potential source of environmental contamination. As previously mentioned, cancer is one word that describes many different diseases. Therefore, for the purposes of this evaluation, the geographic distribution of each cancer type was evaluated separately to determine whether an atypical pattern of any one type was occurring.

With two exceptions, review of the geographic distribution of cancer for the years 1982–2002 in Attleboro and Norton did not reveal any unusual spatial patterns or concentrations of cases at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents of these communities. Although statistically significant elevations of some cancer types were noted in some census tracts of Norton and Attleboro during one or more time periods evaluated, the geographic distribution of diagnoses of these statistically significantly elevated cancers seemed to coincide closely with the pattern of the population in these areas. While four individuals diagnosed with the same subtype of kidney cancer between 1988 and 1999 lived in close proximity to one another in the northernmost corner of Attleboro, near Mansfield, their residences were in the most densely populated area of this part of Attleboro and were over 3 miles from the Shpack Landfill. Also, while three individuals were diagnosed with the same subtype of leukemia between 1994 and 1999, they were all over the age of 65 (which is consistent with what would be expected) and resided approximately 1.5 miles from the Shpack Landfill. Finally, no apparent concentrations of any

specific cancer type were observed in the vicinity of the Shpack Landfill. Although a statistically significant elevation of brain cancer was observed during 2000-2002 in Norton males living in CT 6112, where the Shpack Landfill is located, the residences of the five males diagnosed during this period were spread throughout the census tract. In Attleboro CT 6317, where the Shpack Landfill is located, a statistically significant elevation in breast cancer in females occurred during 1988-1993; the geographic distribution of place of residence of these females closely followed the population distribution in this census tract and no atypical spatial pattern was noted adjacent to the Shpack Landfill itself.

VIII. Cancer Incidence in Shpack Landfill Neighborhoods

To further address the concerns of residents living in close proximity to the Shpack Landfill, an analysis of all types of cancer diagnosed in this neighborhood from 1982 to the present was completed. For this evaluation, the pattern of all cancer diagnoses was reviewed for the area that is within 1 mile of the perimeter of the Shpack Landfill (Figure 2). Seventy-six streets, in part or whole, are included in this area, which is roughly defined by Wilmarth Street to the south, Rambler Road to the west, Precourt Lane to the north, and Dearborn Drive to the east.

In general, our review found no atypical patterns of cancer in the neighborhood surrounding the site. From 1982 to the present, a total of 35 different types of cancer were diagnosed among residents of this area, representing the occurrence of many different diseases.

The most commonly reported diagnoses included cancers of the lung and bronchus, breast, prostate, and colon/rectum. These are the four most common types of cancer diagnosed among men and women in Massachusetts and this pattern is consistent with national and statewide trends in cancer incidence (MDPH 2005, Ries et al. 2005). Together, these cancer types represented more than half (60%) of the cancer diagnoses in this area. There were also a number of other cancer types diagnosed among residents of this area over the 24-year period reviewed including cancers of the bladder, bone, brain, cervix, esophagus, kidney, larynx, liver, oral cavity and pharynx, ovary, pancreas, stomach, testes, thyroid, and uterus as well as Hodgkin's disease, leukemia, melanoma of the skin, Non-Hodgkin's Lymphoma, and other more rare types of cancer. There was no specific pattern or geographic concentration of any one cancer type within

this neighborhood. Also, the years of diagnosis for these individuals varied throughout the 24 years reviewed, indicating no apparent trend or pattern in the time of diagnosis.

The majority of cancer types diagnosed among residents of the neighborhood surrounding the site are predominantly associated with nonenvironmental factors such as family history, smoking, diet, and other lifestyle behaviors. Because the MCR collects some information related to risk factors (e.g., smoking history) for individuals diagnosed with cancer, these data were reviewed to better characterize the incidence patterns of cancers in these areas of Norton and Attleboro. This included a review of age at diagnosis, gender, smoking history, and occupation.

Age is an important risk factor in many cancers. Different cancers occur with different frequencies among the various age groups, and most cancer types occur more frequently in older populations (i.e., age 50 and over). The average age at diagnosis among individuals diagnosed with any type of cancer within a 1-mile radius of the Shpack Landfill was approximately 60 and the majority of individuals (78%) were age 50 or older when they were diagnosed. Review of the age and gender pattern among these individuals indicates that the incidence of cancers in this area is consistent with established prevalence patterns of disease in the general population.

Because cigarette smoking is also an important risk factor in the development of several cancer types, including cancers of the bladder, colon/rectum, esophagus, kidney, lung and bronchus, oral and pharynx, pancreas, and stomach, smoking history was reviewed for each individual living within 1-mile of the Shpack Landfill who was diagnosed with a smoking-related cancer. Eighty-seven individuals were diagnosed with a smoking-related cancer. Of these 87 individuals, smoking history was reported to the MCR for 74 individuals while smoking history was unknown for 13 individuals. Forty-six (62%) of the 74 individuals with a reported smoking history were current or former smokers at the time of diagnosis. Twenty-eight of the 74 individuals were reported to the MCR as nonsmokers. Therefore, it is likely that smoking played some role in the development of cancer among some residents of the neighborhood surrounding the site. Further, an evaluation of the geographic distribution of place of residence for the nonsmokers did not demonstrate any unusual spatial patterns.

Some occupational exposures, such as jobs involving contact with chemicals, have been associated with an increased risk for developing certain types of cancer. Therefore, occupational

information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with cancer within 1 mile of the Shpack Landfill, to determine the role that occupational factors may have played in the development of cancer. It should be restated, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individuals. Further, these data are often incomplete as occupation can be reported as unknown, at home, or retired. From 1982 to the present, approximately 26 individuals (12%) diagnosed with cancer living within 1 mile of the site worked in jobs that could be associated with an increased risk for developing cancer. Ninety-four individuals (42%) had an occupation reported as unknown, at home, or retired. Although the information reviewed suggests the possibility that occupational exposures may have contributed to the development of cancer among some individuals, it is difficult to determine what role, if any, occupational exposures played in the incidence of cancer in the Shpack Landfill neighborhood.

IX. DISCUSSION

Six of the 13 cancer types evaluated in this report occurred approximately at or near expected rates in Attleboro and Norton and their individual census tracts during the four time periods evaluated: 1982–1987, 1988–1993, 1994–1999, and 2000–2002. These cancer types included bone, kidney, leukemia, multiple myeloma, non-Hodgkin's lymphoma, and pancreas. Across all four time periods, the incidence of these cancer types was at or near what would be expected. Six of the 13 cancer types were statistically significantly elevated during only one of the four time periods evaluated, in either Attleboro or Norton or an individual census tract within either community, while only one cancer type, lung and bronchus, was statistically significantly elevated over two consecutive time periods. This occurred in Attleboro.

A city-wide statistically significant elevation of lung and bronchus cancer occurred among females during 1988–1993 and among males during1994–1999 in Attleboro. Individual census tracts in Attleboro that experienced a statistically significant elevation of lung and bronchus cancer during these time periods included: CT 6311 (females, 1988–1993); CT 6314 (males and females combined and females only, 1988–1993; males only, 1994–1999); and CT 6318 (males and females combined, 1988–1993; females only, 1988-1993). During 1994–1999, the rate of

lung and bronchus cancer was also statistically significantly elevated among males and females combined and females only in Norton CT 6111. In general, review of available risk factor information for individuals diagnosed with lung and bronchus cancer in these geographic areas did not suggest any patterns or trends that were inconsistent with established incidence patterns. Smoking history information collected by the MCR for those individuals diagnosed with lung and bronchus cancer revealed that tobacco use likely played a role in the incidence of lung and bronchus cancer among residents of Attleboro and Norton. Occupational exposures thought to be associated with lung and bronchus cancer may have been possible for a small percentage of individuals in Attleboro and Norton diagnosed with this cancer type. As stated earlier, the MDPH conducted a radon survey in Attleboro as part of its July 2001 investigation (MDPH 2001). Results of the radon survey in Attleboro showed that radon levels measured both in the homes or former homes of female lung cancer cases and in the randomly selected households were below the USEPA recommended remediation level of 4 picocuries per liter. However, the median radon concentration measured in case homes (2.4 picocuries per liter) was higher than the median radon concentration measured in the randomly selected control homes (1.9 picocuries per liter).

In Attleboro, although the city as a whole and three of its eight CTs had statistically significant elevations in lung and bronchus cancer during part of the 21-year time period evaluated, no consistent trends were noted. Among males and females combined, lung and bronchus cancer occurred city-wide less often than expected during 1982–1987, more often than expected during 1988–1993, about as expected during 1994–1999, and more often than expected during 2000–2002. For CT 6314, where elevations occurred over three time periods (two being statistically significant), an elevation occurred first in males (1982–1987), then in males and females (1988–1993), then in males (1994–1999), and in the last time period evaluated (2000–2002), the number of lung and bronchus cancer cases occurred about as expected in males and females. While an elevation persisted in males in CT 6314 during the first three time periods, statistical significance was limited to the third time period.

Although the incidence of bladder cancer was statistically significantly elevated among females in Attleboro during the most recent time period of 2000–2002, it was not elevated in the previous three time periods evaluated. In CT 6313 during 2000–2002, seven diagnoses were reported in

males and females combined compared to approximately two diagnoses expected; this elevation was statistically significant. The incidence of bladder cancer in Attleboro females was lower than expected during 1982–1987 and 1994–1999 and about as expected during 1988–1993.

With one exception, breast cancer occurred about as expected in Attleboro as a whole and its CTs. Breast cancer was statistically significantly elevated among females in Attleboro CT 6317 during 1988–1993; however, no consistent trend over time was seen, with the incidence being as expected or less than expected during the other three time periods evaluated. The age distribution among females diagnosed with breast cancer in this census tract was consistent with established trends for breast cancer. Breast cancer screening information was similar to patterns observed statewide, however slightly more women in this CT were diagnosed at the earliest stage compared to the state as a whole. The distribution of breast cancer diagnoses varied geographically within the CT.

For the city of Attleboro as a whole, the incidence of Hodgkin's disease was about as expected for two of the four time periods evaluated and higher than expected for the middle two time periods evaluated, although not statistically significantly elevated. Two CTs, 6312 & 6315 combined, however, had a statistically significant elevation in Hodgkin's disease for one of the four time periods, 1988-1993. In these CTs, the age and gender distributions and the histological subtypes were consistent with what would be expected based upon the epidemiological literature. As stated earlier, a chronic infectious process has been associated with Hodgkin's disease (Mueller 1999), most notably infection with the Epstein-Barr virus. Although it is beyond the scope of this evaluation to determine if any residents of Attleboro diagnosed with Hodgkin's disease have a history of infection with the EBV, available age and histology data do not suggest an unusual pattern in the occurrence of Hodgkin's disease.

The incidence of liver cancer was statistically significantly elevated among males in Attleboro during the most recent time period of 2000–2002 but was not elevated during 1982–1987 or 1994–1999. During 1988–1993, although more diagnoses of liver cancer occurred among males than expected, the difference was not statistically significant.

A city-wide statistically significant elevation of thyroid cancer was observed among males in Attleboro during 1988–1993 (7 diagnoses observed versus 2.6 expected). However, no time

trends in thyroid cancer incidence were observed because fewer cases of thyroid cancer occurred than expected during 1982–1987, more cases occurred than expected during 1988–1993, about as many occurred as expected during 1994–1999, and more cases occurred than expected during 2000–2002.

The majority of the 13 cancer types diagnosed among residents of Attleboro CT 6317 and Norton CT 6112 (where the Shpack Landfill is located) occurred either near or below the number of expected cases during the four time periods evaluated. As noted earlier, breast cancer was statistically significantly elevated among females in Attleboro CT 6317 during 1988–1993, but occurred at the expected rate during 1982–1987, lower than the expected rate during 1994–1999, and lower than the expected rate during 2000–2002. Rates of breast cancer among females in Norton CT 6112 were either near or below expected rates during the four time periods evaluated.

While some elevations occurred in four different cancer types in Norton CT 6112 throughout the 21-year time period, with one exception, the elevations were not statistically significant and did not persist over time. The number of observed cases of brain and CNS cancer was statistically significantly greater than expected during the last time period evaluated; however, in the earlier time periods, the incidence of this type of cancer was about as expected. In Attleboro CT 6317, the incidence of brain and CNS cancer was about as expected during the four time periods evaluated. No apparent concentrations of any specific cancer type were observed in the vicinity of the Shpack Landfill.

According to American Cancer Society statistics, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of three people develop cancer in their lifetime, but cancer will affect three out of every four families. For this reason, cancer diagnoses often appear to occur in "clusters," and it is understandable that someone may perceive that there are an unusually high number of cancer cases in their surrounding neighborhood or town. Upon close examination, many of these "clusters" are not unusual increases as first thought, but are related to such factors as local population density, variations in reporting, or chance fluctuations in occurrence. In other instances, the "cluster" in question includes a high concentration of individuals who possess related behaviors or risk factors for cancer. Some, however, are unusual; that is, they represent a true excess of cancer in a

workplace, a community, or among a subgroup of people. A suspected cluster is more likely to be a true cancer cluster if it involves a large number of cases of one type of cancer diagnosed in a relatively short time period rather than several different types diagnosed over a long period of time (i.e., 20 years), a rare type of cancer rather than common types, and/or a large number of cases diagnosed among individuals in age groups not usually affected by that cancer type. These types of clusters may warrant further public health investigation.

Over the past 40 years, the dramatic rise in the number of cancer cases largely reflects an increase in the population, particularly in the older age groups. The most commonly diagnosed cancer types for adult males include cancers of the prostate, lung and bronchus, and colon. Breast, lung and bronchus, and colon cancer are the most common cancer types diagnosed among women.

Understanding that cancer is not one disease, but a group of diseases is also very important. Research has shown that there are more than 100 different types of cancer, each with different causative (or risk) factors. In addition, cancers of a certain tissue type in one organ may have a number of causes. Cancer may also be caused by one or several factors acting over time. For example, tobacco use has been linked to lung and bronchus, bladder, pancreatic, and kidney cancers. Other factors related to cancer may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important risk factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer-causing). Environmental contaminants have also been associated with certain types of cancer. In addition, most cancers have a long latency period or period of development that can range from 10 to 30 years and, in some cases, may be more than 40 to 50 years. To provide a better understanding of factors that are related to the development of various cancer types, Appendix C contains a summary of additional information for cancer types that were evaluated in Attleboro and Norton.

The information evaluated in this report does not indicate an atypical pattern of any one cancer type in either Attleboro or Norton. No specific patterns with respect to place of residence at diagnosis or date of diagnosis emerged that would suggest an unusual geographic pattern or common factor (environmental or nonenvironmental) among residents of these communities

diagnosed with cancer. Review of available risk factor information suggests that tobacco use likely played an important role in the incidence of a number of cancers diagnosed among the residents.

In general, the geographic distributions of residences at the time of diagnosis were consistent with what would be expected based on the population distribution and areas of higher population density in Attleboro and Norton. For example, in Attleboro, the majority of diagnoses for each cancer type tended to be located in and around the center of the city and in the southwestern corner of the city, where the population and housing density are greatest. Where the distribution of diagnoses less closely matched the population pattern, diagnoses appeared fairly evenly distributed throughout the towns and did not appear concentrated in any one area of Norton or Attleboro.

X. ATSDR CHILD HEALTH CONSIDERATIONS

ATSDR and MDPH recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at a greater risk than adults from certain kinds of exposure to hazardous substances emitted from waste sites. They are more likely to be exposed because they play outdoors and because they often bring food into contaminated areas. Because of their smaller stature, they might breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of contaminant exposure per body weight. The developing body systems of children can sustain permanent damage if certain toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care. Review of specific diagnosis information (i.e., primary cancer type, histology) and geographic distribution for each child (i.e., ages 0-19) diagnosed with cancer did not suggest that an atypical pattern of cancer occurred among children in Attleboro and Norton.

XI. LIMITATIONS

This health consultation is an investigation that analyzes descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers is unusual. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further analyses are needed. Inherent limitations in this type of analysis and the available data make it difficult at best to determine causal relationships or synergistic roles that may have played a part in the development of individual cancers in these communities. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the diseases. Behavioral factors such as tobacco use, diet, and alcohol consumption are considered the most important risk factors for a number of cancers. Other factors associated with cancer are socioeconomic status, reproductive factors, exposure to infectious agents (i.e., viruses) and heredity/genetics. It is beyond the scope of this report to determine the causal relationship of these factors and the development of cancer or other health outcomes in the CTs of Norton and Attleboro.

XII. CONCLUSIONS

- Of the 13 cancer types evaluated in the city of Attleboro and the town of Norton during four time periods between 1982–2002, the majority occurred approximately at or near the expected rate. The exceptions included statistically significant elevations in the incidence of bladder cancer among females in Attleboro during 2000–2002; liver cancer among males in Attleboro during 2000–2002; lung and bronchus cancer among females in Attleboro during 1988–1993 and among males in Attleboro during 1994–1999, and; thyroid cancer among males in Attleboro during 1988–1993.

- Some census tracts demonstrated statistically significant elevations in the incidence of bladder cancer, brain and CNS cancer, breast cancer, Hodgkin's disease, and lung and bronchus cancer. Further examination of geographic and temporal factors did not suggest a common environmental factor related to cancer diagnoses among residents.

- Analysis of available risk factor information for individuals diagnosed with cancer (e.g., age, gender, smoking history, and occupation) suggests that, for the most part, the trends observed in Attleboro and Norton are similar to those seen in the general population. This information suggests that smoking likely played some role in the incidence of some cancer types in Attleboro and Norton.

- A review of the MCR data for residents of Norton and Attleboro living within about 1-mile of the Shpack Landfill did not reveal any unusual patterns with respect to any one cancer type or geographic or temporal patterns. Further, an evaluation of the geographic distribution of place of residence for the nonsmokers did not demonstrate any unusual spatial patterns.

ATSDR requires that one of five conclusion categories be used to summarize findings of a health consultation. These categories are as follows: (1) Urgent Public Health Hazard; (2) Public Health Hazard; (3) Indeterminate Public Health Hazard; (4) No Apparent Public Health Hazard; (5) No Public Health Hazard. A category is selected from site-specific conditions such as the degree of public health hazard based on the presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns. Evaluation of available environmental data for the Shpack Landfill will be undertaken as a separate report. The pattern of cancer described in this health consultation will be examined in relation to potential exposure pathways to site-related contaminants upon completion of the PHA. Information to date indicates that contaminants of concern include metals, volatile organic compounds (VOCs), and radioactive compounds, principally radium and uranium, and that possible exposure pathways of community concern include direct contact with landfill contaminants (Metcalf & Eddy 2004). ATSDR would classify the Shpack Landfill in the past, present, and future as posing an Indeterminate Public Health Hazard pending further analysis of available environmental data.

XIII. RECOMMENDATIONS

- Opportunities for exposure to contaminants at the Shpack Landfill will be characterized in the public health assessment. The PHA will include an evaluation of data contained in this report.

- Upon request of the local health departments and/or community representatives, MDPH's Environmental Health Education and Outreach Program will prepare educational materials relative to the cancers of concern.

XIV. PUBLIC HEALTH ACTION PLAN

The Public Health Action Plan for Attleboro and Norton, Massachusetts, contains recommendations for actions to be taken at and in the vicinity of the Shpack Landfill. The purpose of the Public Health Action Plan is to ensure that this health consultation not only identifies potential public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the ATSDR/MDPH to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by ATSDR/MDPH are as follows:

- The MDPH will continue to monitor the incidence of all cancer types in the city of Attleboro and the town of Norton through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

- Under a cooperative agreement with ATSDR, the MDPH/CEH will evaluate available environmental data for the Shpack Landfill and potential exposure pathways to contaminants on the site.

- The MDPH/CEH will forward a copy of this health consultation to the Attleboro and Norton Boards of Health for consideration in the planning of community prevention and intervention strategies to reduce cancer risk among residents (e.g., tobacco cessation programs). In addition, the MDPH Environmental Health Education Program will

contact the Boards of Health to offer assistance with follow-up educational and outreach activities.

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PREPARER

This document was prepared by the Center for Environmental Health of the Massachusetts Department of Public Health. If you have any questions about this document, please contact Suzanne K. Condon, Associate Commissioner of CEH/MDPH at 250 Washington Street, 7th Floor, Boston, MA 02108.

CERTIFICATION

The Health Consultation, *Evaluation of Cancer Incidence in Census Tracts of Norton and Attleboro, Massachusetts: 1982–2002, Shpack Landfill, MAD 980503973*, was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated. Editorial review was completed by the cooperative agreement partner.

_____

Technical Project Officer, CAT, SPAB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this Health Consultation and concurs with its findings.

_____

Team Lead, CAT, SPAB, DHAC

FIGURES

Figure 1
Location of Census Tracts
Attleboro and Norton, Massachusetts

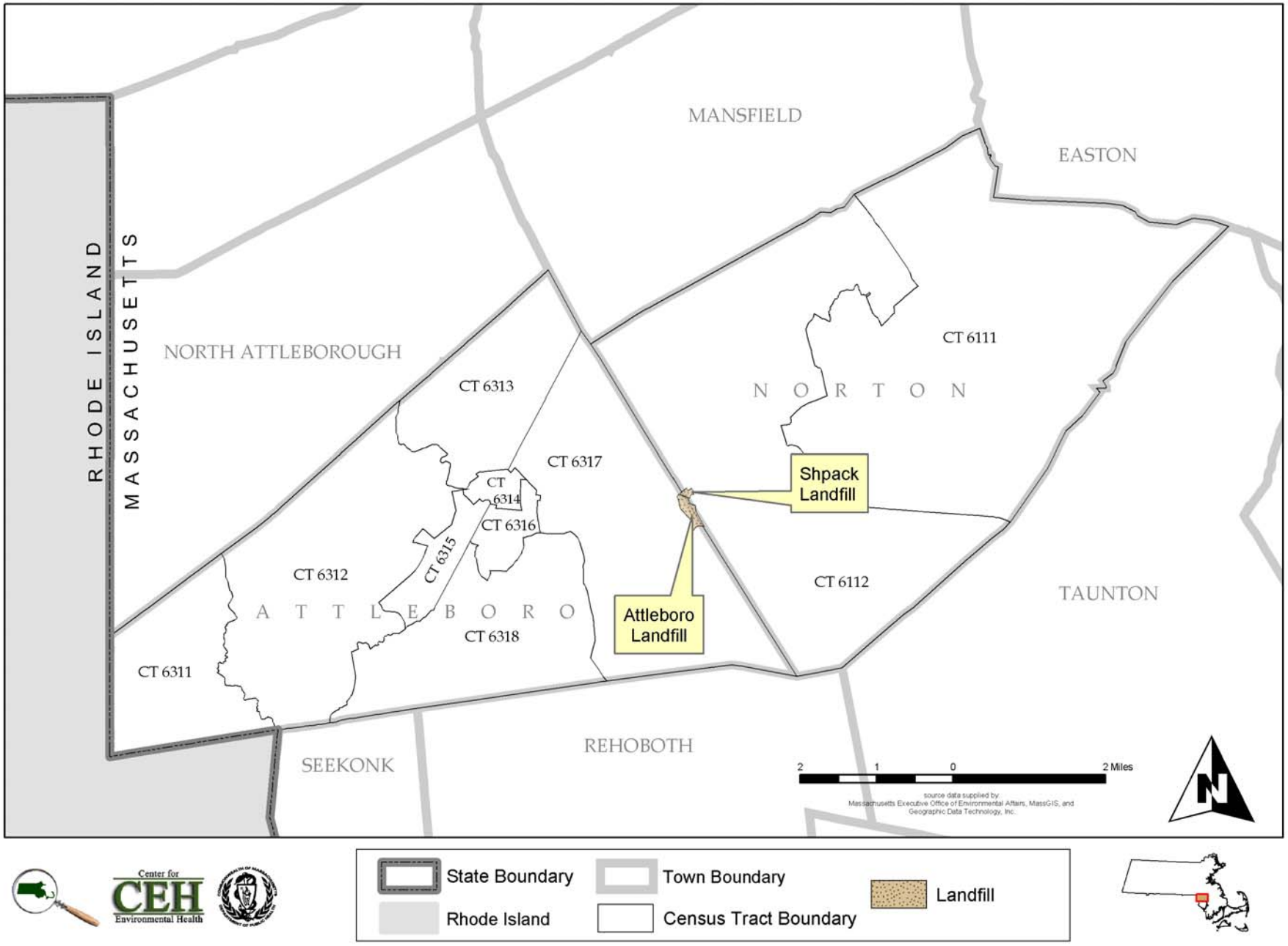
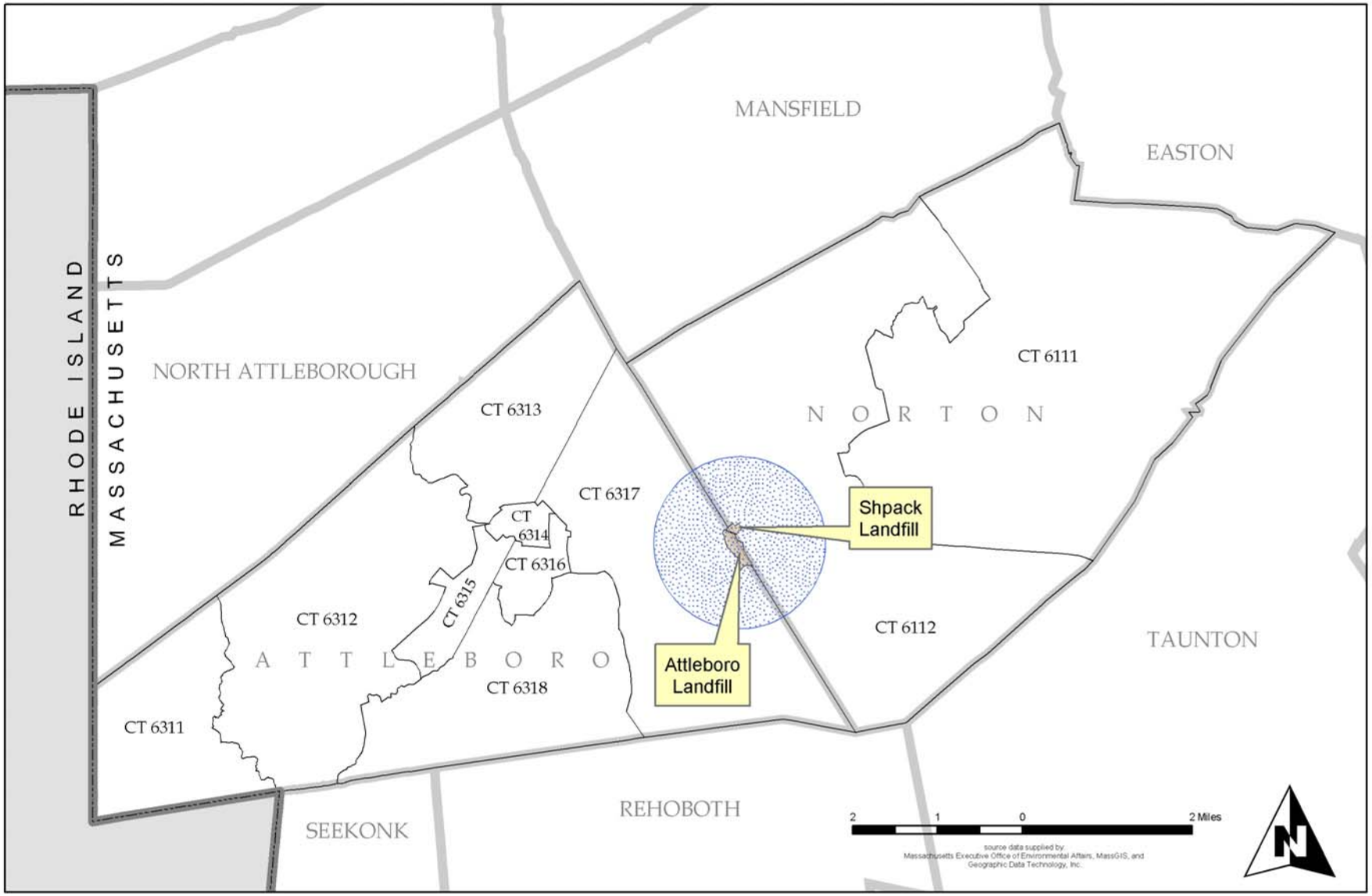


Figure 2
Delineation of Shpack Landfill Neighborhood
Attleboro and Norton, Massachusetts



TABLES

TABLE 1a
Bladder Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	7.3	NC	NC -- NC	0	5.5	NC	NC -- NC	0	1.9	NC	NC -- NC
6312 & 6315	6	8.5	71	26 -- 154	4	6.0	NC	NC -- NC	2	2.4	NC	NC -- NC
6313	3	4.9	NC	NC -- NC	3	3.5	NC	NC -- NC	0	1.4	NC	NC -- NC
6314	2	2.3	NC	NC -- NC	1	1.7	NC	NC -- NC	1	0.6	NC	NC -- NC
6316	1	3.9	NC	NC -- NC	1	2.6	NC	NC -- NC	0	1.3	NC	NC -- NC
6317	5	5.6	89	29 -- 208	4	4.0	NC	NC -- NC	1	1.6	NC	NC -- NC
6318	3	4.6	NC	NC -- NC	1	3.3	NC	NC -- NC	2	1.3	NC	NC -- NC
City Total	20	37.2	54	* 33 -- 83	14	26.6	53	* 29 -- 88	6	10.6	57	21 -- 123

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1b
Bladder Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	7	7.1	98	39 -- 202	5	5.3	94	30 -- 219	2	1.8	NC	NC -- NC
6312 & 6315	4	8.5	NC	NC -- NC	3	6.1	NC	NC -- NC	1	2.4	NC	NC -- NC
6313	2	4.9	NC	NC -- NC	2	3.5	NC	NC -- NC	0	1.4	NC	NC -- NC
6314	3	1.9	NC	NC -- NC	1	1.4	NC	NC -- NC	2	0.5	NC	NC -- NC
6316	4	3.2	NC	NC -- NC	3	2.1	NC	NC -- NC	1	1.1	NC	NC -- NC
6317	8	5.2	154	67 -- 304	5	3.7	135	44 -- 315	3	1.5	NC	NC -- NC
6318	7	5.5	127	51 -- 261	6	3.8	157	57 -- 342	1	1.7	NC	NC -- NC
City Total	35	36.3	96	67 -- 134	25	26.0	96	62 -- 142	10	10.4	97	46 -- 178

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1c
Bladder Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	7	8.0	88	35 -- 181	5	5.9	85	27 -- 199	2	2.1	NC	NC -- NC
6312 & 6315	8	8.4	95	41 -- 187	8	6.1	132	57 -- 260	0	2.4	NC	NC -- NC
6313	3	4.7	NC	NC -- NC	3	3.3	NC	NC -- NC	0	1.5	NC	NC -- NC
6314	1	1.8	NC	NC -- NC	0	1.3	NC	NC -- NC	1	0.5	NC	NC -- NC
6316	2	3.3	NC	NC -- NC	1	2.3	NC	NC -- NC	1	1.0	NC	NC -- NC
6317	2	6.3	NC	NC -- NC	2	4.2	NC	NC -- NC	0	2.1	NC	NC -- NC
6318	7	5.8	120	48 -- 247	4	3.9	NC	NC -- NC	3	1.9	NC	NC -- NC
City Total	30	38.3	78	53 -- 112	23	26.9	86	54 -- 128	7	11.4	61	25 -- 126

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1d
Bladder Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	3.7	NC	NC -- NC	0	2.7	NC	NC -- NC	2	1.0	NC	NC -- NC
6312 & 6315	5	3.7	135	44 -- 315	4	2.7	NC	NC -- NC	1	1.0	NC	NC -- NC
6313	7	2.0	351	* 141 -- 724	4	1.4	NC	NC -- NC	3	0.6	NC	NC -- NC
6314	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
6316	1	1.4	NC	NC -- NC	0	1.0	NC	NC -- NC	1	0.4	NC	NC -- NC
6317	4	3.1	NC	NC -- NC	2	2.0	NC	NC -- NC	2	1.0	NC	NC -- NC
6318	3	2.6	NC	NC -- NC	1	1.7	NC	NC -- NC	2	0.9	NC	NC -- NC
City Total	22	17.2	128	80 -- 194	11	12.1	91	45 -- 163	11	5.1	216	* 108 -- 386

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2a
Bone Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.2	NC	NC -- NC
6312 & 6315	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6313	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6314	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6317	1	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6318	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
City Total	2	2.0	NC	NC -- NC	1	1.1	NC	NC -- NC	1	1.0	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2b
Bone Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6312 & 6315	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6313	1	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6314	2	0.1	NC	NC -- NC	2	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6317	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6318	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total	3	2.2	NC	NC -- NC	3	1.2	NC	NC -- NC	0	1.1	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2c
Bone Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.2	NC	NC -- NC
6312 & 6315	1	0.5	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
6313	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6314	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6317	1	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6318	1	0.4	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total	4	2.3	NC	NC -- NC	3	1.2	NC	NC -- NC	1	1.1	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2d
Bone Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6312 & 6315	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6313	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6314	0	0.1	NC	NC -- NC	0	0.0	NC	NC -- NC	0	0.0	NC	NC -- NC
6316	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.0	NC	NC -- NC
6317	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6318	2	0.2	NC	NC -- NC	2	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
City Total	2	1.0	NC	NC -- NC	2	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3a
Brain & Central Nervous System (CNS) Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	3.2	NC	NC -- NC	0	1.6	NC	NC -- NC	1	1.6	NC	NC -- NC
6312 & 6315	3	3.7	NC	NC -- NC	1	1.8	NC	NC -- NC	2	1.9	NC	NC -- NC
6313	1	2.1	NC	NC -- NC	1	1.0	NC	NC -- NC	0	1.1	NC	NC -- NC
6314	1	1.1	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	0	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.9	NC	NC -- NC
6317	0	2.5	NC	NC -- NC	0	1.2	NC	NC -- NC	0	1.3	NC	NC -- NC
6318	0	2.2	NC	NC -- NC	0	1.1	NC	NC -- NC	0	1.1	NC	NC -- NC
City Total	6	16.7	36	* 13 -- 78	3	8.2	NC	NC -- NC	3	8.5	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3b
Brain & Central Nervous System (CNS) Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	4	3.7	NC	NC -- NC	3	1.8	NC	NC -- NC	1	1.8	NC	NC -- NC
6312 & 6315	5	4.3	115	37 -- 269	4	2.1	NC	NC -- NC	1	2.2	NC	NC -- NC
6313	1	2.5	NC	NC -- NC	1	1.2	NC	NC -- NC	0	1.3	NC	NC -- NC
6314	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	1	1.9	NC	NC -- NC	0	0.9	NC	NC -- NC	1	1.0	NC	NC -- NC
6317	3	2.8	NC	NC -- NC	1	1.4	NC	NC -- NC	2	1.4	NC	NC -- NC
6318	0	3.1	NC	NC -- NC	0	1.5	NC	NC -- NC	0	1.6	NC	NC -- NC
City Total	14	19.5	72	39 -- 120	9	9.5	95	43 -- 180	5	10.0	50	16 -- 117

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3c
Brain & Central Nervous System (CNS) Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	3.4	NC	NC -- NC	0	1.9	NC	NC -- NC	0	1.5	NC	NC -- NC
6312 & 6315	3	3.7	NC	NC -- NC	2	2.0	NC	NC -- NC	1	1.7	NC	NC -- NC
6313	2	2.1	NC	NC -- NC	2	1.1	NC	NC -- NC	0	1.0	NC	NC -- NC
6314	2	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC
6316	1	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC
6317	0	2.6	NC	NC -- NC	0	1.4	NC	NC -- NC	0	1.2	NC	NC -- NC
6318	2	2.8	NC	NC -- NC	1	1.5	NC	NC -- NC	1	1.3	NC	NC -- NC
City Total	10	17.1	58	28 -- 108	7	9.3	75	30 -- 155	3	7.8	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3d
Brain & Cental Nervous System Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	1.9	NC	NC -- NC	2	1.0	NC	NC -- NC	1	0.8	NC	NC -- NC
6312 & 6315	4	2.0	NC	NC -- NC	3	1.1	NC	NC -- NC	1	0.9	NC	NC -- NC
6313	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
6314	0	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
6316	2	0.9	NC	NC -- NC	2	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
6317	3	1.5	NC	NC -- NC	2	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
6318	2	1.5	NC	NC -- NC	0	0.8	NC	NC -- NC	2	0.7	NC	NC -- NC
City Total[†]	15	9.4	159	89 -- 262	10	5.0	200	96 -- 368	5	4.4	113	36 -- 263

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 4a
Breast Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	15	25.6	59	* 33 -- 97	0	0.1	NC	NC -- NC	15	25.5	59	* 33 -- 97
6312 & 6315	40	31.2	128	92 -- 174	1	0.2	NC	NC -- NC	39	31.1	126	89 -- 172
6313	18	17.9	100	59 -- 159	0	0.1	NC	NC -- NC	18	17.8	101	60 -- 159
6314	13	8.2	158	84 -- 270	0	0.0	NC	NC -- NC	13	8.2	159	84 -- 272
6316	13	15.0	87	46 -- 148	0	0.1	NC	NC -- NC	13	14.9	87	46 -- 149
6317	19	20.1	94	57 -- 147	0	0.1	NC	NC -- NC	19	20.0	95	57 -- 148
6318	17	17.5	97	57 -- 156	0	0.1	NC	NC -- NC	17	17.4	98	57 -- 156
City Total†	137	135.6	101	85 -- 119	1	0.7	NC	NC -- NC	136	134.9	101	85 -- 119

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 4b
Breast Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	30	30.4	99	67 -- 141	0	0.2	NC	NC -- NC	30	30.2	99	67 -- 142
6312 & 6315	48	37.5	128	94 -- 170	0	0.3	NC	NC -- NC	48	37.2	129	95 -- 171
6313	18	22.0	82	48 -- 129	0	0.2	NC	NC -- NC	18	21.9	82	49 -- 130
6314	13	8.6	151	80 -- 258	0	0.1	NC	NC -- NC	13	8.6	152	81 -- 260
6316	21	15.6	134	83 -- 205	0	0.1	NC	NC -- NC	21	15.5	135	84 -- 207
6317	38	23.7	160	* 113 -- 220	0	0.2	NC	NC -- NC	38	23.5	162	* 114 -- 222
6318	17	26.5	64	37 -- 103	0	0.2	NC	NC -- NC	17	26.4	64	38 -- 103
City Total†	186	164.4	113	97 -- 131	0	1.2	NC	NC -- NC	186	163.3	114	98 -- 132

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 4c
Breast Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	29	35.8	81	54 -- 116	0	0.3	NC	NC -- NC	29	35.5	82	55 -- 117
6312 & 6315	32	40.5	79	54 -- 111	0	0.3	NC	NC -- NC	32	40.2	80	54 -- 112
6313	25	23.5	106	69 -- 157	1	0.2	NC	NC -- NC	24	23.3	103	66 -- 153
6314	11	8.8	125	62 -- 223	0	0.1	NC	NC -- NC	11	8.7	126	63 -- 225
6316	11	16.2	68	34 -- 121	0	0.1	NC	NC -- NC	11	16.1	68	34 -- 122
6317	20	30.2	66	40 -- 102	0	0.2	NC	NC -- NC	20	30.0	67	41 -- 103
6318	30	30.4	99	67 -- 141	0	0.2	NC	NC -- NC	30	30.2	99	67 -- 142
City Total	158	185.4	85	72 -- 100	1	1.4	NC	NC -- NC	157	184.0	85	72 -- 100

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 4d
Breast Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	22	19.5	113	71 -- 171	1	0.2	NC	NC -- NC	21	19.4	108	67 -- 166
6312 & 6315	17	21.4	80	46 -- 127	0	0.2	NC	NC -- NC	17	21.2	80	47 -- 128
6313	11	12.0	91	46 -- 164	0	0.1	NC	NC -- NC	11	12.0	92	46 -- 165
6314	4	4.5	NC	NC -- NC	0	0.0	NC	NC -- NC	4	4.5	NC	NC -- NC
6316	6	8.3	73	27 -- 158	0	0.1	NC	NC -- NC	6	8.2	73	27 -- 159
6317	14	17.1	82	45 -- 137	0	0.1	NC	NC -- NC	14	17.0	82	45 -- 138
6318	16	16.2	99	56 -- 160	0	0.1	NC	NC -- NC	16	16.1	99	57 -- 161
City Total[†]	92	99.1	93	75 -- 114	1	0.7	NC	NC -- NC	91	98.4	92	74 -- 114

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 5a
Hodgkin's Disease Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
6312 & 6315	3	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.8	NC	NC -- NC
6313	3	1.0	NC	NC -- NC	2	0.5	NC	NC -- NC	1	0.5	NC	NC -- NC
6314	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC
6316	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
6317	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
6318	0	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
City Total	7	7.9	89	36 -- 183	3	4.3	NC	NC -- NC	4	3.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 5b
Hodgkin's Disease Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	1.6	NC	NC -- NC	0	0.9	NC	NC -- NC	3	0.7	NC	NC -- NC
6312 & 6315	6	1.9	320	* 117 -- 696	4	1.0	NC	NC -- NC	2	0.9	NC	NC -- NC
6313	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
6314	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.3	NC	NC -- NC
6316	0	1.0	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
6317	1	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
6318	2	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
City Total	12	9.0	134	69 -- 234	5	4.8	105	34 -- 245	7	4.2	167	67 -- 344

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 5c
Hodgkin's Disease Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
6312 & 6315	3	1.9	NC	NC -- NC	2	1.0	NC	NC -- NC	1	0.8	NC	NC -- NC
6313	3	1.1	NC	NC -- NC	3	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
6314	1	0.6	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
6316	1	0.9	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
6317	2	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
6318	1	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.7	NC	NC -- NC
City Total	13	8.8	148	79 -- 253	10	4.8	207	99 -- 380	3	4.0	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 5d
Hodgkin's Disease Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.4	NC	NC -- NC
6312 & 6315	3	0.9	NC	NC -- NC	3	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
6313	0	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
6314	1	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.1	NC	NC -- NC
6316	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6317	2	0.6	NC	NC -- NC	1	0.3	NC	NC -- NC	1	0.3	NC	NC -- NC
6318	0	0.7	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC
City Total	6	4.1	146	53 -- 318	4	2.2	NC	NC -- NC	2	1.9	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 6a
Kidney Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	3.3	NC	NC -- NC	0	2.0	NC	NC -- NC	2	1.3	NC	NC -- NC
6312 & 6315	3	3.9	NC	NC -- NC	0	2.2	NC	NC -- NC	3	1.6	NC	NC -- NC
6313	2	2.2	NC	NC -- NC	1	1.3	NC	NC -- NC	1	0.9	NC	NC -- NC
6314	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
6316	0	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
6317	1	2.5	NC	NC -- NC	0	1.5	NC	NC -- NC	1	1.0	NC	NC -- NC
6318	2	2.1	NC	NC -- NC	1	1.2	NC	NC -- NC	1	0.9	NC	NC -- NC
City Total	10	16.8	60	29 -- 110	2	9.8	NC	NC -- NC	8	7.0	115	49 -- 226

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 6b
Kidney Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	5	4.7	106	34 -- 247	4	3.0	NC	NC -- NC	1	1.7	NC	NC -- NC
6312 & 6315	9	5.5	164	75 -- 311	5	3.3	149	48 -- 349	4	2.1	NC	NC -- NC
6313	4	3.1	NC	NC -- NC	2	1.9	NC	NC -- NC	2	1.3	NC	NC -- NC
6314	1	1.3	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.5	NC	NC -- NC
6316	1	2.1	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC
6317	3	3.4	NC	NC -- NC	1	2.1	NC	NC -- NC	2	1.3	NC	NC -- NC
6318	3	3.7	NC	NC -- NC	1	2.1	NC	NC -- NC	2	1.5	NC	NC -- NC
City Total	26	23.8	109	71 -- 160	14	14.5	97	53 -- 162	12	9.3	129	66 -- 225

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 6c
Kidney Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	5.7	NC	NC -- NC	2	3.5	NC	NC -- NC	1	2.2	NC	NC -- NC
6312 & 6315	6	6.2	97	35 -- 211	3	3.8	NC	NC -- NC	3	2.4	NC	NC -- NC
6313	3	3.4	NC	NC -- NC	3	2.0	NC	NC -- NC	0	1.4	NC	NC -- NC
6314	2	1.4	NC	NC -- NC	2	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	1	2.4	NC	NC -- NC	1	1.4	NC	NC -- NC	0	1.0	NC	NC -- NC
6317	3	4.4	NC	NC -- NC	2	2.5	NC	NC -- NC	1	1.8	NC	NC -- NC
6318	2	4.3	NC	NC -- NC	1	2.5	NC	NC -- NC	1	1.8	NC	NC -- NC
City Total	20	27.8	72	44 -- 111	14	16.6	84	46 -- 141	6	11.1	54	20 -- 117

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 6d
Kidney Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	6	3.5	170	62 -- 370	4	2.2	NC	NC -- NC	2	1.4	NC	NC -- NC
6312 & 6315	2	3.7	NC	NC -- NC	1	2.3	NC	NC -- NC	1	1.5	NC	NC -- NC
6313	3	2.0	NC	NC -- NC	2	1.2	NC	NC -- NC	1	0.8	NC	NC -- NC
6314	1	0.8	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC
6316	2	1.4	NC	NC -- NC	2	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC
6317	2	2.8	NC	NC -- NC	1	1.6	NC	NC -- NC	1	1.2	NC	NC -- NC
6318	2	2.6	NC	NC -- NC	1	1.5	NC	NC -- NC	1	1.1	NC	NC -- NC
City Total[†]	21	17.0	124	76 -- 189	14	10.1	139	76 -- 233	7	6.9	101	41 -- 209

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 7a
Leukemia Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	3.2	NC	NC -- NC	0	1.9	NC	NC -- NC	2	1.3	NC	NC -- NC
6312 & 6315	1	3.9	NC	NC -- NC	0	2.1	NC	NC -- NC	1	1.7	NC	NC -- NC
6313	3	2.3	NC	NC -- NC	2	1.2	NC	NC -- NC	1	1.0	NC	NC -- NC
6314	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	1	1.9	NC	NC -- NC	1	1.0	NC	NC -- NC	0	0.9	NC	NC -- NC
6317	2	2.7	NC	NC -- NC	1	1.5	NC	NC -- NC	1	1.2	NC	NC -- NC
6318	2	2.3	NC	NC -- NC	2	1.3	NC	NC -- NC	0	1.0	NC	NC -- NC
City Total†	12	17.2	70	36 -- 122	6	9.5	63	23 -- 137	6	7.7	78	28 -- 169

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 7b
Leukemia Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	3.4	NC	NC -- NC	1	2.0	NC	NC -- NC	2	1.4	NC	NC -- NC
6312 & 6315	4	4.1	NC	NC -- NC	3	2.3	NC	NC -- NC	1	1.8	NC	NC -- NC
6313	3	2.4	NC	NC -- NC	2	1.3	NC	NC -- NC	1	1.1	NC	NC -- NC
6314	3	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	2	0.4	NC	NC -- NC
6316	1	1.8	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.9	NC	NC -- NC
6317	3	2.6	NC	NC -- NC	2	1.5	NC	NC -- NC	1	1.2	NC	NC -- NC
6318	4	2.9	NC	NC -- NC	3	1.6	NC	NC -- NC	1	1.3	NC	NC -- NC
City Total	21	18.3	115	71 -- 175	12	10.2	117	60 -- 205	9	8.1	112	51 -- 212

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 7c
Leukemia Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	4.9	NC	NC -- NC	3	2.8	NC	NC -- NC	0	2.1	NC	NC -- NC
6312 & 6315	5	5.4	92	30 -- 214	2	3.0	NC	NC -- NC	3	2.4	NC	NC -- NC
6313	5	3.1	162	52 -- 379	3	1.6	NC	NC -- NC	2	1.4	NC	NC -- NC
6314	1	1.3	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	1	2.3	NC	NC -- NC	0	1.2	NC	NC -- NC	1	1.1	NC	NC -- NC
6317	4	4.1	NC	NC -- NC	1	2.1	NC	NC -- NC	3	2.0	NC	NC -- NC
6318	3	4.1	NC	NC -- NC	2	2.1	NC	NC -- NC	1	2.0	NC	NC -- NC
City Total	22	25.2	87	55 -- 132	12	13.6	88	46 -- 155	10	11.6	86	41 -- 158

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 7d
Leukemia Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	3.0	NC	NC -- NC	0	1.7	NC	NC -- NC	2	1.3	NC	NC -- NC
6312 & 6315	4	3.1	NC	NC -- NC	2	1.7	NC	NC -- NC	2	1.4	NC	NC -- NC
6313	1	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.8	NC	NC -- NC
6314	0	0.7	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC
6316	0	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
6317	2	2.6	NC	NC -- NC	1	1.2	NC	NC -- NC	1	1.3	NC	NC -- NC
6318	2	2.4	NC	NC -- NC	2	1.2	NC	NC -- NC	0	1.2	NC	NC -- NC
City Total[†]	12	14.8	81	42 -- 142	6	7.8	77	28 -- 168	6	7.0	86	31 -- 187

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 8a
Liver Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
6312 & 6315	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC
6313	0	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
6314	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6317	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
6318	1	0.5	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
City Total	1	3.6	NC	NC -- NC	1	2.4	NC	NC -- NC	0	1.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 8b
Liver Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	1.0	NC	NC -- NC	3	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
6312 & 6315	1	1.1	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
6313	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
6314	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	2	0.4	NC	NC -- NC	1	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC
6317	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
6318	1	0.8	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total	8	4.9	162	70 -- 319	7	3.6	197	79 -- 405	1	1.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 8c
Liver Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	1.6	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.4	NC	NC -- NC
6312 & 6315	2	1.7	NC	NC -- NC	1	1.2	NC	NC -- NC	1	0.5	NC	NC -- NC
6313	0	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
6314	1	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC
6316	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
6317	1	1.2	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC
6318	2	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC	2	0.4	NC	NC -- NC
City Total	7	7.5	93	37 -- 191	3	5.4	NC	NC -- NC	4	2.1	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 8d
Liver Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	1.3	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.3	NC	NC -- NC
6312 & 6315	4	1.3	NC	NC -- NC	3	1.0	NC	NC -- NC	1	0.3	NC	NC -- NC
6313	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
6314	2	0.3	NC	NC -- NC	2	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
6317	2	1.0	NC	NC -- NC	2	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
6318	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
City Total	11	6.1	180	90 -- 322	10	4.6	217 *	104 -- 399	1	1.5	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 9a
Lung & Bronchus Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	22	26.6	83	52 -- 125	15	16.5	91	51 -- 150	7	10.0	70	28 -- 144
6312 & 6315	26	29.9	87	57 -- 128	15	18.0	83	47 -- 138	11	11.9	92	46 -- 165
6313	14	17.1	82	45 -- 138	10	10.3	97	47 -- 179	4	6.8	NC	NC -- NC
6314	11	8.2	135	67 -- 242	9	5.0	180	82 -- 341	2	3.1	NC	NC -- NC
6316	10	12.7	79	38 -- 145	8	7.3	109	47 -- 215	2	5.3	NC	NC -- NC
6317	19	18.6	102	61 -- 159	15	11.4	131	74 -- 217	4	7.2	NC	NC -- NC
6318	15	16.3	92	52 -- 152	14	9.8	143	78 -- 240	1	6.5	NC	NC -- NC
City Total†	119	129.2	92	76 -- 110	88	78.3	112	90 -- 138	31	50.1	61	* 41 -- 86

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 9b
Lung & Bronchus Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	30	28.9	104	70 -- 148	9	17.7	51	* 23 -- 97	21	11.2	187	* 116 -- 286
6312 & 6315	33	33.2	99	68 -- 140	20	19.4	103	63 -- 159	13	13.7	95	50 -- 162
6313	16	18.8	85	49 -- 138	10	10.9	92	44 -- 169	6	8.0	75	28 -- 164
6314	17	7.6	222	* 129 -- 356	7	4.6	152	61 -- 312	10	3.0	331	* 158 -- 608
6316	12	12.0	100	52 -- 174	9	6.6	136	62 -- 258	3	5.4	NC	NC -- NC
6317	17	20.2	84	49 -- 135	8	11.9	67	29 -- 133	9	8.3	108	49 -- 206
6318	35	21.6	162	* 113 -- 225	15	12.1	124	69 -- 205	20	9.5	210	* 128 -- 324
City Total†	163	142.4	114	98 -- 133	78	83.2	94	74 -- 117	85	59.2	144	* 115 -- 178

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 9c
Lung & Bronchus Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	38	34.3	111	78 -- 152	26	18.5	140	92 -- 205	12	15.8	76	39 -- 133
6312 & 6315	30	36.5	82	55 -- 117	17	19.1	89	52 -- 143	13	17.5	74	40 -- 127
6313	19	20.4	93	56 -- 145	15	10.2	147	82 -- 243	4	10.3	NC	NC -- NC
6314	13	7.7	168	90 -- 288	10	4.2	237	* 113 -- 436	3	3.5	NC	NC -- NC
6316	13	13.6	96	51 -- 164	8	6.9	116	50 -- 228	5	6.7	75	24 -- 175
6317	18	25.8	70	41 -- 110	9	12.8	70	32 -- 133	9	13.0	69	32 -- 132
6318	29	25.1	116	77 -- 166	19	12.2	155	93 -- 242	10	12.8	78	37 -- 143
City Total†	161	163.4	99	84 -- 115	104	83.9	124	* 101 -- 150	57	79.4	72	* 54 -- 93

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 9d
Lung and Bronchus Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	18	19.5	92	55 -- 146	12	10.3	116	60 -- 203	6	9.2	65	24 -- 143
6312 & 6315	21	19.9	105	65 -- 161	13	10.4	125	67 -- 214	8	9.5	84	36 -- 165
6313	14	10.9	129	70 -- 216	7	5.3	133	53 -- 273	7	5.6	125	50 -- 258
6314	3	4.0	NC	NC -- NC	1	2.2	NC	NC -- NC	2	1.8	NC	NC -- NC
6316	8	7.4	107	46 -- 212	5	3.9	129	42 -- 301	3	3.6	NC	NC -- NC
6317	12	15.8	76	39 -- 133	4	7.5	NC	NC -- NC	8	8.3	97	42 -- 190
6318	22	14.0	158	99 -- 239	10	6.7	149	71 -- 275	12	7.3	165	85 -- 289
City Total[†]	99	91.5	108	88 -- 132	52	46.2	112	84 -- 147	47	45.2	104	76 -- 138

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 10a
Multiple Myeloma Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	1.5	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
6312 & 6315	0	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.9	NC	NC -- NC
6313	0	1.0	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
6314	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6316	1	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.5	NC	NC -- NC
6317	2	1.1	NC	NC -- NC	0	0.5	NC	NC -- NC	2	0.6	NC	NC -- NC
6318	3	0.9	NC	NC -- NC	1	0.5	NC	NC -- NC	2	0.5	NC	NC -- NC
City Total	7	7.6	92	37 -- 190	1	3.7	NC	NC -- NC	6	3.9	154	56 -- 335

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 10b
Multiple Myeloma Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
6312 & 6315	2	2.0	NC	NC -- NC	2	1.0	NC	NC -- NC	0	1.0	NC	NC -- NC
6313	1	1.2	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.6	NC	NC -- NC
6314	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6316	0	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.4	NC	NC -- NC
6317	1	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.6	NC	NC -- NC
6318	2	1.4	NC	NC -- NC	2	0.6	NC	NC -- NC	0	0.7	NC	NC -- NC
City Total	7	8.7	80	32 -- 165	6	4.3	140	51 -- 305	1	4.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 10c
Multiple Myeloma Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	2.2	NC	NC -- NC	1	1.2	NC	NC -- NC	1	1.0	NC	NC -- NC
6312 & 6315	4	2.3	NC	NC -- NC	3	1.2	NC	NC -- NC	1	1.1	NC	NC -- NC
6313	4	1.3	NC	NC -- NC	2	0.6	NC	NC -- NC	2	0.7	NC	NC -- NC
6314	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC
6316	2	0.9	NC	NC -- NC	2	0.4	NC	NC -- NC	0	0.5	NC	NC -- NC
6317	0	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.9	NC	NC -- NC
6318	1	1.7	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.9	NC	NC -- NC
City Total†	15	10.6	142	79 -- 234	10	5.3	189	90 -- 347	5	5.3	95	31 -- 221

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 10d
Multiple Myeloma Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.6	NC	NC -- NC
6312 & 6315	1	1.5	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.6	NC	NC -- NC
6313	0	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.4	NC	NC -- NC
6314	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
6316	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.3	NC	NC -- NC
6317	1	1.2	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.6	NC	NC -- NC
6318	0	1.1	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
City Total	3	6.9	NC	NC -- NC	1	3.7	NC	NC -- NC	2	3.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 11a
Non-Hodgkin's Lymphoma (NHL) Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	5.1	NC	NC -- NC	2	2.7	NC	NC -- NC	1	2.4	NC	NC -- NC
6312 & 6315	3	6.1	NC	NC -- NC	2	3.0	NC	NC -- NC	1	3.1	NC	NC -- NC
6313	3	3.5	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.8	NC	NC -- NC
6314	1	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.8	NC	NC -- NC
6316	1	2.9	NC	NC -- NC	1	1.3	NC	NC -- NC	0	1.5	NC	NC -- NC
6317	4	4.0	NC	NC -- NC	1	2.0	NC	NC -- NC	3	2.0	NC	NC -- NC
6318	4	3.5	NC	NC -- NC	1	1.8	NC	NC -- NC	3	1.7	NC	NC -- NC
City Total	19	26.8	71	43 -- 111	9	13.5	67	30 -- 127	10	13.3	75	36 -- 138

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 11b
Non-Hodgkin's Lymphoma (NHL) Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	9	6.7	134	61 -- 254	5	3.6	137	44 -- 320	4	3.1	NC	NC -- NC
6312 & 6315	3	8.1	NC	NC -- NC	0	4.2	NC	NC -- NC	3	4.0	NC	NC -- NC
6313	7	4.7	147	59 -- 304	1	2.4	NC	NC -- NC	6	2.4	251	92 -- 546
6314	0	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.9	NC	NC -- NC
6316	2	3.4	NC	NC -- NC	0	1.6	NC	NC -- NC	2	1.7	NC	NC -- NC
6317	7	5.1	137	55 -- 282	4	2.6	NC	NC -- NC	3	2.5	NC	NC -- NC
6318	8	5.7	140	60 -- 277	5	2.8	177	57 -- 413	3	2.9	NC	NC -- NC
City Total	36	35.8	101	70 -- 139	15	18.4	82	46 -- 135	21	17.4	121	75 -- 185

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 11c
Non-Hodgkin's Lymphoma (NHL) Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	9	8.7	103	47 -- 196	3	4.7	NC	NC -- NC	6	4.0	149	54 -- 325
6312 & 6315	11	9.6	114	57 -- 205	5	5.0	99	32 -- 231	6	4.6	131	48 -- 285
6313	4	5.4	NC	NC -- NC	3	2.7	NC	NC -- NC	1	2.7	NC	NC -- NC
6314	2	2.3	NC	NC -- NC	0	1.3	NC	NC -- NC	2	1.0	NC	NC -- NC
6316	4	3.9	NC	NC -- NC	3	2.0	NC	NC -- NC	1	1.9	NC	NC -- NC
6317	6	7.1	85	31 -- 185	2	3.5	NC	NC -- NC	4	3.6	NC	NC -- NC
6318	7	7.0	99	40 -- 205	3	3.5	NC	NC -- NC	4	3.6	NC	NC -- NC
City Total	43	44.1	98	71 -- 131	19	22.7	84	50 -- 131	24	21.4	112	72 -- 167

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 11d
Non-Hodgkin's Lymphoma Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	5.1	NC	NC -- NC	1	2.7	NC	NC -- NC	1	2.4	NC	NC -- NC
6312 & 6315	6	5.4	111	40 -- 241	3	2.8	NC	NC -- NC	3	2.6	NC	NC -- NC
6313	4	3.0	NC	NC -- NC	3	1.5	NC	NC -- NC	1	1.5	NC	NC -- NC
6314	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	0	2.2	NC	NC -- NC	0	1.1	NC	NC -- NC	0	1.0	NC	NC -- NC
6317	3	4.3	NC	NC -- NC	2	2.0	NC	NC -- NC	1	2.3	NC	NC -- NC
6318	4	4.0	NC	NC -- NC	0	1.9	NC	NC -- NC	4	2.1	NC	NC -- NC
City Total[†]	20	25.2	79	48 -- 122	9	12.8	70	32 -- 133	11	12.4	89	44 -- 159

[†] Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 12a
Pancreatic Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	3.6	NC	NC -- NC	0	1.8	NC	NC -- NC	0	1.8	NC	NC -- NC
6312 & 6315	3	4.4	NC	NC -- NC	1	2.0	NC	NC -- NC	2	2.3	NC	NC -- NC
6313	1	2.5	NC	NC -- NC	0	1.2	NC	NC -- NC	1	1.4	NC	NC -- NC
6314	1	1.2	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.6	NC	NC -- NC
6316	1	2.1	NC	NC -- NC	1	0.9	NC	NC -- NC	0	1.2	NC	NC -- NC
6317	3	2.8	NC	NC -- NC	1	1.3	NC	NC -- NC	2	1.5	NC	NC -- NC
6318	1	2.4	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.3	NC	NC -- NC
City Total	10	19.0	53	* 25 -- 97	4	8.8	NC	NC -- NC	6	10.2	59	22 -- 129

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 12b
Pancreatic Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	3	3.8	NC	NC -- NC	2	1.9	NC	NC -- NC	1	1.9	NC	NC -- NC
6312 & 6315	3	4.6	NC	NC -- NC	1	2.2	NC	NC -- NC	2	2.5	NC	NC -- NC
6313	2	2.7	NC	NC -- NC	0	1.2	NC	NC -- NC	2	1.5	NC	NC -- NC
6314	3	1.0	NC	NC -- NC	3	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
6316	2	1.8	NC	NC -- NC	2	0.8	NC	NC -- NC	0	1.1	NC	NC -- NC
6317	4	2.8	NC	NC -- NC	2	1.3	NC	NC -- NC	2	1.5	NC	NC -- NC
6318	2	3.1	NC	NC -- NC	0	1.4	NC	NC -- NC	2	1.8	NC	NC -- NC
City Total	19	19.9	95	57 -- 149	10	9.3	108	52 -- 198	9	10.7	84	39 -- 160

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 12c
Pancreatic Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	6	4.9	123	45 -- 268	2	2.4	NC	NC -- NC	4	2.4	NC	NC -- NC
6312 & 6315	6	5.3	113	41 -- 245	1	2.5	NC	NC -- NC	5	2.8	180	58 -- 421
6313	1	3.0	NC	NC -- NC	1	1.4	NC	NC -- NC	0	1.7	NC	NC -- NC
6314	2	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	2	0.5	NC	NC -- NC
6316	1	2.1	NC	NC -- NC	1	0.9	NC	NC -- NC	0	1.2	NC	NC -- NC
6317	3	4.1	NC	NC -- NC	1	1.8	NC	NC -- NC	2	2.4	NC	NC -- NC
6318	7	3.9	181	72 -- 373	1	1.7	NC	NC -- NC	6	2.2	270	99 -- 589
City Total	26	24.5	106	69 -- 156	7	11.2	62	25 -- 128	19	13.2	144	87 -- 225

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 12d
Pancreatic Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	1	3.1	NC	NC -- NC	0	1.5	NC	NC -- NC	1	1.6	NC	NC -- NC
6312 & 6315	1	3.2	NC	NC -- NC	1	1.5	NC	NC -- NC	0	1.7	NC	NC -- NC
6313	2	1.8	NC	NC -- NC	1	0.8	NC	NC -- NC	1	1.0	NC	NC -- NC
6314	1	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.3	NC	NC -- NC
6316	1	1.3	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.7	NC	NC -- NC
6317	1	2.8	NC	NC -- NC	1	1.1	NC	NC -- NC	0	1.7	NC	NC -- NC
6318	2	2.4	NC	NC -- NC	1	1.0	NC	NC -- NC	1	1.4	NC	NC -- NC
City Total	9	15.3	59	27 -- 111	4	6.9	NC	NC -- NC	5	8.5	59	19 -- 138

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 13a
Thyroid Cancer Incidence
Attleboro, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	0	1.3	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.9	NC	NC -- NC
6312 & 6315	1	1.6	NC	NC -- NC	0	0.5	NC	NC -- NC	1	1.1	NC	NC -- NC
6313	0	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.6	NC	NC -- NC
6314	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.3	NC	NC -- NC
6316	1	0.8	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.5	NC	NC -- NC
6317	0	1.1	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.7	NC	NC -- NC
6318	0	1.0	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.7	NC	NC -- NC
City Total†	3	7.2	NC	NC -- NC	0	2.4	NC	NC -- NC	3	4.8	NC	NC -- NC

† Cases for which census tract designation was not possible are included in the city total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 13b
Thyroid Cancer Incidence
Attleboro, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	5	1.8	285	92 -- 665	2	0.5	NC	NC -- NC	3	1.2	NC	NC -- NC
6312 & 6315	4	2.1	NC	NC -- NC	3	0.6	NC	NC -- NC	1	1.5	NC	NC -- NC
6313	0	1.2	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.9	NC	NC -- NC
6314	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.4	NC	NC -- NC
6316	1	1.0	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.7	NC	NC -- NC
6317	0	1.4	NC	NC -- NC	0	0.4	NC	NC -- NC	0	1.0	NC	NC -- NC
6318	2	1.6	NC	NC -- NC	1	0.4	NC	NC -- NC	1	1.2	NC	NC -- NC
City Total	12	9.6	125	65 -- 219	7	2.6	266	* 107 -- 548	5	6.9	72	23 -- 168

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 13c
Thyroid Cancer Incidence
Attleboro, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	2.8	NC	NC -- NC	1	0.7	NC	NC -- NC	1	2.0	NC	NC -- NC
6312 & 6315	6	3.2	187	68 -- 407	1	0.8	NC	NC -- NC	5	2.4	211	68 -- 493
6313	3	1.8	NC	NC -- NC	1	0.5	NC	NC -- NC	2	1.3	NC	NC -- NC
6314	1	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.6	NC	NC -- NC
6316	0	1.4	NC	NC -- NC	0	0.4	NC	NC -- NC	0	1.1	NC	NC -- NC
6317	3	2.2	NC	NC -- NC	1	0.6	NC	NC -- NC	2	1.6	NC	NC -- NC
6318	1	2.5	NC	NC -- NC	1	0.6	NC	NC -- NC	0	1.9	NC	NC -- NC
City Total	16	14.8	108	62 -- 176	5	3.9	130	42 -- 302	11	10.9	101	50 -- 180

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 13d
Thyroid Cancer Incidence
Attleboro, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6311	2	2.3	NC	NC -- NC	0	0.5	NC	NC -- NC	2	1.8	NC	NC -- NC
6312 & 6315	1	2.7	NC	NC -- NC	1	0.6	NC	NC -- NC	0	2.1	NC	NC -- NC
6313	2	1.4	NC	NC -- NC	0	0.3	NC	NC -- NC	2	1.1	NC	NC -- NC
6314	4	0.7	NC	NC -- NC	2	0.2	NC	NC -- NC	2	0.5	NC	NC -- NC
6316	1	1.1	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.9	NC	NC -- NC
6317	1	1.8	NC	NC -- NC	0	0.4	NC	NC -- NC	1	1.4	NC	NC -- NC
6318	4	2.0	NC	NC -- NC	1	0.4	NC	NC -- NC	3	1.6	NC	NC -- NC
City Total	15	12.1	124	69 -- 204	5	2.8	182	59 -- 424	10	9.4	107	51 -- 197

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 14a
Bladder Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	4.7	NC	NC -- NC	0	3.4	NC	NC -- NC	2	1.3	NC	NC -- NC
6112	1	5.5	NC	NC -- NC	0	4.0	NC	NC -- NC	1	1.5	NC	NC -- NC
Town Total	3	10.2	NC	NC -- NC	0	7.4	NC	NC -- NC	3	2.8	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 14b
Bladder Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	5	5.0	100	32 -- 234	4	3.6	NC	NC -- NC	1	1.4	NC	NC -- NC
6112	2	5.4	NC	NC -- NC	2	3.9	NC	NC -- NC	0	1.6	NC	NC -- NC
Town Total	7	10.4	67	27 -- 139	6	7.5	80	29 -- 175	1	2.9	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 14c
Bladder Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	5	5.2	96	31 -- 224	4	3.8	NC	NC -- NC	1	1.4	NC	NC -- NC
6112	5	6.1	81	26 -- 190	4	4.3	NC	NC -- NC	1	1.8	NC	NC -- NC
Town Total	10	11.4	88	42 -- 162	8	8.1	98	42 -- 194	2	3.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 14d
Bladder Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	2.4	NC	NC -- NC	2	1.7	NC	NC -- NC	1	0.6	NC	NC -- NC
6112	2	2.9	NC	NC -- NC	1	2.1	NC	NC -- NC	1	0.8	NC	NC -- NC
Town Total	5	5.3	94	30 -- 220	3	3.8	NC	NC -- NC	2	1.5	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 15a
Bone Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6112	1	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	1	0.7	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 15b
Bone Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6112	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	0	0.8	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 15c
Bone Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
6112	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 15d
Bone Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
6112	1	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	1	0.1	NC	NC -- NC
Town Total	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 16a
Brain & Central Nervous System (CNS) Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	2.6	NC	NC -- NC	1	1.2	NC	NC -- NC	0	1.4	NC	NC -- NC
6112	3	2.6	NC	NC -- NC	1	1.3	NC	NC -- NC	2	1.3	NC	NC -- NC
Town Total	4	5.2	NC	NC -- NC	2	2.6	NC	NC -- NC	2	2.7	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 16b
Brain & Central Nervous System (CNS) Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	3.1	NC	NC -- NC	0	1.5	NC	NC -- NC	1	1.6	NC	NC -- NC
6112	4	3.2	NC	NC -- NC	1	1.6	NC	NC -- NC	3	1.6	NC	NC -- NC
Town Total	5	6.3	79	25 -- 184	1	3.1	NC	NC -- NC	4	3.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 16c
Brain & Central Nervous System (CNS) Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	4	2.9	NC	NC -- NC	2	1.6	NC	NC -- NC	2	1.4	NC	NC -- NC
6112	1	3.1	NC	NC -- NC	1	1.7	NC	NC -- NC	0	1.4	NC	NC -- NC
Town Total	5	6.0	83	27 -- 193	3	3.3	NC	NC -- NC	2	2.8	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 16d
Brain & Central Nervous System (CNS) Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.8	NC	NC -- NC
6112	6	1.8	328	* 120 -- 714	5	1.0	507	* 163 -- 1182	1	0.8	NC	NC -- NC
Town Total	7	3.5	197	79 -- 406	5	1.9	263	85 -- 614	2	1.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 17a
Breast Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	10	18.5	54	* 26 -- 99	0	0.1	NC	NC -- NC	10	18.4	54	26 -- 100
6112	22	19.9	111	69 -- 168	1	0.1	NC	NC -- NC	21	19.7	106	66 -- 163
Town Total†	33	38.4	86	59 -- 121	1	0.2	NC	NC -- NC	32	38.2	84	57 -- 118

† Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 17b
Breast Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	28	23.8	118	78 -- 170	0	0.2	NC	NC -- NC	28	23.6	118	79 -- 171
6112	24	25.9	93	59 -- 138	0	0.2	NC	NC -- NC	24	25.7	93	60 -- 139
Town Total	52	49.7	105	78 -- 137	0	0.3	NC	NC -- NC	52	49.3	105	79 -- 138

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 17c
Breast Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	28	28.7	98	65 -- 141	0	0.2	NC	NC -- NC	28	28.5	98	65 -- 142
6112	25	32.3	78	50 -- 114	0	0.2	NC	NC -- NC	25	32.0	78	51 -- 115
Town Total[†]	54	61.0	89	67 -- 116	0	0.4	NC	NC -- NC	54	60.5	89	67 -- 116

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 17d
Breast Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	9	16.4	55	25 -- 104	0	0.1	NC	NC -- NC	9	16.3	55	25 -- 105
6112	12	18.5	65	34 -- 113	0	0.1	NC	NC -- NC	12	18.3	65	34 -- 114
Town Total†	22	34.9	63	* 40 -- 95	0	0.2	NC	NC -- NC	22	34.7	63	* 40 -- 96

† Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 18a
Hodgkin's Disease Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	1.5	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.8	NC	NC -- NC
6112	0	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
Town Total	0	2.9	NC	NC -- NC	0	1.5	NC	NC -- NC	0	1.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 18b
Hodgkin's Disease Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	1.6	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.9	NC	NC -- NC
6112	2	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.8	NC	NC -- NC
Town Total[†]	3	3.3	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.6	NC	NC -- NC

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 18c
Hodgkin's Disease Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
6112	0	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
Town Total	1	3.5	NC	NC -- NC	1	1.9	NC	NC -- NC	0	1.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 18d
Hodgkin's Disease Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	0.9	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.4	NC	NC -- NC
6112	0	0.8	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
Town Total[†]	2	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	2	0.8	NC	NC -- NC

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 19a
Kidney Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.2	NC	NC -- NC	1	1.3	NC	NC -- NC	1	0.9	NC	NC -- NC
6112	3	2.5	NC	NC -- NC	1	1.5	NC	NC -- NC	2	1.0	NC	NC -- NC
Town Total[†]	6	4.7	127	46 -- 276	3	2.8	NC	NC -- NC	3	1.9	NC	NC -- NC

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 19b
Kidney Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	6	3.4	176	64 -- 383	3	2.1	NC	NC -- NC	3	1.3	NC	NC -- NC
6112	5	3.7	135	44 -- 315	4	2.3	NC	NC -- NC	1	1.4	NC	NC -- NC
Town Total	11	7.1	155	77 -- 277	7	4.4	160	64 -- 329	4	2.7	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 19c
Kidney Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	6	4.2	143	52 -- 312	4	2.6	NC	NC -- NC	2	1.6	NC	NC -- NC
6112	2	4.7	NC	NC -- NC	1	2.9	NC	NC -- NC	1	1.8	NC	NC -- NC
Town Total[†]	9	8.9	101	46 -- 191	5	5.5	91	29 -- 213	4	3.4	NC	NC -- NC

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 19d
Kidney Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	2.7	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1	NC	NC -- NC
6112	3	3.1	NC	NC -- NC	3	1.9	NC	NC -- NC	0	1.2	NC	NC -- NC
Town Total	6	5.8	104	38 -- 225	5	3.5	141	45 -- 329	1	2.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 20a
Leukemia Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.5	NC	NC -- NC	1	1.4	NC	NC -- NC	1	1.2	NC	NC -- NC
6112	2	2.7	NC	NC -- NC	1	1.5	NC	NC -- NC	1	1.2	NC	NC -- NC
Town Total[†]	5	5.2	96	31 -- 223	3	2.9	NC	NC -- NC	2	2.4	NC	NC -- NC

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 20b
Leukemia Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.8	NC	NC -- NC	2	1.5	NC	NC -- NC	0	1.3	NC	NC -- NC
6112	1	3.0	NC	NC -- NC	0	1.7	NC	NC -- NC	1	1.3	NC	NC -- NC
Town Total	3	5.8	NC	NC -- NC	2	3.2	NC	NC -- NC	1	2.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE20c
Leukemia Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	4.0	NC	NC -- NC	2	2.2	NC	NC -- NC	1	1.9	NC	NC -- NC
6112	0	4.4	NC	NC -- NC	0	2.4	NC	NC -- NC	0	2.0	NC	NC -- NC
Town Total	3	8.4	NC	NC -- NC	2	4.5	NC	NC -- NC	1	3.9	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 20d
Leukemia Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.4	NC	NC -- NC	2	1.3	NC	NC -- NC	0	1.1	NC	NC -- NC
6112	2	2.7	NC	NC -- NC	1	1.4	NC	NC -- NC	1	1.2	NC	NC -- NC
Town Total	4	5.1	NC	NC -- NC	3	2.7	NC	NC -- NC	1	2.3	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 21a
Liver Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC
6112	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	2	1.0	NC	NC -- NC	1	0.7	NC	NC -- NC	1	0.3	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 21b
Liver Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	1	0.2	NC	NC -- NC
6112	0	0.8	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	1	1.5	NC	NC -- NC	0	1.1	NC	NC -- NC	1	0.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 21c
Liver Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.1	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
6112	1	1.3	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC
Town Total	2	2.4	NC	NC -- NC	2	1.8	NC	NC -- NC	0	0.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 21d
Liver Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	1	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.2	NC	NC -- NC
6112	0	1.1	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC
Town Total†	3	2.1	NC	NC -- NC	2	1.6	NC	NC -- NC	1	0.5	NC	NC -- NC

† Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 22a
Lung & Bronchus Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	13	16.6	78	42 -- 134	9	10.2	88	40 -- 168	4	6.4	NC	NC -- NC
6112	18	18.8	96	57 -- 152	10	11.7	85	41 -- 157	8	7.1	113	49 -- 224
Town Total†	32	35.4	90	62 -- 128	19	21.9	87	52 -- 135	13	13.5	97	51 -- 165

† Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 22b
Lung & Bronchus Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	15	19.8	76	42 -- 125	12	11.9	101	52 -- 177	3	7.9	NC	NC -- NC
6112	29	21.4	136	91 -- 195	20	12.7	158	96 -- 244	9	8.7	103	47 -- 195
Town Total†	45	41.2	109	80 -- 146	33	24.5	135	93 -- 189	12	16.7	72	37 -- 126

† Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 22c
Lung & Bronchus Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	38	22.9	166	* 117 -- 227	17	12.1	140	81 -- 224	21	10.8	194	* 120 -- 297
6112	21	26.6	79	49 -- 121	9	13.9	65	30 -- 123	12	12.7	94	49 -- 165
Town Total	59	49.5	119	91 -- 154	26	26.0	100	65 -- 146	33	23.5	140	96 -- 197

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 22d
Lung and Bronchus Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	9	13.1	68	31 -- 130	8	6.9	116	50 -- 229	1	6.3	NC	NC -- NC
6112	17	15.8	108	63 -- 173	9	8.1	111	51 -- 210	8	7.6	105	45 -- 206
Town Total[†]	29	28.9	100	67 -- 144	19	15.0	126	76 -- 198	10	13.9	72	34 -- 132

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 23a
Multiple Myeloma Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	0	1.0	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
6112	1	1.1	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
Town Total	1	2.0	NC	NC -- NC	1	1.0	NC	NC -- NC	0	1.0	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 23b
Multiple Myeloma Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.6	NC	NC -- NC
6112	1	1.3	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.7	NC	NC -- NC
Town Total	2	2.5	NC	NC -- NC	1	1.2	NC	NC -- NC	1	1.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 23c
Multiple Myeloma Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
6112	2	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	2	0.9	NC	NC -- NC
Town Total	4	3.2	NC	NC -- NC	1	1.7	NC	NC -- NC	3	1.6	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 23d
Multiple Myeloma Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
6112	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
Town Total	1	2.1	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 24a
Non-Hodgkin's Lymphoma (NHL) Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	5	3.7	136	44 -- 317	3	1.9	NC	NC -- NC	2	1.8	NC	NC -- NC
6112	2	4.0	NC	NC -- NC	1	2.1	NC	NC -- NC	1	1.9	NC	NC -- NC
Town Total	7	7.7	91	36 -- 187	4	4.0	NC	NC -- NC	3	3.7	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 24b
Non-Hodgkin's Lymphoma (NHL) Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	5.2	NC	NC -- NC	2	2.7	NC	NC -- NC	1	2.5	NC	NC -- NC
6112	11	5.7	194	97 -- 347	6	3.0	201	73 -- 437	5	2.7	186	60 -- 434
Town Total	14	10.9	129	70 -- 216	8	5.7	140	60 -- 276	6	5.2	116	42 -- 253

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 24c
Non-Hodgkin's Lymphoma (NHL) Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	6	6.7	90	33 -- 195	1	3.6	NC	NC -- NC	5	3.1	161	52 -- 376
6112	6	7.5	80	29 -- 173	2	4.0	NC	NC -- NC	4	3.5	NC	NC -- NC
Town Total	12	14.2	84	43 -- 147	3	7.6	NC	NC -- NC	9	6.6	135	62 -- 257

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 24d
Non-Hodgkin's Lymphoma (NHL) Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	3.9	NC	NC -- NC	1	2.1	NC	NC -- NC	2	1.8	NC	NC -- NC
6112	3	4.5	NC	NC -- NC	1	2.4	NC	NC -- NC	2	2.1	NC	NC -- NC
Town Total[†]	7	8.5	83	33 -- 171	2	4.5	NC	NC -- NC	5	4.0	126	41 -- 294

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 25a
Pancreatic Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	4	2.4	NC	NC -- NC	2	1.1	NC	NC -- NC	2	1.2	NC	NC -- NC
6112	3	2.7	NC	NC -- NC	3	1.3	NC	NC -- NC	0	1.4	NC	NC -- NC
Town Total	7	5.1	136	55 -- 281	5	2.5	202	65 -- 472	2	2.7	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 25b
Pancreatic Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	3	2.7	NC	NC -- NC	0	1.3	NC	NC -- NC	3	1.4	NC	NC -- NC
6112	6	3.0	201	73 -- 437	2	1.4	NC	NC -- NC	4	1.6	NC	NC -- NC
Town Total[†]	10	5.7	176	84 -- 323	3	2.7	NC	NC -- NC	7	3.0	236	95 -- 487

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 25c
Pancreatic Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	4	3.3	NC	NC -- NC	1	1.6	NC	NC -- NC	3	1.7	NC	NC -- NC
6112	5	3.9	127	41 -- 296	2	1.9	NC	NC -- NC	3	2.1	NC	NC -- NC
Town Total[†]	10	7.2	138	66 -- 254	3	3.5	NC	NC -- NC	7	3.7	188	75 -- 387

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 25d
Pancreatic Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.1	NC	NC -- NC	1	1.1	NC	NC -- NC	1	1.1	NC	NC -- NC
6112	2	2.6	NC	NC -- NC	0	1.2	NC	NC -- NC	2	1.4	NC	NC -- NC
Town Total	4	4.7	NC	NC -- NC	1	2.3	NC	NC -- NC	3	2.5	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 26a
Thyroid Cancer Incidence
Norton, Massachusetts
1982-1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.3	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.9	NC	NC -- NC
6112	0	1.2	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.8	NC	NC -- NC
Town Total	1	2.5	NC	NC -- NC	0	0.8	NC	NC -- NC	1	1.8	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 26b
Thyroid Cancer Incidence
Norton, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	1	1.7	NC	NC -- NC	0	0.4	NC	NC -- NC	1	1.3	NC	NC -- NC
6112	1	1.7	NC	NC -- NC	0	0.5	NC	NC -- NC	1	1.3	NC	NC -- NC
Town Total	2	3.4	NC	NC -- NC	0	0.9	NC	NC -- NC	2	2.5	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 26c
Thyroid Cancer Incidence
Norton, Massachusetts
1994-1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	2	2.9	NC	NC -- NC	0	0.7	NC	NC -- NC	2	2.2	NC	NC -- NC
6112	1	2.9	NC	NC -- NC	0	0.7	NC	NC -- NC	1	2.1	NC	NC -- NC
Town Total	3	5.8	NC	NC -- NC	0	1.4	NC	NC -- NC	3	4.4	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 26d
Thyroid Cancer Incidence
Norton, Massachusetts
2000-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
6111	4	2.6	NC	NC -- NC	0	0.5	NC	NC -- NC	4	2.0	NC	NC -- NC
6112	3	2.5	NC	NC -- NC	0	0.6	NC	NC -- NC	3	1.9	NC	NC -- NC
Town Total[†]	8	5.0	159	69 -- 314	1	1.1	NC	NC -- NC	7	4.0	177	71 -- 365

[†] Cases for which census tract designation was not possible are included in the town total.

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

APPENDICES

Appendix A

Phase I: Evaluation of Cancer Incidence in Attleboro and Norton, Massachusetts, 1994–1998

Massachusetts
Department
Of
Public Health

Phase I:

Evaluation of Cancer Incidence in Attleboro and Norton, MA

1994-1998

June, 2002

Bureau of Environmental
Health Assessment,
Community Assessment
Program

Introduction/Methods

At the request of concerned citizens, the Community Assessment Program (CAP) of the Bureau of Environmental Health Assessment (BEHA) reviewed the available cancer incidence data for the years 1994-1998 for Attleboro and Norton. Resident concerns focused on suspected increases of cancer in neighborhoods near the Shpack Landfill, located on the border of the two towns (see Figure 1). Data for cancer in Attleboro and Norton was obtained from the Massachusetts Cancer Registry (MCR). The MCR has been monitoring cancer incidence in the Commonwealth since 1982. All newly diagnosed cancer cases are required by law to be reported to the MCR within six months of the date of diagnosis (M.G.L. c.111, s. 111b). This information is kept in a confidential database. The 5-year period from 1994-1998 constitutes the time period for which the most recent and complete cancer data were available at the time of this review.

Tables 1-4 summarize cancer incidence data for these towns for the 5-year period 1994-1998 for 23 different cancer types. The tables provide information on the number of cancer cases that occurred in Attleboro and Norton, the number of cancer cases expected in each area based on the towns' populations and the statewide cancer experience, and the Standardized Incidence Ratio (SIR). The SIR is a statistical measure that indicates whether the incidence of cancer is higher or lower than expected. An SIR greater than 100 indicates that more cancer cases occurred than expected while an SIR less than 100 means that fewer cases occurred than expected. A more detailed explanation of an SIR and the 95% Confidence Interval (95% CI), a statistical test used to interpret SIRs, is provided in Attachment A.

Cancer Incidence in Attleboro and Norton

A review of data from the *City and Town Supplement* showed that in Attleboro and Norton the majority of cancer types occurred approximately at or below expected rates for the period 1994-1998. That is, for most of the cancer types the SIR was approximately at or below 100. In Attleboro, overall incidence rates for six cancer types were elevated among males and females combined compared to state rates. These included colorectal cancer, Hodgkin's disease, laryngeal cancer, melanoma, multiple myeloma, and pancreatic cancer (see Tables 1 and 2). However, the difference between the number of observed and expected cases did not represent a statistically significant elevation. In Norton, elevations were observed in the incidence of lung and bronchus cancer (46 cases observed vs. approximately 40 expected) and pancreatic cancer (9 cases observed vs. 5.6 expected). While several additional cases

occurred beyond the expected number, these elevations were not statistically significant (see Tables 3 and 4).

More detailed review of this information for the city of Attleboro as a whole showed that two cancer types displayed statistically significant elevations during 1994-1998 when evaluated separately by gender. Hodgkin's disease occurred more often than expected among males in this city. While females in this city experienced Hodgkin's disease at about the rate expected (i.e., 3 cases observed vs. 3.1 expected), 9 males were diagnosed with Hodgkin's disease where approximately 4 were expected (SIR=231; 95% CI=106-439). However, the relatively wide 95% confidence interval indicates that the SIR for Hodgkin's disease among males is a somewhat unstable statistic (please see Attachment A for additional information). Among females, a statistically significant elevation in the incidence of liver cancer was also observed citywide. Six females were diagnosed with liver cancer during 1994-1998 where approximately 2 were expected (SIR=291; 95% CI=106-634). Again, the wide 95% confidence interval suggests that this SIR may be somewhat unstable. Liver cancer was diagnosed less often than expected among males in Attleboro (2 cases observed vs. approximately 5 expected). No statistically significant elevations were observed in the town of Norton during the five-year time period 1994-1998.

Discussion

When evaluating cancer incidence, it is important to keep in mind that cancer is a common disease. In fact, one out of every three Americans will develop some form of cancer during his or her lifetime. Over the past forty years, the dramatic rise in the number of cancer cases reflects the increase in the population, particularly in the older age groups. Understanding that cancer is not one disease, but a group of different diseases is also very important. Research has shown that there are more than 100 different types of cancer, each with different causative (or risk) factors. In addition, cancer of a certain tissue type in one organ may have a number of causes. Cancer may also be caused by one or several factors acting over time. For example, tobacco use has been linked to lung, pancreatic, stomach, bladder and several other cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer causing). Environmental contamination has also been associated with certain types of cancer.

Finally, it is important to note that cancer in general has a long period of development or latency period that can range from 10 to 30 years and in some cases may be more than 40 to 50 years. In order to

provide a better understanding of factors that are related to the development of cancer, we have attached a summary of additional information for cancer types that were elevated in Attleboro and Norton (see Attachment B).

According to American Cancer Society statistics, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of every three people develop cancer in their lifetime, but this tragedy will also affect three out of every four families. For this reason, cancers often appear to occur in "clusters," and it is understandable that someone may perceive that there are an unusually high number of cancer cases in their surrounding neighborhoods. Upon close examination, many of the "clusters" are not unusual increases, as first thought, but are related to such factors as local population density, variations in reporting, or chance fluctuation in occurrence. In other instances, the "cluster" in question includes a high concentration of individuals who possess related behaviors or risk factors for cancer. Some, however, are unusual; that is, they represent a true excess of cancer in a workplace, a community, or among a subgroup of people. These types of clusters warrant further public health investigation.

Certain chemical exposures have been suggested to be related to the development of Hodgkin's disease and liver cancer, the two cancer types for which statistically significant elevations were observed in Attleboro during 1994-1998, however, the most common risk factors for these cancer types are viral infections (hepatitis B and hepatitis C for liver cancer and an unknown agent for Hodgkin's disease) (see Appendix B).

Next Steps

It is important to note that the information provided addresses cancer rates only for each town as a whole. To evaluate cancer and environmental concerns in a specific area or neighborhoods within Attleboro or Norton, further investigation and analysis of data at a smaller geographic level is needed. In addition, when investigating the patterns of cancer in relation to a particular environmental exposure, it is important to consider the ways in which individuals may come in contact with environmental contaminants from a particular site (i.e. ingestion, inhalation, or skin contact) and the types of contaminants present. In response to recent requests to investigate the incidence of cancer in the Attleboro/Norton area in relation to concerns about the Shpack Landfill, the Community Assessment

Program (CAP) will investigate the pattern of cancer at a smaller geographic level, specifically in relation to potential environmental exposures present at this site.

Because cancer is a group of individual diseases that may be caused by separate and distinct factors, it is important to evaluate not only whether a relationship between a certain cancer type and environmental exposure exists but also, the types of chemicals or other hazardous substances present, which may be related to different cancer types. Based on the information summarized in this preliminary review as well as the information collected to date on the types of contaminants present at the Shpack Landfill site, we believe it is important to focus our more detailed analysis on 13 different cancer types. These cancer types were selected for further investigation because they are cancer types for which a statistically significant elevation in incidence occurred at the city/town level and they may be associated with either radiological or chemical contamination detected at the Shpack Landfill. The 13 cancer types include cancers of the bladder, brain and CNS, breast, bone, kidney, liver, lung and bronchus, pancreas, and thyroid as well as Hodgkin's disease, leukemia, multiple myeloma, and NHL. This would provide the most meaningful and timely information relative to cancer concerns and the Shpack Landfill. Other cancer types, such as cervical cancer, colorectal cancer, esophageal cancer, laryngeal cancer, melanoma, oral cancers, ovarian cancer, prostate cancer, stomach cancer, and uterine cancer, are generally not elevated and the most important risk factors cited in the scientific literature for these cancers are non-environmental factors such as genetics and family history, diet and exercise, and other lifestyle behaviors (e.g., smoking and alcohol use).

In order to investigate concerns regarding suspected elevations of cancer in Attleboro and Norton, the CAP will calculate incidence rates as well as examine the spatial and temporal pattern of these cancer types town-wide and by smaller geographic areas within each town (e.g., census tracts and neighborhoods) both quantitatively and qualitatively. Because accurate age-group and gender specific population data is necessary to calculate incidence rates, the census tract (CT) is the smallest geographic area for which a rate can be accurately calculated. The city of Attleboro is geographically subdivided into eight census tracts and the town of Norton is subdivided into two census tracts (see Figure 1). Therefore, this investigation will focus primarily on the census tract areas and neighborhoods adjacent to the Shpack Landfill (i.e., Attleboro CT 6317 and Norton CT 6112) (see Figure 2) and specifically the possible association between environmental exposure opportunities related to the Shpack Landfill and disease. As noted previously, cancer incidence data for geographic areas below the town level is not

readily available from the MCR. Therefore, to evaluate cancer and environmental concerns in these areas at a smaller geographic level, it will be necessary to review and map (i.e., geographically assign) the addresses of all individuals from Attleboro and Norton reported to the MCR with the 13 cancer types suggested for further analysis to determine the census tract and individual address location of individuals in CTs 6317 and 6112 as well as to determine the specific spatial pattern of cancer within one mile of the Shpack Landfill. Additionally, this investigation will include a review of available environmental data, determination of possible exposure scenarios, and a discussion of cancer incidence data in these towns in the context of environmental and other risk factor information available from the MCR.

Attachment A

Explanation of a Standardized Incidence Ratio (SIR) And 95% Confidence Interval

In order to evaluate cancer incidence a statistic known as a standardized incidence ratio (SIR) was calculated for each cancer type. An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as some larger comparison population designated as "normal" or average. Usually, the state as a whole is selected to be the comparison population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed.

Specifically, an SIR is the ratio of the observed number of cancer cases to the expected number of cases multiplied by 100. An SIR of 100 indicates that the number of cancer cases observed in the population evaluated is equal to the number of cancer cases expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer cases occurred than expected and an SIR less than 100 indicates that fewer cancer cases occurred than expected. Accordingly, an SIR of 150 is interpreted as 50% more cases than the expected number; an SIR of 90 indicates 10% fewer cases than expected.

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on 4 expected cases and 6 observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone.

To determine if the observed number of cases is significantly different from the expected number or if the difference may be due solely to chance, a 95% confidence interval (CI) was calculated for each SIR. A 95% CI assesses the magnitude and stability of an SIR. Specifically, a 95% CI is the range of estimated SIR values that has a 95% probability of including the true SIR for the population. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or "normal" population. "Significantly different" means there is less than 5% percent chance that the observed difference is the result of random fluctuation in the number of observed cancer cases.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105-130), then there is statistically significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45-96), then the number of cancer cases is statistically significantly lower than expected. If the confidence interval range includes 100, then the true SIR may be 100, and it cannot be concluded with sufficient confidence that the observed number of cases is not the result of chance and reflects a real cancer increase or decrease. Statistical significance is not assessed when fewer than five cases are observed.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval (e.g., 103--115) allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval (e.g., 85--450) leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic.

Source: Massachusetts Department of Public Health, Bureau of Environmental Health Assessment (October 2001)

Attachment B

Hodgkin's disease

Hodgkin's disease (or Hodgkin's lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin's lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,400 new cases of this disease in the U.S. in 2001, accounting for less than 1% of all cancer types, and approximately 1,300 deaths (ACS, 2001). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS, 1999).

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women and more common among whites than blacks. People of Jewish descent appear to be at higher risk of Hodgkin's disease compared to people of non-Jewish descent (Mueller, 1996). Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 to 40 (typically ages 25-30) and the second peak occurs in adults aged 55 years and above.

No major risk factors for Hodgkin's disease have been found (ACS, 1999). However, the clinical and cellular features of Hodgkin's disease suggest a chronic infectious process (Mueller, 1996). The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. Researchers have proposed that among young adults, Hodgkin's disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is probably similar to those of other lymphomas (Mueller, 1996). The virus that has been linked most specifically to this disease is the Epstein-Barr virus (EBV). EBV, a herpesvirus, is common in the general population and causes mononucleosis or "mono." Approximately 40% to 50% of Hodgkin's disease cases are associated with EBV (Weiss, 2000). In addition, several studies have also shown that young adults who have developed infectious mononucleosis have a significantly higher risk of developing Hodgkin's disease (ACS, 1999). However, the absence of EBV infection in about half the cases and the high prevalence of EBV in the general population suggest that EBV may be only one of several factors in the development of this cancer. Although cytomegalovirus (CMV) and the more recently identified human herpesvirus type 6 have been considered as possible factors in the development of Hodgkin's disease, results of antibody studies are inconsistent and these viruses do not appear to be related to risk of Hodgkin's disease (Mueller, 1996).

Slightly higher rates of Hodgkin's disease occur among people with reduced immunity, such as those with AIDS, people with congenital immune deficiencies, and individuals on immunosuppressant medication following organ transplants. However, Hodgkin's disease occurs at a much lower rate than non-Hodgkin's lymphomas among this group of individuals (ACS, 1999).

Hodgkin's disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin's disease is associated with an infectious agent (Mueller, 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller, 1996). Based on an examination of medical

and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin's disease does not seem to be caused by genetic, lifestyle (e.g., dietary), or environmental factors (ACS, 1999).

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Liver Cancer

An estimated 16,200 people in the U.S. (10,700 men and 5,500 women) will be diagnosed with liver cancer in 2001, accounting for approximately 1% of all new cancers (ACS, 2001a). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Rarer forms of malignant liver cancer include cholangiocarcinomas, angiosarcomas, and hepatoblastomas in children. Although HCC is approximately ten times more common in developing countries in East and Southeast Asia and Africa, incidence is rapidly increasing in the United States (ACS, 2001b). Rates of HCC in the U.S. have increased by 70% over the past two decades (Yu et al., 2000). Similar trends have been observed in Canada and Western Europe. The primary reason for the higher rates observed in recent years is the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag, 2001; El-Serag and Mason, 2000). Men are at least two to three times more likely to develop liver cancer than women (Yu et al., 2000). Incidence rates are also higher among African Americans than whites. Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn, 1996).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS, 2001b). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al., 2000). However, HBV accounts for only about a quarter of the cases in the U.S. and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS, 2001b).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that causes inflammation and scar tissue to form on the liver, which can often lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al., 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS, 2001b). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS, 2001b). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al., 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan, 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn, 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands, 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan, 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS, 2001b; London and McGlynn, 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS, 2001b). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS, 2001b; ATSDR, 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS, 2001b; London and McGlynn, 1996). Long-term anabolic steroid use may slightly increase the risk of HCC; however, a definitive relationship has not been established (ACS, 2001b; London and McGlynn, 1996). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al., 2000l; London and McGlynn, 1996).

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Appendix B

Cancer Incidence Coding Definitions

Appendix B:
Coding Definitions of Cancer Site/Type*

	ICD-O-1 and Other Pre-ICD-O-2 Codes		*ICD-O-2 Codes*		*ICD-O-3 Codes*	
Cancer Site / Type	***Site code***	***Histology code***	***Site code***	***Histology code***	***Site code***	***Histology code***
Bladder	188.0-188.9	except 9590-9980	C67.0-C67.9	except 9590-9989	C67.0-C67.9	except 9590-9989
Bone	170.0-170.9	except O8010-O8140, O8723, O9391-O9580, O9590-O9980, B9593-B9733	C40.0-C41.9	except 8010-8140, 8723, 9391-9580, 9590-9980	C40.0-C41.9	except 9590-9989
Kidney & Renal Pelvis	189.0, 189.1	except 9590-9980	C64.9, C65.9	except 9590-9989	C64.9, C65.9	except 9590-9989
Leukemia	140.0-199.9	includes O9800-O9943, O9951, P9803-P9943, B9803-B9943	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9800-9822, 9824-9826, 9828-9941 2. includes 9823, 9827	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964 2. includes 9823, 9827
Liver	155.0	except 9590-9980	C22.0	except 9590-9989	C22.0	except 9590-9989
Lung & Bronchus	162.2-162.9	except 9050-9053, 9590-9980	C34.0-C34.9	except 9590-9989	C34.0-C34.9	except 9590-9989
Multiple Myeloma	140.0-199.9	includes O9730-O9732, P9733, B9733	C00.0-C80.9	includes 9731, 9732	C00.0-C80.9	includes 9731, 9732, 9734
Non-Hodgkin's Lymphoma (NHL)	140.0-199.9	includes O9590-O9642, O9670-O9710, O9750, P9593-P9643, P9693-P9713, P9753, B9593-B9643, B9703	1. C00.0-C80.9 AND 2. All sites except C42.0, C42.1, C42.4	1. includes 9590-9595, 9670-9717 2. includes 9823, 9827	1. C00.0-C80.9 AND 2. All sites except C42.0, C42.1, C42.4	1. includes 9590-9596, 9670-9729 2. includes 9823, 9827
Stomach	151.0-151.9	except 9590-9980	C16.0-C16.9	except 9590-9989	C16.0-C16.9	except 9590-9989
Thyroid	193.9	except 9590-9980	C73.9	except 9590-9989	C73.9	except 9590-9989

*Note: Includes invasive tumors only, selected by excluding in situ stages J0, S0, TTISNXM0, TTANXMX, TTANXM0, TTAN0MX, TTISN0M0, TTISNXMX, TTISN0MX, TTISN0M0, TTIN0M0, TTIN0MX, TTINXM0, and TTINXMX (1982-1994 data) or by specifying behavior code (1995-2000 data).

Appendix C

Risk Factor Information for Selected Cancer Types

Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 63,210 people in the U.S. in 2005, accounting for 7% of all cancers diagnosed in the United States among men and 2% among women. In Massachusetts, bladder cancer accounts for approximately 6% of all cancers diagnosed among males and females combined (ACS, 2005). Males are three times more likely to develop bladder cancer than females and whites are two times more likely to develop this disease than blacks. The risk of bladder cancer increases with age and the mean age at diagnosis is 68-69 years (ACS, 2000).

The greatest risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS, 2000). The risk of developing bladder cancer increases with the number of packs smoked per day and with duration of smoking. Further, the risk of bladder cancer may be higher in women than in men who smoke comparable numbers of cigarettes (Castelao et al., 2001). Approximately 25-60% of all bladder cancers can be attributed to tobacco use (Johansson and Cohen, 1997). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30% to 60% (Silverman et al., 1996).

Studies have also revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the U.S. (Johansson and Cohen, 1997). Occupational exposure to aromatic amines, such as benzidine and 2-naphthylamine, increases the risk of bladder cancer (ACS, 2000). These chemicals were common in the dye industry in the past. A higher risk of bladder cancer has also been observed among aromatic amine manufacturing workers as well as among workers in the rubber, leather, textiles, printing, and paint products industries (ACS, 2000; Silverman et al., 1996). The development of new chemicals, changed worker exposures, and the elimination of many known bladder carcinogens in the workplace have caused shifts in those occupations considered to be high risk. For example, risks among dye, rubber, and leather workers have declined over time, while other occupations such as motor vehicle operation (e.g., drivers of trucks, buses, and taxis) and the aluminum industry have emerged as potential high-risk occupations (Silverman et al., 1996). However, specific occupational exposures in these occupations have not been confirmed and study findings are not consistent. Further, the risk of bladder cancer from occupational exposures may be increased among smokers (ACS, 2000).

Dietary factors such as consumption of fried foods as well as foods high in fat and cholesterol have been found to be associated with increased bladder cancer risk (Silverman et al., 1996). Use of the Chinese herb, *Aristocholia fangchi,* found in some dietary supplements, has also been linked with bladder cancer (ACS, 2000). Use of some anti-cancer drugs (e.g., cyclophosphamide and chlornaphazine), use of phenacetin, and infection with *Shistosoma haematobium* (a parasite found in Africa) are thought to be associated with the development of bladder cancer, however, not all epidemiological studies have produced convincing findings (Silverman et al., 1996).

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS, 2000; Silverman et al., 1996). Exposure to chlorinated by-products in drinking water has also been suggested to

increase bladder cancer risk, however, a recent population-based study found that an association was present only among smokers (Cantor et al., 1998).

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Bone Cancer

The occurrence of bone cancer is extremely rare; fewer than 2,600 primary cancers of the bone and joints will be diagnosed in the U.S. in 2005, accounting for less than 0.2% of all cancer types (ACS, 2005). There are several different types of malignant or cancerous bone tumors: osteosarcoma (about 35% of all cases), chondrosarcoma (26%), Ewing's sarcoma (or Ewing's tumor) (16%), chordoma (8%), and malignant fibrous histiocytoma/fibrosarcoma (6%). Several rare types account for the remainder of cases (ACS, 2000). Osteosarcoma affects the bones themselves and primarily occurs in young people between the ages of 10 and 30. However, about 10% of cases develop in people aged 60 to 80. Chondrosarcoma is a cancer of the cartilage cells and is uncommon in people under the age of 20. After age 20, the risk of this disease increases with age. Ewing's sarcomas most often develop in the cavity of the bone and are usually diagnosed in children and adolescents (ACS, 2000). Among the major bone cancer types, males experience a higher incidence of bone cancer than females. Similar incidence rates for osteosarcoma have been observed among whites and blacks. However, in the United States and Africa, the occurrence of Ewing's sarcoma among black individuals is almost non-existent. The incidence of Ewing's sarcoma among the Asian population is also very low (Miller et al., 1996).

Very little is known about factors associated with the development of bone cancer. In fact, most people with bone cancer do not have any known risk factors (ACS, 2000). Several pre-existing medical conditions are associated with the development of certain primary bone cancers. For example, osteosarcomas develop in about 5% to 10% of severe cases of Paget's disease, which primarily affects people over the age of 50 and results in the formation of abnormal bone tissue (ACS, 2000). The presence of multiple exostoses (overgrowth of bone tissue) increases the risk of osteosarcoma, as does the presence of multiple osteochondromas (benign tumors formed by bone and cartilage). In addition, an increased risk of chondrosarcoma has been observed among people with multiple enchondromas (benign cartilage tumors), although this risk is very low (ACS, 2000).

Very few bone cancers appear to have a hereditary basis. However, an elevated risk of developing bone cancer (especially osteosarcoma) has been associated with a family history of Li-Fraumeni syndrome. In addition, children with an inherited form of retinoblastoma, a rare eye cancer, have an increased risk for developing osteosarcoma due to an abnormal mutation of the retinoblastoma gene (ACS, 2000).

Ionizing radiation has been identified as one of the only environmental factors known to play a role in the development of certain types of bone cancer (e.g., osteosarcoma and chondrosarcoma). A typical x-ray of a bone does not pose a significant risk, but exposure to high-dose radiation (e.g., radiation therapy to treat another type of cancer) and ongoing exposure to internally deposited radionuclides (used to treat bone disease or for diagnostic radiography) may increase the risk of bone cancer (ACS, 2000; Miller et al., 1996). Although the use of high dose radiation has been identified as a risk factor, it is likely that less than 0.2% of patients treated develop bone cancer (Miller et al., 1996). However, children with certain cancers seem to be particularly susceptible to radiogenic bone cancer and it appears that radiotherapy may

interact with genetic susceptibility (e.g., due to a mutation in the retinoblastoma gene) (Miller et al., 1996).

Some studies have suggested that injury to a bone can cause cancer, but this has not been corroborated and most doctors do not believe that trauma is a significant risk factor. It is more likely that a diagnosis prompts patients to remember an injury to a site or that an injury draws their attention to a pre-existing bone mass (ACS, 2000).

Limited information is available regarding bone cancer and occupational risk factors (Hoppin et al., 1999). In the past, occupational exposure to radium was found to increase the risk of developing bone cancer. One study revealed that prior to 1930, women employed as radium dial painters in the United States were found to be ingesting radium orally by licking their paintbrushes to produce finer tips for finer lines. Of the 1,474 women in the study, 4% developed bone cancer (Miller et al., 1996). Some studies have suggested that certain woodworking occupations (e.g. carpenters, furniture workers) are associated with increased bone cancer mortality, but findings are inconsistent (Hoppin et al., 1999).

Metal implants (e.g., hip replacement) are also thought to play a causal role in the development of bone cancer. This is thought to be due to the use of metals such as chromium (a known human carcinogen) and nickel (a suspected human carcinogen), or the use of bone cement. While an association between bone cancer and metal implants is suggested, no definitive links in humans have been identified (Miller et al., 1996).

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Brain and Central Nervous System Cancer

Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 50% of all reported primary brain and spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. Therefore, approximately 7.5% of brain and CNS tumors are malignant meningiomas. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the primitive stem cells of the cerebellum and are most often seen in children. Also, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS, 1999a). The American Cancer Society estimates that 18,500 Americans (10,620 men and 7,880 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,760 people (7,280 men and 5,480 women) will die from this disease in 2005 (ACS, 2005).

Brain and spinal cord cancers account for over 20% of all cancer types diagnosed among children aged 0-14 (ACS, 2005). About half of all childhood brain tumors are astrocytomas and 25% are medulloblastomas (ACS, 1999b). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme). Incidence rates are higher in males than in females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack, 1996).

Despite numerous scientific and medical investigations, and analyses, the causes of brain cancer are still largely unknown. Among the possible risk factors investigated in relation to this type of cancer are ionizing radiation, electromagnetic fields, occupational exposures, exposure to N-nitroso compounds, head trauma, and genetic disorders.

The most established risk factor (and only established environmental risk factor) for brain tumors (either cancerous or non-cancerous) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain tumors are caused by radiation to the head from the treatment of other cancers (ACS, 1999a). Meningiomas are the most common type of tumors that occur from this type of exposure, but gliomas may also occur (Preston-Martin and Mack, 1996). Among adults, the risk of developing meningiomas has been associated with full-mouth dental x-rays taken decades ago when radiation doses were higher than today. Although the relationship between low-dose radiation exposure and increased risk of brain tumors has been debated in several studies, prenatal exposure from diagnostic x-rays has been related to an increase in childhood brain tumors (Preston-Martin and Mack, 1996).

In recent years, there has been increasing public concern and scientific interest regarding the relationship of electromagnetic fields (EMF) to brain cancer. However, results from recent epidemiological investigations provide little or no evidence of an association between residential EMF exposure (e.g., from power lines and home appliances) and brain tumors (Kheifets, 2001). Studies also suggest that the use of handheld cellular telephones is not associated with an increased risk of primary brain cancer (Muscat et al., 2000). However, given the relatively recent use of cellular phones, evidence is preliminary and few studies have been conducted.

Other environmental factors such as exposure to vinyl chloride (used in the manufacturing of some plastics) and aspartame (a sugar substitute) have been suggested as possible risk factors for brain cancer but no conclusive evidence exists implicating these factors (ACS, 1999a). Although some occupational studies have suggested that electrical and electric utility workers may be at a slightly increased risk of brain cancer, these studies have important limitations, such as exposure misclassifications and a lack of dose-response relationships (Kheifets, 2001). Some researchers have also reported an increased risk of brain tumors in adults among veterinarians and farmers. Exposures to farm animals and pets have been considered as possible risk factors because of their association with bacteria, pesticides, solvents, and certain animal oncogenic (cancer-related) viruses (Yeni-Komshian and Holly, 2000). However, the relationship between farm life and brain cancer remains controversial.

Recent reports have proposed a link between occupational exposure to lead and brain cancer risk, but further analytic studies are warranted to test this hypothesis (Cocco et al., 1998). In a recent case-control study, the concentrations of metal and non-metal compounds in brain biopsies from patients with primary brain tumors were compared to results from an analysis of tumor-free brain tissue. Statistically significant associations were observed between the presence of brain tumors and the concentrations of silicon, magnesium, and calcium (Hadfield et al., 1998). However, further research using a larger sample size is needed to determine whether exposure to these elements plays a role in the development of brain cancer. Other occupations that may be associated with elevated risks include workers in certain health professions (e.g., pathologists and physicians), agricultural workers, workers in the nuclear industry, and workers in the rubber industry, although specific exposures have not been established (Preston-Martin and Mack, 1996). Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, metal, construction, and electric workers) and the onset of brain tumors in their children have provided inconsistent results (Preston-Martin and Mack, 1996).

The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A recent study concluded that an increased risk of pediatric brain tumor may be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin, 2001). However, the role of nitrites and cured meats in the development of brain cancer remains controversial (Blot et al., 1999; Bunin, 2000). Because most people have continuous, low level exposure to N-nitroso compounds throughout their lives, further studies,

especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin, 1996).

Injury to the head has been suggested as a possible risk factor for later development of brain tumors but most researchers agree that there is no conclusive evidence for an association (ACS, 1999b). Head trauma is most strongly associated with the development of meningiomas compared with other types of brain tumor. Several studies have found an increased risk in women with histories of head trauma; in men who boxed; and in men with a previous history of head injuries. Gliomas are the most common type of childhood brain tumor and have been positively associated with trauma at birth (e.g., Cesarean section, prolonged labor, and forceps delivery). However, other studies have found no association (Preston-Martin and Mack, 1996).

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-Martin and Mack, 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS, 1999b). Of those afflicted with the disease, about 5-10% will develop a central nervous system tumor (Preston-Martin and Mack, 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS, 1999a). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack, 1996).

Other possible risk factors investigated for brain cancer have included alcohol consumption, use of barbiturates, smoking and exposure to second-hand smoke, pesticides, and infectious diseases (i.e., tuberculosis and chicken pox). To date, studies on these risk factors have yielded inconclusive results. Further, the majority of individuals diagnosed with brain cancer have no known risk factors (ACS, 1999a).

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Breast Cancer

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the North American Association of Central Cancer Registries, female breast cancer incidence in Massachusetts is the fifth highest among all states (Chen et al, 2000). Although during the 1980s breast cancer in the U.S. increased by about 4% per year, the incidence has leveled off to about 110.6 cases per 100,000 (ACS 2000). A similar trend occurred in Massachusetts and there was even a slight decrease in incidence (1%) between 1993 and 1997 (MCR 2000).

In the year 2005, approximately 211,240 women in the U.S. will be diagnosed with breast cancer (ACS 2005). Worldwide, female breast cancer incidence has increased, mainly among women in older age groups whose proportion of the population continues to increase as well (van Dijck, 1997). A woman's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon the well-established risk factors for breast cancer. These include increased age, an early age at menarche (menstruation) and/or late age at menopause, late age at first full-term pregnancy, family history of breast cancer, and high levels of estrogen. Other risk factors that may contribute to a woman's risk include benign breast disease and lifestyle factors such as diet, body weight, lack of physical activity, consumption of alcohol, and exposure to cigarette smoke. Data on whether one's risk may be affected by exposure to environmental chemicals or radiation remains inconclusive. However, studies are continuing to investigate these factors and their relationship to breast cancer.

Family history of breast cancer does affect one's risk for developing the disease. Epidemiological studies have found that females who have a first-degree relative with premenopausal breast cancer experience a 3-fold greater risk. However, no increase in risk has been found for females with a first degree relative with postmenopausal breast cancer. If women have a first-degree relative with bilateral breast cancer (cancer in both breasts) at any age then their risk increases five-fold. Moreover, if a woman has a mother, sister or daughter with bilateral premenopausal breast cancer, their risk increases nine fold (Broeders and Verbeek, 1997). In addition, twins have a higher risk of breast cancer compared to non-twins (Weiss et al, 1997).

A personal history of benign breast disease is also associated with development of invasive breast cancer. Chronic cystic or fibrocystic disease is the most commonly diagnosed benign breast disease. Women with cystic breast disease experience a 2-3 fold increase in risk for breast cancer (Henderson et al, 1996).

According to recent studies, approximately 10% of breast cancers can be attributed to inherited mutations in breast cancer related genes. Most of these mutations occur in the BRCA1 and BRCA2 genes. Approximately 50% to 60% of women who inherit BRCA1 or BRCA2 gene mutations will develop breast cancer by the age of 70 (ACS 2001).

Cumulative exposure of the breast tissue to estrogen and progesterone hormones may be one of the greatest contributors to risk for breast cancer (Henderson et al, 1996). Researchers suspect that early exposures to a high level of estrogen, even during fetal development, may add to one's

risk of developing breast cancer later in life. Other studies have found that factors associated with increased levels of estrogen (i.e., neonatal jaundice, severe prematurity, and being a fraternal twin) may contribute to an elevated risk of developing breast cancer (Ekbom et al, 1997). Conversely, studies have revealed that women whose mothers experienced toxemia during pregnancy (a condition associated with low levels of estrogen) had a significantly reduced risk of developing breast cancer. Use of estrogen replacement therapy is another factor associated with increased hormone levels and it has been found to confer a modest (less than two-fold) elevation in risk when used for 10-15 years or longer (Kelsey, 1993). Similarly, more recent use of oral contraceptives or use for 12 years or longer seems to confer a modest increase in risk for bilateral breast cancer in premenopausal women (Ursin et al, 1998).

Cumulative lifetime exposure to estrogen may also be increased by certain reproductive events during one's life. Women who experience menarche at an early age (before age 12) have a 20% increase in risk compared to women who experience menarche at 14 years of age or older (Broeders and Verbeek, 1997; Harris et al, 1992). Women who experience menopause at a later age (after the age of 50) have a slightly elevated risk for developing the disease (ACS 2001). Furthermore, the increased cumulative exposure from the combined effect of early menarche and late menopause has been associated with elevated risk (Lipworth, 1995). In fact, women who have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than women with 30 years or less of menstrual activity (Henderson et al, 1996). Other reproductive events have also shown a linear association with risk for breast cancer (Wohlfahrt, 2001). Specifically, women who gave birth for the first time before age 18 experience one-third the risk of women who have carried their first full-term pregnancy after age 30 (Boyle et al, 1988). The protective effect of earlier first full-term pregnancy appears to result from the reduced effect of circulating hormones on breast tissue after pregnancy (Kelsey, 1993).

Diet, and particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, a hypothesis exists that the type of fat in a woman's diet may be more important than her total fat intake (ACS, 1998; Wynder et al, 1997). Monounsaturated fats (olive oil and canola oil) are associated with lower risk while polyunsaturated (corn oil, tub margarine) and saturated fats (from animal sources) are linked to an elevated risk. However, when factoring in a woman's weight with her dietary intake, the effect on risk becomes less clear (ACS, 1998). Many studies indicate that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey, 1993), probably due to fat tissue as the principal source of estrogen after menopause (McTiernan, 1997). Therefore, regular physical activity and a reduced body weight may decrease one's exposure to the hormones believed to play an important role in increasing breast cancer risk (Thune et al, 1997).

Aside from diet, regular alcohol consumption has also been associated with increased risk for breast cancer (Swanson et al, 1996; ACS, 2001). Women who consumed one alcoholic beverage per day experienced a slight increase in risk (approximately 10%) compared to non-drinkers, however those who consumed 2 to 5 drinks per day experienced a 1.5 times increased risk (Ellison et al., 2001; ACS, 2001). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al, 1996).

To date, no specific environmental factor, other than ionizing radiation, has been identified as a cause of breast cancer. The role of cigarette smoking in the development of breast cancer is unclear. Some studies suggest a relationship between passive smoking and increased risk for breast cancer; however, confirming this relationship has been difficult due to the lack of consistent results from studies investigating first-hand smoke exposure (Laden and Hunter, 1998).

Studies on exposure to high doses of ionizing radiation demonstrate a strong association with breast cancer risk. These studies have been conducted in atomic bomb survivors from Japan as well as patients that have been subjected to radiotherapy in treatments for other conditions (i.e., Hodgkin's Disease, non-Hodgkin's Lymphoma, tuberculosis, post-partum mastitis, and cervical cancer) (ACS, 2001). However, it has not been shown that radiation exposures experienced by the general public or people living in areas of high radiation levels, from industrial accidents or nuclear activities, are related to an increase in breast cancer risk (Laden and Hunter, 1998). Investigations of electromagnetic field exposures in relation to breast cancer have been inconclusive as well.

Occupational exposures associated with increased risk for breast cancer have not been clearly identified. Experimental data suggests that exposure to certain organic solvents and other chemicals (e.g., benzene, trichloropropane, vinyl chloride, polycyclic aromatic hydrocarbons (PAHs)) causes the formation of breast tumors in animals and thus may contribute to such tumors in humans (Goldberg and Labreche, 1996). Particularly, a significantly elevated risk for breast cancer was found for young women employed in solvent-using industries (Hansen, 1999). Although risk for premenopausal breast cancer may be elevated in studies on the occupational exposure to a combination of chemicals, including benzene and PAHs, other studies on cigarette smoke (a source of both chemicals) and breast cancer have not shown an associated risk (Petralia et al, 1999). Hence, although study findings have yielded conflicting results, evidence does exist to warrant further investigation into the associations.

Other occupational and environmental exposures have been suggested to confer an increased risk for breast cancer in women, such as exposure to polychlorinated biphenyls (PCBs), chlorinated hydrocarbon pesticides (DDT and DDE), and other endocrine-disrupting chemicals. Because these compounds affect the body's estrogen production and metabolism, they can contribute to the development and growth of breast tumors (Davis et al, 1997; Holford et al, 2000; Laden and Hunter, 1998). However, studies on this association have yielded inconsistent results and follow-up studies are ongoing to further investigate any causal relationship (Safe, 2000).

When considering a possible relationship between any exposure and the development of cancer, it is important to consider the latency period. Latency refers to the time between exposure to a causative factor and the development of the disease outcome, in this case breast cancer. It has been reported that there is an 8 to 15 year latency period for breast cancer (Petralia, 1999; Aschengrau, 1998; Lewis-Michl, 1996). That means that if an environmental exposure were related to breast cancer, it may take 8 to 15 years after exposure to a causative factor for breast cancer to develop.

Socioeconomic differences in breast cancer incidence may be a result of current screening participation rates. Currently, women of higher socioeconomic status (SES) have higher screening rates, which may result in more of the cases being detected in these women. However, women of higher SES may also have an increased risk for developing the disease due to different reproductive patterns (i.e., parity, age at first full-term birth, and age at menarche). Although women of lower SES show lower incidence rates of breast cancer in number, their cancers tend to be diagnosed at a later stage (Segnan, 1997). Hence, rates for their cancers may appear lower due to the lack of screening participation rather than a decreased risk for the disease. Moreover, it is likely that SES is not in itself the associated risk factor for breast cancer. Rather, SES probably represents different patterns of reproductive choices, occupational backgrounds, environmental exposures, and lifestyle factors (i.e., diet, physical activity, cultural practices) (Henderson et al, 1996).

Despite the vast number of studies on the causation of breast cancer, known factors are estimated to account for less than half of breast cancers in the general population (Madigan et al, 1995). Researchers are continuing to examine potential risks for developing breast cancer, especially environmental factors.

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Hodgkin's disease

Hodgkin's disease (or Hodgkin's lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin's lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,350 new cases of this disease in the U.S. in 2005, accounting for less than 1% of all cancer types, and approximately 1,410 deaths (ACS, 2005). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS, 1999).

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women and more common among whites than blacks. People of Jewish descent appear to be at higher risk of Hodgkin's disease compared to people of non-Jewish descent (Mueller, 1996). Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 to 40 (typically ages 25-30) and the second peak occurs in adults aged 55 years and above.

No major risk factors for Hodgkin's disease have been found (ACS, 1999). However, the clinical and cellular features of Hodgkin's disease suggest a chronic infectious process (Mueller, 1996). The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. Researchers have proposed that among young adults, Hodgkin's disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is probably similar to those of other lymphomas (Mueller, 1996). The virus that has been linked most specifically to this disease is the Epstein-Barr virus (EBV). EBV, a herpesvirus, is common in the general population and causes mononucleosis or "mono." Approximately 40% to 50% of Hodgkin's disease cases are associated with EBV (Weiss, 2000). In addition, several studies have also shown that young adults who have developed infectious mononucleosis have a significantly higher risk of developing Hodgkin's disease (ACS, 1999). However, the absence of EBV infection in about half the cases and the high prevalence of EBV in the general population suggest that EBV may be only one of several factors in the development of this cancer. Although cytomegalovirus (CMV) and the more recently identified human herpesvirus type 6 have been considered as possible factors in the development of Hodgkin's disease, results of antibody studies are inconsistent and these viruses do not appear to be related to risk of Hodgkin's disease (Mueller, 1996).

Slightly higher rates of Hodgkin's disease occur among people with reduced immunity, such as those with AIDS, people with congenital immune deficiencies, and individuals on immunosuppressant medication following organ transplants. However, Hodgkin's disease occurs at a much lower rate than non-Hodgkin's lymphomas among this group of individuals (ACS, 1999).

Hodgkin's disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin's disease is associated with an infectious

agent (Mueller, 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller, 1996). Based on an examination of medical and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin's disease does not seem to be caused by genetic, lifestyle (e.g., dietary), or environmental factors (ACS, 1999).

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Kidney Cancer

Kidney cancer involves a number of tumor types located in various areas of the kidney and renal system. Renal cell cancer (which affects the main area of the kidney) accounts for over 90% of all malignant kidney tumors (ACS, 2001). The American Cancer Society estimates that there will be approximately 36,160 cases of kidney and upper urinary tract cancer, resulting in more than 12,660 deaths in 2005 (ACS, 2004). The incidence and mortality from kidney cancer is higher in urban areas, which may be due to increased access to diagnostic services and other factors such as smoking. Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in the fifth and sixth decades of life (50-70 year age group) (ACS, 2001). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

Since 1970, U.S. incidence rates for renal cell cancer have risen between 2 and 4% annually among the four major race and gender groups (i.e., white males, white females, black males, and black females) (Chow et al., 1999; McLaughlin et al., 1996). Rapid increases in incidence among blacks as compared to among whites have resulted in an excess of the disease among blacks; age-adjusted incidence rates between 1975 and 1995 for white men, white women, black men, and black women were 9.6, 4.4, 11.1, and 4.9 per 100,000 person-years, respectively (Chow et al., 1999). Rising incidence rates may be partially due to the increased availability of screening for kidney cancer.

The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 30% to 100% (ACS, 2001). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of those in women may be caused by cigarette smoking (ACS, 2001).

Virtually every study that has examined body weight and renal cell cancer has observed a positive association. Some studies suggest that obesity is a factor in 20% of people who develop kidney cancer (ACS, 2001). This is especially true among women and researchers suspect that this may be related to changes in certain hormones, such as estrogen in women (ACS, 2001; McLaughlin et al., 1996). A diet high in protein (meat, animal fats, milk products, margarine and oils) has been implicated in epidemiological studies as a risk factor for renal cell carcinoma (ACS, 2001; McLaughlin et al., 1996). Consumption of adequate amounts of fruits and vegetables lowers the risk of renal cell cancer. In addition, use of diuretics and antihypertensive medications are associated with increased risk of renal cell carcinoma. However, hypertension has also been linked to kidney cancer and it is not clear whether the disease or the medications used to treat them is the cause (ACS, 2001). Long-term use of pain relievers such as phenacetin (and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (ACS, 2001).

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among

patients on long-term dialysis for renal failure (Linehan et al., 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS, 2001; McLaughlin et al., 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS, 2001; Linehan et al,. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS, 2001). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al., 1997; McLaughlin et al., 1996).

Wilms' tumor is the most common type of kidney cancer affecting children and accounts for approximately 5% to 6% of all kidney cancers and about 6% of all childhood cancers. This cancer is more common among African Americans than other races and among females than males. Wilms' tumor most often occurs in children under the age of 5 years. The causes of Wilms' tumor are not known, but certain birth defect syndromes and other genetic risk factors (such as family history or genetic mutations) are connected with this cancer. However, most children who develop Wilms' tumor do not have any known birth defects or inherited gene changes. No environmental risk factors, either before or after a child's birth, have been shown to be associated with the development of Wilms' tumor (ACS, 1999).

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Leukemia

Leukemia is the general term that includes a group of different cancers that occur in the blood forming organs and result in the formation of abnormal amounts and types of white blood cells in the blood and bone marrow. Individuals with leukemia generally maintain abnormally high amounts of leukocytes or white blood cells in their blood. This condition results in an individual's inability to maintain certain body functions, particularly a person's ability to combat infection.

In 2005, leukemia is expected to affect approximately 34,810 individuals (19,640 males and 15,420 females) in the United States, resulting in 22,570 deaths. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2005, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML and CLL. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority of these cases are of the ALL type (ACS, 2005).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals. The increase in incidence among older individuals begins at approximately 40-50 years of age, peaking at about age 85 (Linet and Cartwright, 1996). ALL is more common among whites than African Americans and among males than females (Weinstein and Tarbell, 1997). Exposure to high-dose radiation (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents) is a known environmental risk factor associated with the development of ALL (Scheinberg et al., 1997). Significant radiation exposure (e.g., diagnostic x-rays) before birth may carry up to a 5-fold increased risk of developing ALL (ACS 2000b). However, few studies report an increased risk of leukemia associated with residing in proximity to nuclear plants or occupational exposure to low-dose radiation (Linet and Cartwright, 1996; Scheinberg et al., 1997). It is unclear whether exposure to electromagnetic fields (EMF) plays a role in the development of ALL, however, most studies to date have found little or no risk (ACS 2000b).

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell, 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2000b). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al., 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS, 2000a). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection.

However, no specific virus has been identified as related to ALL (Linet and Cartwright, 1996). Recent reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care do not support this hypothesis (Greaves MF, 1997; Kinlen and Balkwill, 2001; Rosenbaum et al., 2000).

Although AML can occur in children (usually during the first two years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS, 2000a and 2000b). This type of leukemia is more common among males than among females but affects African Americans and whites at similar rates (Scheinberg et al., 1997). High-dose radiation exposure (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents), long-term occupational exposure to benzene, and exposure to certain chemotherapy drugs, especially alkylating agents (e.g., mechlorethamine, cyclophosphamide), have been associated with an increased risk of developing AML among both children and adults (ACS, 2000a and 2000b; Linet and Cartwright, 1996). The development of childhood AML is suspected to be related to parental exposure to pesticides and other chemicals, although findings are inconsistent (Linet and Cartwright, 1996). Recent studies have suggested a link between electromagnetic field (EMF) exposure (e.g., from power lines) and leukemia (Minder and Pfluger, 2001; Schuz et al., 2001). However, there is conflicting evidence regarding EMF exposure and leukemia and it is clear that most cases are not related to EMF (ACS, 2000a; Kleinerman et al., 2000).

Other possible risk factors related to the development of AML include cigarette smoking and genetic disorders. It is estimated that approximately one-fifth of cases of AML are caused by smoking (Scheinberg et al., 1997). Also, a small number of AML cases can be attributed to rare inherited disorders. These include Down's syndrome in children, Fanconi's anemia, Wiskott-Aldrich syndrome, Bloom's syndrome, Li-Fraumeni syndrome, and ataxia telangiectasia (ACS, 2000a and 2000b). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al., 2001).

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 1999). Twice as many men as women are affected by this type of leukemia (Deisseroth et al., 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS, 1999; Weinstein and Tarbell, 1997). It is thought that individuals with a family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright, 1996). In addition, many researchers believe that cigarette smoking plays a role in some chronic leukemias. The role of EMF in the development of chronic leukemia remains controversial (ACS, 1999). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al., 1997).

Of all the leukemias, CML is among the least understood. While this disease can occur at any age, CML is extremely rare in children (about 2% of leukemias in children) and the average age

of diagnosis is 40 to 50 years (ACS 1999). Incidence rates are higher in males than in females, but unlike the other leukemia types, rates are higher in blacks than in whites in the U.S. (Linet and Cartwright, 1996). High-dose radiation exposure may increase the risk of developing CML (ACS, 1999). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell, 1997).

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Liver Cancer

An estimated 17,550 people in the U.S. (12,130 men and 5,420 women) will be diagnosed with liver cancer in 2005, accounting for approximately 1% of all new cancers (ACS, 2005). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Rarer forms of malignant liver cancer include cholangiocarcinomas, angiosarcomas, and hepatoblastomas in children. Although HCC is approximately ten times more common in developing countries in East and Southeast Asia and Africa, incidence is rapidly increasing in the United States (ACS, 2001). Rates of HCC in the U.S. have increased by 70% over the past two decades (Yu et al., 2000). Similar trends have been observed in Canada and Western Europe. The primary reason for the higher rates observed in recent years is the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag, 2001; El-Serag and Mason, 2000). Men are at least two to three times more likely to develop liver cancer than women (Yu et al., 2000). Incidence rates are also higher among African Americans than whites. Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn, 1996).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS, 2001). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al., 2000). However, HBV accounts for only about a quarter of the cases in the U.S. and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS, 2001).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that causes inflammation and scar tissue to form on the liver, which can often lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al., 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS, 2001). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS, 2001). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al., 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan, 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn, 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands, 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be

exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan, 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS, 2001; London and McGlynn, 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS, 2001). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS, 2001; ATSDR, 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS, 2001; London and McGlynn, 1996). Long-term anabolic steroid use may slightly increase the risk of HCC; however, a definitive relationship has not been established (ACS, 2001; London and McGlynn, 1996). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al., 2000; London and McGlynn, 1996).

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Lung Cancer

Lung cancer generally arises in the epithelial tissue of the lung. Several different histologic or cell types of lung cancer have been observed. The various types of lung cancer occur in different regions of the lung and each type is associated with slightly different risk factors (Blot and Fraumeni 1996). The most common type of lung cancer in the United States today is adenocarcinoma which accounts for about 40% of all lung cancers (ACS, 2000). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Lung cancer is very rare in people under the age of 40. The incidence is greater among men than women (probably because men are more likely to be smokers than women) and among blacks than whites (Blot and Fraumeni, 1996). The American Cancer Society estimates that lung cancer will be diagnosed in 172,570 people in the U.S. in 2005, accounting for about 13% of all cancers (ACS, 2005). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS, 2000). In Massachusetts, incidence rates in 1997 were 76.7 per 100,000 and 49.2 per 100,000 for males and females, respectively (MCR, 2000). Nationwide, the incidence rate declined significantly in men during the 1990s, most likely as a result of decreased smoking rates over the past 30 years. Rates for women have continued to increase, but at a much slower pace and have begun to level off. This is because decreasing smoking patterns among women have lagged behind those of men (ACS, 2005). Trends in lung cancer incidence suggest that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals have higher rates of smoking than individuals of other groups (Blot and Fraumeni 1996).

More than 80% of all lung cancers are caused directly by smoking cigarettes and many of the rest are due to exposure to second hand smoke, or environmental tobacco smoke. The longer a person has been smoking and the higher the number of cigarettes smoked per day, the greater the risk of lung cancer. Smoking cessation decreases the elevated risk by about 50%, however, former smokers still carry a greater risk than those who have never smoked (ACS, 2000).

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS, 2000). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (ACS, 2000; Samet and Eradze, 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni, 1996; Pohlablen et al., 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS, 2000; Blot and Fraumeni, 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al., 2001; Steenland and

Sanderson, 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni, 1996).

As noted above, exposure to radon (a naturally occurring radioactive gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer among miners. Recently, a number of studies have demonstrated that exposure to elevated levels of residential radon may also increase lung cancer risk (Lubin and Boice, 1997; Kreienbrock et al., 2001; Tomasek et al., 2001). Epidemiological evidence suggests that radon may be the second leading cause of lung cancer after smoking (Samet and Eradze, 2000). However, actual lung cancer risk is determined by cumulative lifetime exposure to indoor radon. Therefore, normal patterns of residential mobility suggest that most people living in high-radon homes experience lifetime exposures equivalent to residing in homes with lower radon levels (Warner et al., 1996).

Tuberculosis and some types of pneumonia may increase the risk of lung cancer due to scarred lung tissue (ACS, 2000). In addition, people who have had lung cancer have a higher risk of developing another tumor. A family history of lung cancer may also slightly increase the risk, however, it is unclear whether this is due to inherited factors or environmental tobacco smoke (ACS, 2000).

Air pollution may increase the risk of developing lung cancer, however, this risk is much lower than that due to cigarette smoking (ACS, 2000).

Diet has also been implicated in the etiology of lung cancer, however, the exact relationship is unclear. Diets high in fruits and vegetables decrease lung cancer risk, but the reasons for this are unknown (Brownson et al., 1998). A recent study showed a positive association between total fat, monounsaturated fat, and saturated fat and lung cancer among males, however, this effect was not observed among women (Bandera et al., 1997).

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Multiple Myeloma

Multiple myeloma is a cancer of the plasma cells. Plasma cells are usually found in the bone marrow and produce immunoglobins or antibodies that circulate in the blood to help in fighting disease. In the United States, multiple myeloma will affect approximately 15,980 people (8,600 men and 7,380 women) in 2005, accounting for approximately 1% of all cancers (ACS, 2005). Among Massachusetts' males, multiple myeloma occurred at a rate of 4.1 cases per 100,000 in 1998. Multiple myeloma occurred at a slightly lower rate among females in Massachusetts in 1998: 3.1 cases per 100,000. This cancer type accounts for about 1% of all cancers diagnosed in Massachusetts (MCR, 2001). For reasons that remain unknown, multiple myeloma is about twice as common among African Americans as whites. The onset of the disease generally occurs late in life and the average age at diagnosis is 70 (ACS, 1999).

The exact causes of multiple myeloma remain largely unknown, however, a number of potential risk factors have been suggested. Besides age and race, the most well established risk factors for multiple myeloma include the presence of pre-existing medical conditions and exposure to ionizing radiation (ACS, 1999). Pre-existing medical conditions such as monoclonal gammopathy of unknown significance (MGUS) increase a person's likelihood of developing multiple myeloma (ACS, 1999; Herrinton et al., 1996). MGUS is an asymptomatic, non-cancer disorder that causes production of certain components within the immune system and proliferation of plasma cells but usually has no impact on a person's health (Herrinton et al., 1996). However, about 20% of people with MGUS will eventually develop multiple myeloma (ACS, 1999). In addition, some patients with solitary plasmacytomas (a tumor formed by myeloma cells that have collected in only one bone) or extramedullary plasmacytomas (localized plasma cell neoplasms which arise within the soft tissues) eventually develop multiple myelomas (ACS, 1999). Some case reports have suggested an increase in the risk of multiple myeloma after prolonged stimulation of the immune system by repeated infection, allergic conditions, or autoimmune disease. However, experimental evidence to support this hypothesis is lacking (Herrinton et al., 1996).

Although it accounts for a very small number of cases, exposure to ionizing radiation is an important risk factor for multiple myeloma (ACS, 1999). Increases in the incidence of multiple myeloma among atomic bomb survivors have provided the most evidence of an association between radiation exposure and this cancer. Occupational exposure to x-rays and radioactive materials (e.g., by medical radiology workers and nuclear power plant workers) may also increase the risk for multiple myeloma (Herrinton et al., 1996). At this time, however, scientists do not have clear evidence that large numbers of medical x-rays increase the risk for multiple myeloma (NCI, 2000).

Certain occupational exposures have been suggested to carry an increased risk of multiple myeloma; however, the actual role of occupational exposures in the development of this cancer remains unclear. Studies have consistently linked a greater multiple myeloma risk with agricultural occupations, presumably due to exposures to pesticides (such as dioxins) (Herrinton et al., 1996; Schwartz, 1997; Nanni et al., 1998). Other studies have shown associations between the risk of multiple myeloma and employment in paint manufacturing, rubber and plastics manufacturing, and metal industries. Among chemical agents, some studies have reported

positive associations between exposure to asbestos, pesticides, engine exhaust, metals, and paints and solvents and an increased risk of multiple myeloma (Herrinton et al., 1996).

Workers in certain petroleum-related industries may also be at a higher risk (ACS, 1999; Nillson et al., 1998). For these workers, exposure to benzene, a known carcinogen, has been suggested as a possible cause. However, published literature on the relationship between benzene exposure and multiple myeloma is inconclusive and does not indicate that exposure to benzene or other petroleum products is a risk factor for this disease (Bergsagel et al., 1999).

The occurrence of multiple myeloma among siblings and other family members suggests that family history may play a role in the development of this cancer. A recent population-based case-control study found that the risk of multiple myeloma was significantly elevated for subjects who reported that a first-degree relative had the disease. Increased risk was also associated with a family history of certain types of cancers such as leukemia and lymphomas (Brown et al., 1999).

A number of viruses have been linked as triggers or cofactors for multiple myeloma. Recently, researchers have linked infection with Kaposi's sarcoma-associated herpesvirus (also called human herpesvirus-8 or HHV-8) with multiple myeloma (Goedert et al., 1998). In fact, the virus has been found in the blood of most patients with this disease. However, more studies are needed to confirm this possible association (ACS, 1999). An increased risk of multiple myeloma has also recently been linked to obesity (Brown et al., 2001). While several studies have indicated a link between smoking and the risk of multiple myeloma, more recent studies provide no evidence of a relationship (Adami et al., 1998). Finally, although use of permanent dark hair dye has been suggested as a risk factor for multiple myeloma previously, recent studies indicate that it is unlikely to be a major contributor to the incidence of this disease (Altekruse et al., 1999).

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Lymphoma

Lymphomas are cancers involving the cells of the lymphatic system. The majority of lymphomas involve the lymph nodes and spleen but the disease may also affect other areas within the body. Non-Hodgkin's lymphoma (NHL) is a classification of all lymphomas except Hodgkin's disease. Thus NHL is a mixed group of diseases that is characterized by the malignant increase in specific cells of the immune system (B or T lymphocytes). B-cell lymphomas are more common than T-cell lymphomas, accounting for about 85% of all cases of NHL (ACS, 2003). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller, 1996). NHL can occur at all ages, however, the average age at diagnosis is in the early 60s and the incidence of this disease generally increases with age. This disease is more common in men than in women and affects whites more often than African Americans or Asian Americans (ACS, 2003). The American Cancer Society estimates that approximately 56,390 Americans will be diagnosed with NHL in 2005, making it the fifth most common cancer in the U.S. among women and the sixth most common cancer among men, excluding non-melanoma skin cancers (ACS, 2005).

Overall, between 1973 and 1997, the incidence of NHL in the U.S. grew 81% (Garber, 2001), although during the 1990s, the rate of increase appears to have stabilized (ACS, 2005). In Massachusetts, the incidence of NHL increased 50% during 1982-1997 from 10.5 cases per 100,000 to 15.7 cases per 100,000 (MCR, 1997 and 2000). The increase in NHL incidence has been attributed to better diagnosis, greater exposure to causative agents, and, to a lesser extent, the increasing incidence of AIDS-related lymphomas (Devesa and Fears, 1992; Scherr and Mueller, 1996). Although the primary factors related to the development of NHL include conditions that suppress the immune system, viral infections, and certain occupational exposures, these factors are thought to account for only a portion of the increase observed in this cancer type (Scherr and Mueller, 1996). The observation that the rate of increase is declining for NHL may be attributed in part to increased use of antiretroviral therapy to slow HIV progression (Wingo et al., 1998).

NHL is more common among people who have abnormal or compromised immune systems, such as those with inherited diseases that suppress the immune system, individuals with autoimmune disorders, and people taking immunosuppressant drugs following organ transplants. Genetic predisposition (e.g., inherited immune deficiencies) only accounts for a small proportion of NHL cases (Scherr and Mueller, 1996). AIDS patients have a 100- to 300-fold higher risk for NHL than the general population (again, these cases account for only a minor part of overall NHL incidence) (Garber, 2001). NHL has also been reported to occur more frequently among individuals with conditions that require medical treatment resulting in suppression of the immune system, such as cancer chemotherapy. However, current evidence suggests that the development of NHL is related to suppression of the individual's immune system as a result of treatment, rather than the treatment itself (Scherr and Mueller, 1996).

Several viruses have been shown to play a role in the development of NHL. Among organ transplant recipients, suppression of the immune system required for acceptance of the transplant leads to a loss of control or the reactivation of viruses that have been dormant in the body (e.g., Epstein-Barr Virus [EBV] and herpesvirus infections). In addition, because cancer-causing viruses

are known to cause lymphomas in various animals, it has been proposed that these types of viruses may also be associated with the development of NHL among humans without compromised immune systems. Infection with the human T-cell leukemia/lymphoma virus (HTLV-I) is known to cause T-cell lymphoma among adults. However, this is a relatively rare infection and most likely contributes only a small amount to the total incidence of NHL (Scherr and Mueller, 1996). EBV infection is common among the general population and has been shown to play a role in the development of most cases of transplant and AIDS related NHL. The combination of immune system deficiencies and EBV infection may cause some people to develop NHL (ACS, 2003). Although viruses are causal factors for some subtypes of NHL, to date, studies have shown that the role of EBV in the development of NHL in the general population may not be large (Scherr and Mueller, 1996). Moreover, the high prevalence of EBV in the general population suggests that EBV may be only one of several factors in the development of this cancer.

Recent studies have found that a type of bacteria, *Helicobacter pylori*, a common cause of stomach ulcers, can also cause some lymphomas of the stomach (ACS, 2003). An important implication of this finding is that treatment with antibiotics could prevent some NHL of the stomach.

Some occupations have been associated with an increased risk of developing NHL, such as occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm, 1990 and 1993; Tatham et al., 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and organophosphate insecticides (Wigle et al., 1990; Zahm et al., 1990; Zahm et al., 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD alone have not demonstrated an increased risk of NHL (Scherr and Mueller, 1996). An elevated risk for NHL development has also been noted among fence workers, orchard workers, and meat workers. High-dose exposure to benzene has been associated with NHL (ACS, 2003), however, a recent international cohort study indicated that petroleum workers exposed to benzene were not at an increased risk of NHL (Wong and Raabe, 2000).

In addition, epidemiological studies of long-term users of permanent hair coloring products have suggested an increased incidence of NHL (Zahm et al., 1992; Scherr and Mueller, 1996). However, a recent population based study found no association between the use of hair color products and an increased risk of developing NHL. The researchers further stated that results from this study and previous studies, including experimental animal studies, provide little convincing evidence linking NHL with normal use of hair dye (Holly et al., 1998).

Although radiation (e.g., nuclear explosions or radioactive fallout from reactor accidents) has been implicated in the development of some cancers, including NHL (ACS, 2003), there is little evidence for an increased risk of lymphoma due to radiation (Scherr and Mueller, 1996).

Recent studies have suggested that contamination of drinking water with nitrate may be associated with an increased risk of NHL (Ward et al., 1996). Nitrate forms N-nitroso compounds which are

known carcinogens and can be found in smoked or salt-dried fish, bacon, sausages, other cured meats, beer, pickled vegetables, and mushrooms.

Smoking has also been suggested to increase the risk of NHL. A study that evaluated the history of tobacco use and deaths from NHL determined that people who had ever smoked had a two-fold increase of dying from NHL as compared to those who never smoked. Further, a four-fold increase was found among the heaviest smokers (Linet et al., 1992). In addition, a more recent study that primarily examined occupation and NHL risk found a significant association with high levels of cigarette smoking and all NHL types (Tatham et al., 1997). However, a recent review of 5 cohort studies and 14 case-control studies concludes that results of epidemiological studies have been inconsistent and that smoking has not been determined to be a definitive risk factor in the development of NHL (Peach and Barnett, 2000).

A recent Danish study has linked the use of tricyclic and tetracyclic antidepressants to NHL, however, more research is needed on this possible association (Dalton et al., 2000).

Although NHL is associated with a number of risk factors, the causes of this disease remain unknown. Most patients with NHL do not have any known risk factors (ACS, 2003).

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Pancreatic Cancer

The American Cancer Society estimates that approximately 32,180 people in the U.S. (16,100 men and 16,080 women) will develop pancreatic cancer in 2005. This disease accounts for approximately 2% of all new cases of cancer in both men and women, but between 5% and 6% of all cancer deaths (ACS, 2005). This discrepancy has been attributed to detection of pancreatic cancer at an advanced stage and the short median survival time for this cancer of approximately three months. Between 1920 and 1965, mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people. These increases are believed to be due, in part, to improved diagnosis during this time period (Anderson et al., 1996). However, over the past 25 years, incidence rates have declined slowly but consistently in men and a slight decline in rates among women has been observed since the mid-1980s. Further, since about 1975, men have experienced a slight decrease in mortality from pancreatic cancer, although rates among women have not dropped (ACS, 2005). The risk of developing pancreatic cancer increases with age and the majority of cases occur between age 60 and 80. Men are approximately 30% more likely to develop pancreatic cancer than are women (ACS, 2000).

Very little is known about what causes pancreatic cancer and how to prevent it. However, a number of risk factors have been identified. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS, 2000). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al., 1996).

Certain medical conditions, such as chronic pancreatitis, diabetes mellitus, and cirrhosis, have been associated with pancreatic cancer, but the reasons for these associations are largely unknown (ACS, 2000). More recently, a possible role for the bacteria *Helicobacter pylori,* which causes ulcers and some gastric cancers, has been suggested in the development of pancreatic cancer (Stolzenberg-Solomon et al., 2001).

There is also some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer may be associated with animal protein and fat consumption as evidenced by higher rates of this cancer in countries whose populations eat a diet high in fat (ACS, 2005). Decreased risks for the disease are usually associated with fruit and vegetable consumption (ACS, 2000). Obesity is also a risk factor for pancreatic cancer (ACS, 2000). Although older studies suggested that coffee and alcohol consumption may be risk factors, more recent studies do not support this association (Michaud et al., 2001).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS, 2000; Ji et al., 2001; Porta et al., 1999). Exposure to certain dyes and certain chemicals related to gasoline, in addition to asbestos and ionizing radiation, have also been associated with the development of pancreatic cancer in some studies, however, other studies have found no link between these agents and pancreatic cancer (ACS, 2000; Anderson et al., 1996). A recent

evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this claim (Ojajarvi et al., 2000). Although occupational exposures may have played a role in the incidence of this cancer in the past, currently most newly diagnosed patients with pancreatic cancer do not have evidence of a specific chemical exposure or relevant occupational history (Evans et al., 1997).

Finally, pancreatic cancer seems to run in some families. According to the American Cancer Society, an inherited tendency to develop pancreatic cancer may account for approximately 5% to 10% of cases (ACS, 2000). Pancreatic cancer has been observed in both familial clusterings among siblings as well as in individuals of consecutive generations (Anderson et al., 1996).

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Thyroid Cancer

The American Cancer Society estimates that thyroid cancer will affect 30,180 people in the U.S. in 2006, accounting for 3% of all cancers diagnosed in the United States among females and 1% among males (ACS 2006). In Massachusetts, thyroid cancer accounts for approximately 1.6% of all cancers diagnosed among males and females combined (MCR 2005). Females are three times more likely to develop thyroid cancer than males. The risk of thyroid cancer is highest among individuals between the ages of 20 and 55. A 2% annual increase in the incidence of thyroid cancer in the U.S. is occurring, making thyroid cancer one of the few cancers that has an increasing incidence (ACS 2006). The prognosis for most thyroid cancers is extremely good with a five-year survival rate of approximately 97% (Ries 2004).

There are several different subtypes of thyroid cancer. Eighty percent of thyroid cancers are of the papillary carcinoma subtype. The second most common subtype is follicular carcinoma of the thyroid (10% of thyroid cancers). Other subtypes of thyroid cancer include medullary thyroid carcinoma (3%) and anaplastic carcinoma (2%) (ACS 2006). While thyroid cancer is one of the most common cancers for individuals below 40 years of age, each subtype of thyroid cancer has a different age-specific incidence pattern. Papillary carcinoma has a peak in incidence between 45 and 55 years of age, while follicular carcinomas have a peak in incidence among individuals around the age of 60. Anaplastic carcinomas are rare in individuals under 50, but the incidence increases after 50 years of age (Hall and Adami 2002). Each subtype of thyroid cancer may have different risk factors associated with its development (ACS 2006).

Ionizing radiation is the only established risk factor for thyroid cancer. The earliest indication of radiation exposure causing thyroid cancer occurred in the early part of the 20th century when radiation was used to treat many different diseases of childhood. Numerous epidemiological investigations have looked at several groups of individuals treated with radiation in the early 20th century: children with ringworm of the scalp, infants with enlarged thymus glands, adolescents with enlarged tonsils, children with cancer, young adults with Hodgkin's disease, patients given whole-body irradiation, and women treated for cervical cancer. These groups all experienced an elevated incidence of thyroid cancer (Hall and Adami 2002). There is also a marked increase in the incidence of thyroid cancer among atomic bomb survivors in Japan. Presently, exposure to ionizing radiation is limited in the United States. Individuals receiving treatment for certain cancers may receive ionizing radiation. Also, certain occupations may expose individuals to ionizing radiation on a regular basis. However, data on the occupational risks of ionizing radiation are inconclusive.

Exposure to ionizing radiation in childhood appears to be more strongly linked with the development of thyroid cancer than exposure in adulthood. For thyroid cancer the latency period (i.e., the time period between exposure to an environmental risk factor and the development of clinically significant disease) is thought to be 10 to 25 years or longer (Upton 1998).

Approximately 3% of individuals diagnosed with thyroid cancer have a family history of the disease. Individuals with a genetic predisposition for thyroid cancer are more likely to develop the medullary thyroid carcinoma subtype (Ron 1996). Familial thyroid cancer is also more

aggressive in nature than sporadic (non-familial) thyroid cancer. Individuals with certain inherited medical conditions are also at higher risk of thyroid cancer. Higher rates of thyroid cancer occur among people with conditions such as Gardner syndrome and familial polyposis. These conditions also increase a person's risk for developing colorectal cancer as well as other types of cancer (ACS 2006).

Few other risk factors for thyroid cancer are known. A diet low in iodine may increase the risk of follicular carcinomas (ACS 2006). However, this is not generally considered a cause of thyroid cancer among individuals in the U.S. as salt in the United States is fortified with iodine.

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Appendix D

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

General Terms

Absorption
The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Anaerobic
Requiring the absence of oxygen [compare with aerobic].

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP [see Community Assistance Panel.]

Cancer
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

CERCLA [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure]

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)
A group of people from a community and from health and environmental agencies who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Delayed health effect
A disease or an injury that happens as a result of exposures that might have occurred in the past.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see route of exposure].

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease prevention
Measures used to prevent a disease or reduce its severity.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

DOD
United States Department of Defense.

DOE
United States Department of Energy.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction
A method of estimating the amount of people's past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Half-life (t½)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remain.

Hazard
A source of potential harm from past, current, or future exposures.

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to evaluate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.

Health statistics review
The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

mg/cm2
Milligram per square centimeter (of a surface).

mg/m3
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, a condition, or an injury) is stated.

Mutagen
A substance that causes mutations (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see National Priorities List for Uncontrolled Hazardous Waste Sites]

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevalence survey
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public availability session
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might

be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Public health statement
The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public health surveillance
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [see Resource Conservation and Recovery Act (1976, 1984)]

Receptor population
People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)
This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA
RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or an environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Stakeholder
A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Surveillance [see public health surveillance]

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen
A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)

National Center for Environmental Health (CDC)
(http://www.cdc.gov/nceh/dls/report/glossary.htm)

National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

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