Massachusetts
Department Of
Public Health

Evaluation of Health Outcome Data in Northampton and Easthampton, MA and among Neighborhoods in Closest Proximity to the Northampton Regional Landfill

September 2008

Bureau of
Environmental Health,
Community Assessment
Program

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I. INTRODUCTION

At the request of concerned residents, the Northampton Board of Health, and the Mayor of Northampton, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health conducted an evaluation of available health outcome data for the city of Northampton and the town of Easthampton. This evaluation was initiated due to community concerns about potential environmental exposures in the area surrounding the Northampton Regional Landfill on Glendale Road in the southwestern part of Northampton, near the border of Easthampton, and potential past and current health effects (see Figure 1). To best address these concerns, the CAP focused this investigation on the neighborhoods/census tracts (CTs) in closest proximity to the landfill, CT 8222.00 in Northampton and CT 8224.01[1] in Easthampton (see Figure 1), as well as the communities of Northampton and Easthampton as a whole. In addition, the CAP conducted an evaluation of cancer incidence within a one-mile radius of the Northampton Regional Landfill.

At a scoping meeting with Northampton officials in August 2007, it was agreed that MDPH would review readily available health outcome data as a service to the community. These health outcomes include cancer, low birthweight, birth defects, asthma, childhood blood lead levels, and autism. It is important to note that this evaluation is meant to serve as a screening-level assessment. Each health outcome

[1] In 1990, the U.S. Census Bureau split what was then Easthampton CT 8224 into two census tracts referred to as 8224.01and 8224.02. CT 8224 in 1980 was the Easthampton CT closest to the Northampton Regional Landfill, and then in 1990, this area of Easthampton became CT 8224.01. For this report, MDPH refers to CT 8224.01 as the one in closest proximity to the landfill. For accuracy, it is important to note that the population data for the years between 1982 and 1991 reflect the CT 8224 boundaries.

evaluated in this report has several risk factors/causes associated with its development. The primary purposes of this type of assessment are to evaluate whether any unusual patterns emerge when assessing disease incidence in the community, in this case particularly in relation to the Northampton Regional Landfill, and/or to generate hypotheses for possible future public health investigations.

II. CANCER INCIDENCE

This investigation provides a review of the pattern of nine cancer types in each community as well as in each of the neighborhoods/ CTs mentioned previously and compares the incidence of these cancers with the cancer experience of the state of Massachusetts as a whole. Cancer incidence data were obtained from the Massachusetts Cancer Registry (MCR) for the years 1982-2004. The MCR began collecting population-based cancer incidence data in January of 1982. The 1982-2004 time period was evaluated by assessing four time periods within the 23-year period: 1982-1986, 1987-1991, 1992-1996 and 1997-2004[2]; this allowed for consideration of possible patterns or trends as compared to the statewide cancer experience. The nine cancer types selected for this evaluation were based on contaminants of concern at the Northampton Regional Landfill and/or resident concern over suspected elevations of some cancer types.

In addition to calculating cancer incidence rates, a qualitative analysis of the geographic distribution of individuals diagnosed with each of the nine types of cancer was conducted by mapping their residence at time of diagnosis. This was done to assess whether the

[2] The first three time periods constitute five-year periods while the last time period constitutes an eight - year period. Because the City of Northampton hired a consulting firm to conduct similar analyses, MDPH chose similar time periods for clarity. For this reason, the numbers of reported cancer diagnoses, as well as the numbers of expected diagnoses based on statewide cancer incidence data, most likely will be higher in the latest time period compared to the earlier time periods.

geographic patterns of any particular type of cancer in either community or in the census tracts of interest appeared unusual. Available risk factor information from the MCR related to age and gender, as well as other factors related to the development of cancer such as smoking and occupation, was reviewed in those instances where the incidence rate of a particular cancer type was higher than expected. This information was evaluated to compare known or established risk factor patterns, as reported in the medical and epidemiological literature for particular cancer types, to risk factor information for individuals diagnosed in Northampton or Easthampton, to assess whether any unexpected patterns existed among individuals diagnosed in Northampton or Easthampton. Finally, a qualitative analysis of cancer incidence within a one-mile radius of the landfill was conducted for the period 1982 to the present[3]. This included evaluating the types of cancer diagnosed, their spatial and temporal distribution, and available risk factor information.

A. *Methods for Analyzing Cancer Incidence*

1. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for Northampton and Easthampton for the years 1982-2004 were obtained from the MCR, a division of the MDPH Bureau of Health Information, Statistics, Research and Evaluation (BHISRE). As mentioned, the MCR is a population-based surveillance system that began collecting information in 1982 on Massachusetts residents diagnosed with cancer in the state. All

[3] Because the MCR is a continual surveillance system, it is possible to review cancer incidence reports for more recent years (i.e., 2005 to the present) for defined geographic areas such as the one-mile radius around the landfill, even though complete statewide data are not available to allow for the calculation of actual cancer incidence rates.

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.111 s.111B).

Nine cancer types were evaluated in this investigation, including cancers of the bladder, brain and central nervous system, breast, kidney, liver, and lung and bronchus as well as leukemia, Hodgkin lymphoma and non-Hodgkin lymphoma. [Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system[4]. See Appendix A for the coding definitions used in this report.] All diagnoses reported to the MCR as primary cancers among residents of Northampton and Easthampton for the nine cancer types were included in the analysis. Individuals diagnosed with cancer were selected for inclusion based on the residential 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.

[4] For purposes of this report, MDPH selected ICD-O coding definitions to address discrepancies that may arise in the interpretation of cancer incidence data due to the fact that City of Northampton hired a consulting firm to conduct similar analyses.

It should be noted that duplicate records have been eliminated from the MCR data used in this report. 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. In general, a diagnosis of a multiple primary cancer 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 2003).

2. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether an elevation occurred among individuals diagnosed with cancer in Northampton or Easthampton, cancer incidence data were tabulated by gender according to eighteen age groups to compare the observed number of cancer diagnoses to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for four time periods, 1982-1986, 1987-1991, 1992-1996, and 1997-2004, for each of the nine primary cancer types for each community and each CT, in order to evaluate patterns or trends in cancer incidence as compared to the statewide cancer experience.

To calculate an SIR, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 U.S. census data for Northampton and Easthampton (U.S. DOC 1980, 1990, and 2000).

Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1989, 1994 and 2000). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the ten-year interval between each census.[5]

A CT 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 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. CTs usually contain between 1,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (U.S. DOC 2000).

SIRs were not calculated for some cancer types in the smaller time periods and/or CTs due to the small number of observed cases (less than five). It is standard BHISRE policy not to calculate rates with fewer than five observed diagnoses due to the instability of the rate. However, the expected number of diagnoses was calculated during each time period and for each CT, and the observed and expected numbers of diagnoses were compared to determine whether excess numbers of cancer diagnoses were occurring.

3. 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

[5] 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.

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 diagnoses in an area to the expected number of diagnoses multiplied by 100. The population structure of each town is adjusted to the statewide incidence rate to calculate the number of expected cancer diagnoses. The SIR is a comparison of the number of diagnoses in the specific area (i.e., city/town or census tract) to the statewide rate. Comparison of SIRs between communities or census tracts is not possible because each of these areas has different population characteristics.

An SIR of 100 indicates that the number of cancer diagnoses observed in the population being evaluated is equal to the number of cancer diagnoses expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer diagnoses occurred than were expected, and an SIR less than 100 indicates that fewer cancer diagnoses occurred than were expected. Accordingly, an SIR of 150 is interpreted as 50% more cancer diagnoses than the expected number; an SIR of 90 indicates 10% fewer cancer diagnoses 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 diagnoses and six observed diagnoses indicates a 50% excess in cancer, but the excess is actually only two diagnoses. Conversely, an SIR of 150 based on 400 expected

diagnoses and 600 observed diagnoses represents the same 50% excess in cancer, but because the SIR is based upon a greater number of diagnoses, the estimate is more stable. It is very unlikely that 200 excess diagnoses of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of diagnoses, SIRs were not calculated when fewer than five diagnoses were observed for a particular cancer type.

4. 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 diagnoses 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 disease rate in the study population is statistically significantly different from the comparison or "normal" population. "Statistically significantly different" means there is less than a 5% chance that the observed difference (either increase or decrease) in the rate is the result of random fluctuation in the number of observed cancer diagnoses.

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 diagnoses. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer diagnoses 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 diagnoses reflects a real cancer increase or decrease or is the result of chance. It is important to note that statistical significance alone does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret cancer patterns in a community.

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 much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of diagnoses, statistical significance was not assessed when fewer than five diagnoses were observed.

5. Determination of Geographic Distribution of Cancer Cases

In addition to calculating SIRs, the address at the time of diagnosis for each individual diagnosed with one of the nine cancer types in Northampton or Easthampton was geographically mapped using a computerized geographic information system (GIS) (ESRI 2005). This allowed assignment of CT location for each individual diagnosed with cancer as well as an evaluation of the spatial distribution of the individuals at a smaller geographic level within CTs (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer diagnoses in each community as well as in CTs 8222.00 and 8224.01. This evaluation included consideration of the population density variability of each CT 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 individuals diagnosed with cancer (e.g., by using telephone books issued within 2 years of an individual's diagnosis or searching files via the Registry of Motor Vehicles). 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.]

B. *Results*

The following sections present cancer incidence rates for the communities of Northampton and Easthampton and for CT 8222.00 in Northampton and CT 8224.01 in Easthampton during the 23-year time period 1982-2004. As shown in Figure 1, the Northampton Regional Landfill is located in CT 8222.00, near the border with CT 8224.01 in Easthampton. As mentioned, to evaluate possible trends over time as compared to the statewide cancer experience, these data were analyzed by four smaller time periods, 1982-1986, 1987-1991, 1992-1996 and 1997-2004. Tables 1A through 1D summarize cancer incidence data for Northampton as a whole while Tables 2A through 2D summarize data for Northampton's CT 8222.00. Cancer incidence data for the town of Easthampton as a whole and its CT 8224.01 are summarized in Tables 3A through 3D and 4A through 4D, respectively. SIRs were not calculated for some cancer types, in these smaller time periods and/or CTs, due to the small number of observed cases (less than five). As previously mentioned, the expected number of diagnoses was calculated during each time period and for each CT, and the observed and expected numbers of

diagnoses were compared to determine whether excess numbers of cancer diagnoses were occurring.

In addition to calculating cancer incidence rates, the CAP also reviewed the types and distribution of all cancers diagnosed within a one-mile radius of the Northampton Regional Landfill since 1982. This review covered 28 streets, in part or whole, in both Northampton and Easthampton neighborhoods within the one-mile radius (see Figure 2 and Table 5). This qualitative review allowed CAP to assess the types of cancers diagnosed as well as any spatial and/or temporal patterns.

Risk factor summaries for each type of cancer evaluated are included in Appendix B.

1. Northampton

In general, cancer incidence rates in Northampton were approximately at or near the rates expected during each of the four time periods evaluated (see Tables 1A-1D). In each of the four time periods evaluated, the incidence of the following five types of cancers in both genders was either less than expected or about as expected when compared to the statewide cancer experience: brain and CNS[6], lung and bronchus, Hodgkin lymphoma, leukemia, and liver cancer. For these five cancer types, if the number of observed diagnoses was greater than the number expected, the difference was based on one or two diagnoses. For incidence rates greater than expected, none of the differences between the numbers of observed and expected diagnoses were statistically significant, meaning that they most likely represent natural or random variation in the incidence of these five cancer types.

[6] The incidence of brain and CNS cancer in Northampton was statistically significantly lower than expected in two of the four time periods: 1982-1986 and 1997-2004.

Breast Cancer

The incidence rates of four types of cancer – breast, bladder, NHL, and kidney – showed somewhat greater variability. Breast cancer occurred slightly more often than expected during two of the four time periods evaluated: 1987-1991 and 1992-1996 (Tables 1B-1C). However, it is important to note that while there were differences between the numbers of observed and expected diagnoses during these two time periods, none were statistically significant. During the earliest time period, however, more females in Northampton were diagnosed with breast cancer than expected, with 112 diagnoses observed compared to approximately 93 expected (Table 1A). This difference was of borderline statistical significance. During the latest time period, a statistically significant elevation occurred in breast cancer in Northampton females with 285 diagnoses observed compared to approximately 249 expected (SIR = 115; 95% CI: 102-129). An important distinction between the earliest and latest time periods, with respect to breast cancer diagnoses, is that in-situ (non-invasive) diagnoses are included in the latest time period but not the earliest time period. The MCR began collecting in-situ breast cancer diagnoses in 1992. It is important to note that the most recent time period evaluated constitutes an eight-year period while the other three time periods evaluated constitute five-year periods.

Available risk factor information was reviewed for those Northampton women diagnosed with breast cancer between 1982 and 1986 and between 1997 and 2004. According to the American Cancer Society (ACS), the chance of an American woman developing invasive breast cancer at some time in her life is about 1 in 8 (12%). The ACS also reports that about 2 out of 3 (67%) women with invasive breast cancer are age 55 or older when they

are diagnosed while about 1 in 8 invasive breast cancer diagnoses are among women younger than age 45 (ACS 2007a). The average age of females diagnosed with breast cancer in Northampton from 1997-2004 was 61 years of age, which is the same as the average age for females diagnosed with breast cancer statewide during that time period. The average age of females diagnosed with breast cancer in Northampton from 1982-1986 was approximately 67 years of age, compared to 64 years of age for women statewide.

MDPH reviewed cancer staging information for women diagnosed with breast cancer in Northampton for the period 1997-2004. Staging describes the extent of spread of an individual's cancer; from a public health perspective, earlier breast cancer staging reflects to some extent whether women are being screened early and regularly for breast cancer. In Northampton, approximately 33% of the women diagnosed with breast cancer between 1997 and 2004 were diagnosed with in-situ (non-invasive) breast cancer compared to 24% statewide.

The ACS also reports that women, who as children or young adults, had radiation therapy to the chest as treatment for another cancer (such as Hodgkin lymphoma or NHL) are at significantly higher risk for breast cancer. Review of MCR data showed that 14 of the 112 women diagnosed with breast cancer during 1982-1986 had been previously diagnosed with cancer. For the period 1997-2004, 84 of the 285 women diagnosed with breast cancer had been previously diagnosed with cancer. While it is unknown whether radiation was used for treatment of their previous cancers, it is possible that such treatment may have contributed to some breast cancer diagnoses.

Review of address information at the time of diagnosis for individuals diagnosed with breast cancer between 1982-1986 and 1997-2004 in Northampton showed no unusual patterns or geographic clustering in the city of Northampton or in the vicinity of the Northampton Regional Landfill. In addition, a review of the geographic distribution of women diagnosed with breast cancer in the other two time periods similarly did not show any unusual spatial patterns.

Bladder Cancer

The incidence of bladder cancer in Northampton males was lower than expected in each of the four time periods evaluated. For Northampton females, the incidence of bladder cancer was approximately as expected during the first three time periods but greater than expected during the last time period. For 1982-1986, fewer Northampton females were diagnosed with bladder cancer than expected: 5 observed compared to 9 expected. For 1987-1991, 11 females were diagnosed with bladder cancer while approximately 8 were expected. For 1992-1996, 14 females were diagnosed with bladder cancer while approximately 11 were expected. For 1997-2004, 28 females were diagnosed with bladder cancer while approximately 19 were expected. It is important to note that the MCR first began collecting in-situ bladder cancer diagnoses in 1992. Therefore, the number of individuals reported in the MCR with bladder cancer increased statewide. Also, as discussed previously, the latest time period constitutes an eight-year period while the other three time periods evaluated constitute five-year periods.

Available risk factor information was reviewed for those Northampton females diagnosed with bladder cancer between 1987 and 2004. According to the American Cancer Society

(ACS), over 70% of people diagnosed with bladder cancer are over 65 years of age and nearly 90% are over age 55 at diagnosis. For Northampton females diagnosed between 1987 and 2004, 79% were over 65 years of age at diagnosis and 91% were over age 55 at diagnosis. The average age at diagnosis for the Northampton females was 73 compared to 71 for all Massachusetts females diagnosed with bladder cancer during this same time period. The greatest risk factor for bladder cancer is smoking. According to the ACS, smokers are more than twice as likely as non-smokers to develop bladder cancer. Other risk factors associated with an increased risk of bladder cancer include exposure to certain industrial chemicals, particularly in occupational settings such as the dye, rubber, leather, textiles, painting and printing industries; a history of chronic bladder inflammation; bladder birth defects; genetics; certain chemotherapeutic agents and radiation therapies for previous cancers; high levels of arsenic in drinking water; and, an individual's fluid consumption (ACS 2007d).

Of the 53 females, seven reported an occupation associated with an increased risk of developing bladder cancer; however, information regarding specific job duties that could help to further define exposure potential for these individuals was not available. Tobacco use history was reviewed for those Northampton females diagnosed with bladder cancer between 1987 and 2004. Smoking history was unknown for 18 of the 53 females diagnosed during this time period. Eighteen of the 35 females (51%) for whom smoking history was reported to the MCR were current or former smokers at the time of their bladder cancer diagnosis, compared to 60% statewide.

The spatial distribution of place of residence for Northampton females diagnosed with bladder cancer between 1987 and 2004 was also reviewed. The geographic distribution

of the reported residences of Northampton females diagnosed with bladder cancer during this time period did not appear unusual and closely followed the population distribution of the city of Northampton. No unusual clustering around the landfill was observed. MDPH also evaluated the geographic distribution of residence at diagnosis for those females with bladder cancer who did not have a history of smoking as well as for those females with no potential for an occupational exposure, and again found no unusual spatial patterns. The spatial patterns for both groups closely followed the population density of the city.

Non-Hodgkin Lymphoma

For three of the four time periods evaluated (1987-1991, 1992-1996, and 1997-2004), the incidence of NHL in Northampton, as a whole, was either less than expected or as expected for both genders (Tables 1B -1D). From 1982-1986, however, more individuals were diagnosed with NHL than expected when compared to the statewide incidence rate (25 observed versus approximately 20 expected) (Table 1A). This elevation occurred among both males and females, with approximately two more males and three more females being diagnosed with NHL during this time period than expected. Neither of these elevations was statistically significant.

Available risk factor information was reviewed for those Northampton residents diagnosed with NHL between 1982 and 1986. According to the American Cancer Society (ACS), over 95% of individuals diagnosed with NHL are adults. The type of non-Hodgkin lymphoma seen in children is often very different from that seen in adults. The average age of individuals at diagnosis is in the 60s and about half of the individuals

diagnosed with NHL are over 65 years of age (ACS 2007b). The average age of individuals diagnosed with NHL in Northampton during this time period was 64 years and 48% of the individuals were over age 65 at the time of their diagnosis.

There are many different types of NHL. The most common types of NHL can be grouped into two broad classifications, B-cell or T-cell lymphoma. Nationally, B-cell lymphoma accounts for approximately 85% of all NHL diagnoses (ACS 2007b). In Northampton, 84% of individuals diagnosed with NHL between 1982 and 1986 were diagnosed with the B-cell subtype.

Overall, review of address information at the time of diagnosis for individuals diagnosed with NHL between 1982 and 1986 showed no unusual pattern. Two individuals, who were diagnosed with NHL within three months of one another, did reside in close proximity to one another at the time of their diagnosis. However, these individuals lived in a fairly densely populated area of Northampton. It is also worthy of note that this area is approximately four miles away from the landfill.

Kidney Cancer

For Northampton as a whole, kidney cancer in females was statistically significantly elevated during the last time period of 1997–2004 (Table 1D). Twenty-three diagnoses were observed while 13.0 would be expected, based on the statewide incidence of kidney cancer in females; the SIR was 176 (95% CI: 112-265). In the previous three time periods, kidney cancer in females was either as expected or below the expected rate. In males, kidney cancer incidence was about as expected during the four time periods.

According to the ACS, the average age at the time of diagnosis of kidney cancer is 65. With one exception, kidney cancer is very uncommon under age 45, and its incidence is highest between the ages of 55 and 84. Wilm's tumor, which represents about 5% of all kidney cancers, is most common in children. In Northampton, the average age of females diagnosed with kidney cancer between 1997 and 2004 was 64 years. Two of the 23 females were under age 19 at the time of their diagnosis and both were diagnosed with Wilm's tumor.

The histologies or subtypes of kidney cancer in the 21 adult females were also reviewed and compared to what would be expected, based on the medical literature and national cancer statistics. Between 80 and 90% of all kidney cancers are renal cell carcinomas (ACS 2007c). In Northampton, 16 of the 19 (84%) adult females for whom subtype information was available were diagnosed with renal cell carcinomas. Subtype information was not known for two of the 21 adult females.

Some lifestyle-related risk factors are associated with the development of kidney cancer. They include smoking and obesity. Occupational exposure to some chemicals has also played a role in the development of kidney cancer (ACS 2007c). Of the 16 adult females diagnosed with kidney cancer in Northampton between 1997 and 2004 for whom their smoking history was reported to the MCR, 10 reported being current or former smokers at the time of their diagnosis. History of tobacco use was unknown for 5 of the 21 adult females. Review of occupational information reported to the MCR for the 21 adult females diagnosed with kidney cancer showed that five of the 21 reported occupations where exposure to chemicals associated with kidney cancer could have occurred. However, specific job duty information that could further define exposure potential was

not available through the MCR. Occupation was reported as "unknown" or "at home" for 12 of the 21 adult females. Information on factors such as obesity is not reported to the MCR. (A more detailed discussion of all of the risk factors associated with the development of kidney cancer is provided in Appendix B.)

The geographic distribution of the reported residences of Northampton females diagnosed with kidney cancer during this time period did not appear unusual and closely followed the population distribution of the city of Northampton. MDPH also evaluated the geographic distribution of residence at diagnosis for those females with kidney cancer who did not have a history of smoking as well as for those females with no potential for an occupational exposure, and found no unusual spatial patterns. The spatial patterns for both groups closely followed the population density of the city. In addition, the distribution of year at diagnosis among the 23 females was spread fairly evenly across the 8-year period. There was no temporal clustering by year of diagnosis.

2. Northampton CT 8222.00

Review of cancer incidence rates for males and females combined in CT 8222.00 in Northampton showed that cancer incidence occurred as or below expected for most of the nine cancer types evaluated (Tables 2A – 2D). In all four time periods, individuals living in CT 8222.00 were diagnosed with liver cancer less often than expected based on the statewide liver cancer experience, with one individual diagnosed with liver cancer over the 23-year period. The incidence of Hodgkin lymphoma, non-Hodgkin lymphoma, bladder cancer and brain and CNS cancer occurred approximately as or below expected in each of the time periods evaluated. Any observed elevations were based on one or two

individuals above the expected number and were not statistically significant. Elevations did exist for a few of the cancer types in some time periods, most were not statistically significant and likely represent natural variability in rates. These elevations are discussed below. There were two statistically significant elevations in this CT during different time periods. These included leukemia incidence in 1992-1996 and breast cancer incidence in 1997-2004, both of which are also discussed below.

Breast Cancer

For three of the four time periods evaluated (1982-1986, 1992-1996, and 1987-1991), the incidence of breast cancer in Northampton women from CT 8222.00 was not statistically significantly different from the statewide breast cancer experience (Tables 2A, 2B, and 2C). In 1982-1986, fewer females in this CT were diagnosed with breast cancer than would be expected: 13 observed versus 14.2 expected. In 1987-1991, 22 females were diagnosed with breast cancer when approximately 18 were expected. In 1992-1996, the incidence was again below the expected rate: 18 observed versus 25.1 expected. Between 1997 and 2004, 68 women were diagnosed with breast cancer in this CT compared to approximately 53 expected (Table 2D). This elevation was statistically significant (SIR=129; 95% CI: 101-164). As discussed in the previous section on breast cancer incidence citywide in Northampton, it is important to note that the latest time period covers eight years (compared to five years for the previous three time periods) and includes in-situ (non-invasive) breast cancer diagnoses.

Available risk factor information was reviewed for those Northampton females diagnosed with breast cancer during the most recent time period. The average age at diagnosis for

females diagnosed with breast cancer during this time period was younger than the statewide average (56 years of age in Northampton CT 8222.00 compared to 61 years of age statewide). A review of the spatial distribution of residence at diagnosis for Northampton females diagnosed with breast cancer while living in CT 8222.00 showed that the pattern closely followed the population density patterns within the census tract.

MDPH reviewed cancer staging information for women diagnosed with breast cancer in Northampton CT 8222.00 for the period 1997-2004. Staging describes the extent of spread of an individual's cancer; from a public health perspective, earlier breast cancer staging reflects to some extent whether women are being screened early and regularly for breast cancer. In Northampton CT 8222.00, approximately 43% of the women diagnosed with breast cancer between 1997 and 2004 were diagnosed with in-situ (non-invasive) breast cancer compared to 24% statewide.

Leukemia

The incidence of leukemia occurred about as expected or less than expected in Northampton CT 8222.00 for three of the four time periods evaluated. From 1992-1996, however, leukemia was statistically significantly elevated; seven individuals were diagnosed while approximately three would have been expected (SIR 277, 95% CI: 111-571). This elevation was due to the number of reported diagnoses in females, with 6 diagnoses observed versus approximately 1 expected.

Leukemia is grouped into four subtypes based on the type of white blood cell affected and how quickly the disease develops. These subtypes are acute myeloid leukemia (AML), acute lymphocytic leukemia (ALL), chronic myeloid leukemia (CML), and

chronic lymphocytic leukemia (CLL). The etiologies and risk factors for each subtype of leukemia are different. AML is the most commonly diagnosed subtype of leukemia. According to the National Cancer Institute, there are about 10,600 new diagnoses of AML annually, representing about 34% of leukemia diagnoses each year (NCI 2003). AML occurs in both adults and children. In CT 8222.00, four of the six females diagnosed with leukemia were diagnosed with AML.

Risk factors for AML include smoking and long-term occupational exposure to high levels of benzene or formaldehyde. Additionally, individuals previously diagnosed with cancer who were treated with certain cancer-fighting drugs (chemotherapy) or with high-dose radiation may later develop leukemia. For example, drugs known as *alkylating agents* are associated with the development of leukemia many years later (NCI 2003). (For a fuller discussion of risk factors for leukemia, see Appendix B.) Review of risk factor data available from the MCR identified one of the four individuals diagnosed with AML as having had a previous cancer diagnosis. This patient may have received treatment for their initial cancer that could have contributed to their subsequent leukemia diagnosis. However, it is not possible using readily-available information from the MCR to determine whether this individual actually received chemotherapy or radiation therapy for his/her previous cancer. Additionally, two of the four females diagnosed with AML reported to the MCR that they were current or former tobacco users. One individual was reported as a nonsmoker and the smoking status was reported as unknown for the remaining individual.

The geographic distribution of all of the individuals diagnosed with leukemia in CT 8222.00 closely followed the patterns of population density in this CT. No unusual spatial patterns were noted when the residences at diagnosis were mapped.

Kidney Cancer

In the three earliest time periods, with one exception, kidney cancer occurred about as or less often than expected in CT 8222.00 (Tables 2A-2C). During 1982-1986, more males in CT 8222.00 were diagnosed with kidney cancer than expected (4 diagnoses observed versus one expected). The incidence of kidney cancer was also elevated in Northampton CT 8222.00 during the most recent time period among both males and females. From 1997-2004, eight males were diagnosed with kidney cancer while approximately four would have been expected and four females were diagnosed while approximately two would have been expected. This elevation was not statistically significant.

Available risk factor information was reviewed for those Northampton residents of CT 8222.00 diagnosed with kidney cancer during the latter time period. According to the American Cancer Society, the incidence of kidney cancer is the highest in individuals between 55 and 84 years of age (ACS 2007c). In Northampton CT 8222.00, the average age of the 12 individuals diagnosed with kidney cancer was 68 years. In addition, the ACS reports smoking as a risk factor for kidney cancer (ACS 2007c). Of the 12 individuals diagnosed with kidney cancer, tobacco history was reported for 10 individuals. Of these 10, five reported being current or former tobacco users at the time of their diagnosis.

Most of the individuals diagnosed with kidney cancer did not live in close proximity to one another or the Northampton Regional Landfill at the time of their diagnosis. There was one exception. Three males and one female lived in close proximity to one another; however, their residences were in a densely populated area of Northampton and were more than 3 miles from the landfill.

Lung and Bronchus Cancer

In Northampton CT 8222.00, fewer individuals were diagnosed with cancer of the lung or bronchus than expected, based on the statewide experience, during each of the first three time periods. From 1997-2004, however, more individuals were diagnosed with cancer of the lung or bronchus than expected. This elevation, although not statistically significant, was due to an elevated incidence among females. Twenty-one females were diagnosed with cancer of the lung or bronchus while approximately 15 would be expected. Of the 21 females diagnosed, 19 were reported to the MCR as current or former smokers. Smoking status was unknown for one individual and the remaining individual reported being a nonsmoker. The geographic distribution of individuals diagnosed with cancer of the lung or bronchus closely matched the population density of Northampton. Additionally, there was no spatial clustering in the area surrounding the Northampton Regional Landfill.

3. Easthampton

In general, cancer incidence rates in Easthampton were approximately at or near the rates expected during each of the four time periods evaluated (see Tables 3A-3D). In each of the four time periods evaluated, the incidence of the following six types of cancers in

both genders was either less than expected or about as expected when compared to the statewide cancer experience: bladder, breast, Hodgkin lymphoma, leukemia, NHL, and liver cancer. Except for NHL during one time period, any differences between the number of observed diagnoses and the number expected for these six types of cancers were based on one or two diagnoses. (During the 1997-2004 time period, 17 diagnoses of NHL were reported among males in Easthampton compared to approximately 14 expected.) With one exception, none of the differences between the numbers of observed and expected diagnoses were statistically significant, meaning that they most likely represent natural or random variation in the incidence of these six cancer types. The one exception was that the incidence of NHL was statistically significantly lower than expected during 1992-1996.

Brain and CNS Cancer

The incidence rates of three types of cancer – brain and CNS, kidney, and lung and bronchus – showed somewhat greater variability. In three of the four time periods evaluated, more individuals were diagnosed with cancer of the brain or CNS cancer in Easthampton than would be expected (see Tables 3A, 3B, and 3D). None of these elevations were statistically significant.

Between 1982 and 1986, a total of 11 males and females were diagnosed with cancer of the brain or CNS while seven diagnoses would have been expected based on the statewide experience, however this difference was not statistically significant. Of the 11 individuals diagnosed, two were diagnosed with a cancer of the CNS; the remaining nine were diagnosed with brain cancer.

There are few known risk factors for brain or CNS cancer. According to the ACS, the only known environmental risk factor for brain or CNS cancer is exposure to radiation (ACS 2006a). Today, because the health risks associated with most types of radiation are known, it has been established that radiation-induced tumors in non-occupationally exposed populations are generally the result of treatment for a previous cancer diagnosis. Other suspected risk factors for brain or CNS cancer are exposure to chemicals such as vinyl chloride and petroleum products (ACS 2006a). Occupation was reported as unknown or retired to the MCR, with no further information, for six of the 11 individuals. None of the remaining five individuals reported their usual occupation as one that might be associated with exposure to these chemicals; however, a complete occupational history is not provided to the MCR. Review of MCR data for the 11 individuals diagnosed with brain or CNS cancer showed that none had been previously diagnosed with cancer. No geographical concentrations of diagnoses were observed among the 11 individuals that could not be attributed to population density.

From 1987-1991, eleven individuals were diagnosed with cancer of the brain or CNS in Easthampton. Approximately eight diagnoses would have been expected based on the statewide brain and CNS cancer experience. The number of females diagnosed with brain or CNS cancer during this time period was approximately equal to the number expected, while slightly more males were diagnosed than expected (7 observed versus 4 expected). This difference was not statistically significant.

As with the previous time period, a review of MCR data did not indicate that any of the 11 individuals had been previously diagnosed with cancer. Occupation was reported to the MCR as retired or unknown for four of the 11 individuals. Four other individuals

reported their usual occupation as one where an exposure thought to increase the risk of brain or CNS cancer might have been possible. The usual occupations reported for the remaining three individuals were ones not associated with possible occupational exposures related to brain or CNS cancers. Review of the geographical distribution of the 11 individuals diagnosed with a brain or CNS cancer between 1987 and 1991 in Easthampton did not show any unusual geographic clustering in any one part of Easthampton or in the vicinity of the Northampton Regional Landfill.

From 1992-1996, the incidence of brain and CNS cancer in females occurred about as expected in Easthampton while fewer males were diagnosed with this type of cancer than expected.

In the most recent time period (1997-2004), more individuals were diagnosed with cancer of the brain or CNS than expected in the city of Easthampton. This was due to an elevation among females (15 observed versus approximately 10 expected) that was not statistically significant. The incidence of brain or CNS cancer in males was as expected during this time period. One of the 15 females diagnosed with brain or CNS cancer had been previously diagnosed with cancer; however, it is unknown whether radiation was used for treatment of that cancer. Occupational history was not reported to the MCR for three females. For the remaining 12 females, none reported an occupation where an exposure to vinyl chloride or petroleum products seemed likely. Finally, review of address at diagnosis for these females did not show an unusual concentration in any area of Easthampton or surrounding the Northampton Regional Landfill.

The incidence of brain and CNS cancer in Easthampton fluctuated over the course of the 23-year period evaluated, with no consistent pattern emerging. For males, the incidence of brain and CNS cancer was about as expected in the first time period, with six diagnoses observed compared to approximately four expected; the difference was not statistically significant. Similarly, in the second time period, seven males were diagnosed with brain and CNS cancer when approximately four were expected; the difference was not statistically significant. In the third time period, the incidence of brain and CNS cancer in males was lower than expected with one diagnoses reported compared to four expected. In the last time period, the incidence of brain and CNS cancer in males was as expected, with eight diagnoses observed and eight expected. For females, in the first three time periods, the incidence of brain and CNS cancer was either about as expected or lower than expected. For 1982-1986, five diagnoses were reported compared to approximately four expected. For 1987-1991, the incidence of brain and CNS cancer in females was as expected: four diagnoses reported compared to four diagnoses expected. For 1992-1996, fewer diagnoses occurred in females than expected: three observed compared to approximately four expected. In the last time period, more females were diagnosed with brain and CNS cancer than expected: 15 diagnoses observed compared to 10 diagnoses expected; this difference was not statistically significant.

When the spatial distribution of residence at diagnosis was examined, no unusual geographic patterns emerged. The diagnoses were evenly spread across the town, with no particular geographic clustering. CAP also examined the age at diagnosis for the various subtypes of brain and CNS cancer diagnosed in Easthampton residents in relation to what is reported in the medical literature. The subtypes of brain and CNS cancers in

Easthampton, and the ages at diagnosis for the various subtypes, were consistent with what would be expected. The relative percentages of brain and CNS cancer subtypes in Easthampton followed those reported by the American Cancer Society, with the majority of diagnoses being gliomas (a general category that includes astrocytomas, oligodendrogliomas, and ependymomas) followed by meningiomas (ACS 2006a). In addition, no unusual pattern with respect to age at diagnosis was noted. Among the six children/adolescents (ages 19 and younger) diagnosed with brain cancer during the 23-year period, all were diagnosed with the most common subtype among children, astrocytoma.

Kidney Cancer

The number of individuals diagnosed with kidney cancer in Easthampton during three of the four time periods evaluated was below or approximately equal to the expected number (see Table 3B, 3C, and 3D). In the earliest time period evaluated, from 1982-1986, the incidence of kidney cancer in males was about as expected while for females, six diagnoses were reported to the MCR when approximately two would have been expected. This elevation was not statistically significant.

Lung and Bronchus Cancer

The incidence of lung and bronchus cancer in Easthampton was below expected for males and females combined in three of the four time periods and for each gender when evaluated separately. For the 1992-1996 time period, there was an elevation in the incidence of lung and bronchus cancer in females with 29 diagnoses reported while approximately 23 would be expected. This difference was not statistically significant. In

the subsequent time period, 1997-2004, the incidence of lung and bronchus cancer in Easthampton females was statistically significantly lower than expected. During this same time period of 1997-2004, there was an elevation in males, with 59 diagnoses reported while approximately 52 would be expected; again, however, this difference was not statistically significant. Although an elevation in the incidence of lung and bronchus cancer occurred in each gender for one time period, the elevations do not represent a consistent trend and most likely represent natural random variation in the incidence of this type of cancer.

4. Easthampton CT 8224.01

Review of cancer incidence data for individuals who reported an address within CT 8224.01[7] at the time of their diagnosis showed that, for six of the nine cancer types, the incidence was either below expected or about as expected in each of the four time periods evaluated. Any observed elevations were based on one or two diagnoses above the expected number of individuals. These elevations were not statistically significant. For three cancer types – lung and bronchus, brain and CNS, and kidney – there was more variability in their incidence rates. Although more fluctuation in these rates was noted, no statistically significant elevations were seen.

Lung and bronchus cancer in males during the 1992-1996 time period was elevated with 12 diagnoses reported to the MCR when approximately eight would have been expected.

[7] In 1990, the U.S. Census Bureau split what was then Easthampton CT 8224 into two census tracts referred to as 8224.01and 8224.02. CT 8224 in 1980 was the Easthampton CT closest to the Northampton Regional Landfill, and then in 1990, this area of Easthampton became CT 8224.01. For this report, MDPH refers to CT 8224.01 as the one in closest proximity to the landfill. For accuracy, it is important to note that the population data for the years between 1982 and 1991 reflect the CT 8224 boundaries.

This difference was not statistically significant. In the other three time periods evaluated, the incidence of lung and bronchus cancer in males in this CT was either less than or as expected. In the first two time periods, brain and CNS cancer incidence was slightly elevated. In 1982-1986, eight diagnoses in males and females combined were observed compared to approximately four diagnoses expected; this difference was not statistically significant. In 1987-1991, although the number of observed diagnoses in females was as expected, there were six diagnoses of brain and CNS cancer in Easthampton males in this CT when approximately two would be expected; this difference was not statistically significant. In the subsequent two time periods, the incidence of brain and CNS cancer in this CT was either less than expected or near expected. For kidney cancer in Easthampton CT 8224.01, the incidence was about as expected during the last three time periods evaluated. In the first time period, however, it was slightly elevated in females with four diagnoses reported when approximately one was expected. In males during this time period, three diagnoses were reported when approximately two were expected.

5. One Mile Radius Surrounding the Northampton Regional Landfill

To further address the concerns of residents living in close proximity to the Northampton Regional Landfill, an analysis of all types of cancer diagnosed in this area was conducted. For this analysis, the pattern of all cancer diagnoses was reviewed for an area that constituted a one-mile radius around the landfill. This is an area that is bordered to the east by Sovereign Way, to the south by Lexington Drive, to the west by Loudville Road, and to the north by Ryan Road (see Figure 2). Twenty-eight different streets, in part or whole, were included in this area (see Table 5 for a list of the streets). In general, our

review found no atypical pattern of cancer in the neighborhood surrounding the Northampton Regional Landfill. From 1982 to the present, a total of 24 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 also consistent with national trends in cancer incidence (ACS 2006b). Together, these cancer types represented more than half (58%) of the cancer diagnoses in this one-mile area. There were also a number of other cancer types diagnosed among residents of these areas of Northampton and Easthampton over the 26-year period reviewed. These types included cancers of the bladder, cervix, kidney, brain and CNS, testes, stomach, oral cavity and pharynx, ovary, thyroid, and uterus as well as Hodgkin lymphoma, mesothelioma, leukemia, melanoma of the skin, and non-Hodgkin's lymphoma. The types of cancer that occurred varied and there was no specific pattern or geographic concentration of any one cancer type within this one-mile area. Also, for any given type of cancer, the years of diagnosis varied throughout the 26 years reviewed, indicating no apparent trend or pattern in the time of diagnosis.

Because the MCR collects some information related to risk factors for individuals diagnosed with cancer, these data were reviewed to better characterize the cancer incidence patterns within the one-mile radius of the landfill. Age at diagnosis, gender, smoking history, and occupation were reviewed for those individuals diagnosed with cancer and whose residence was within the one-mile radius of the landfill.

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 in the one-mile radius with any type of cancer was 63 years and the majority of individuals (82%) were age 50 or older when they were diagnosed. For each particular type of cancer, the age at diagnosis was reviewed and determined to be consistent with what is reported in the epidemiological literature for that cancer type.

Because cigarette smoking is also an important risk factor in the development of several cancer types, including cancers of the lung and bronchus, oral cavity and pharynx, colon/rectum, bladder, kidney, and stomach, smoking history was reviewed for each individual within the one-mile radius who had been diagnosed with a potentially smoking-related cancer. Of the 43 individuals with one of these cancer types, 27 (63%) reported being current or former smokers at the time of diagnosis, eight reported being non-smokers, and smoking history was unknown for the remaining eight individuals. Therefore, smoking more than likely played some role in the development of cancer among some residents of the one-mile area surrounding the landfill.

In addition, some occupational exposures have been associated with an increased risk for developing certain types of cancer. A review of occupation as reported to the MCR showed that seven individuals diagnosed with cancer in the one-mile radius worked in jobs that may be associated with an increased risk for developing the particular type of cancer. However, specific job duty information that could further define exposure potential was not available through the MCR

Finally, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer within the one-mile radius did not reveal any atypical spatial patterns that would suggest an association with a common environmental factor.

III. REPRODUCTIVE OUTCOME DATA

Birth Defects

In response to concerns about birth defects in areas surrounding the Northampton Regional Landfill, the CAP obtained birth defects data for Northampton and Easthampton from the Massachusetts Center for Birth Defects Research and Prevention within MDPH's Bureau of Family Health and Nutrition. Massachusetts implemented a statewide birth defects surveillance system in 1997, with 2000 being the first year for which data on birth defects are available. The aim of the surveillance system is to identify major structural birth defects in fetuses of at least 20 weeks gestational age and infants up to one year of age. Most structural defects fall into the following categories: central nervous system, eyes, ears, cardiovascular/respiratory, orofacial, gastrointestinal, genitourinary, and musculoskeletal. Other conditions monitored in this system include genetic and chromosomal abnormalities. The surveillance system involves trained physicians and medical/public health personnel who validate birth defects cases reported to the MDPH. The birth defect cases can be evaluated by city or town, with a child assigned to the city/town of the mother's residence at the time of birth.

Population-based birth defect rates are available for the years 2000 through 2003. Birth defect rates are calculated as the number of birth defect diagnoses at a point in time (prevalence) and are presented per 10,000 births. Ninety-five percent confidence intervals

are also provided to assess the magnitude and stability of the prevalence estimate. If the confidence intervals for the community do not overlap with (i.e. fall within the range of) the state's confidence intervals, then the rates are considered to be statistically significantly different. If the confidence intervals around the two rates being compared do overlap, then it is likely that the differences in the two rates may be due to chance or natural random variation.

The causes of birth defects are poorly understood. Genetic and environmental factors have been identified in certain defects. These include: prenatal environmental factors, such as infections (e.g. rubella), exposures to medications, chemical contamination of environmental media (such as drinking water), drug or alcohol abuse, and nutritional deficiencies. It is also known that a single abnormal gene can cause certain birth defects. The gene may have an error in its code, a missing piece or extra genetic material, all of which can result in birth defects/congenital malformations. Other birth defects may be caused by a combination of factors, such as genes interacting with environmental factors. For 60-70% of major birth defects, no known cause has been identified.

Table 6 shows the number of birth defects diagnoses reported to the MDPH Center for Birth Defects Research and Prevention for Northampton and Easthampton for the four-year period of 2000 through 2003, as well as for the state of Massachusetts as a whole for comparison purposes. There were a total of 16 and 12 birth defect diagnoses in Northampton and Easthampton, respectively, over the four-year period. Although both communities experienced a birth defect rate somewhat higher than the state's, neither was statistically significantly elevated. Both rates had large confidence intervals, indicating their relative statistical instability. Also, while birth defects are grouped together due to

the small number of events, like cancer, different outcomes are thought to be etiologically different. For example, cleft palate and cleft lip are considered distinct outcomes.

A clinical geneticist within the MDPH Center for Birth Defects Research and Prevention reviewed the individual birth defects and examined them from the perspective of potential teratogenic exposures, looking for patterns of multiple defects or early exposures within the Northampton and Easthampton children diagnosed with a birth defect. (A teratogen is an agent that can cause developmental abnormalities in a fetus.) The clinical geneticist did not identify any unusual patterns of defects within either community.

In addition, the CAP evaluated birth defects in Northampton and Easthampton during the 2000-2003 time period with respect to geographic distribution of the mother's residence, as reported at the time of the child's birth. Birth defect cases which occurred within this time period in Northampton and Easthampton were geocoded and mapped. The spatial distribution of birth defects in both communities was reviewed to assess if there were any atypical groupings or concentrations of particular birth defects within any particular areas in the communities. No spatial patterns were observed in either community, and the distribution was consistent with population density within each community.

Low Birthweight

The prevalence of low birthweight births was another concern expressed by community members living near the Northampton Regional Landfill. To address this concern, the CAP reviewed rates of low birthweight births in Northampton, Easthampton, and the state as a whole for the years 2000-2005.

Low birthweight is considered to be anything below 2500 grams (approximately 5 pounds, 8 ounces) when a newborn is carried to full term. Babies born with a low birthweight are alternatively referred to as "small for gestational age," and the terms are used interchangeably. There are many risk factors for low birthweight, including insufficient placental growth, growth restriction, maternal smoking, and other factors. Growth restriction is classified by the point during pregnancy when the fetus is affected—symmetrical growth restriction begins in early pregnancy, while asymmetrical growth restriction is a result of an event in the third trimester, such as preeclampsia. Fetuses with asymmetrical growth restriction tend to have a disparity in their length and head circumference when compared to body weight. Maternal risk factors for low birthweight include poor nutrition, smoking, and drug or alcohol abuse. Infections such as rubella, cytomegalovirus, toxoplasmosis, and syphilis can also lead to low birthweight. Chronic renal failure, sickle cell anemia, phenylketonuria, thrombophilia, and preeclampsia are all conditions considered to be risk factors for low birthweight. Research is beginning to emerge on environmental risk factors for low birthweight. A recent British study found that increased maternal exposure to air pollution, particularly sulfur dioxide, was associated with low birthweight prevalence (Bobak 2000).

Low birthweight data are collected by the MDPH Bureau of Health Information, Statistics, Research, and Evaluation (BHISRE) Registry of Vital Records and Statistics. The number of low birthweight births for each city and town and the state as a whole are available through the Massachusetts Community Health Information Profile (MassCHIP). For this analysis, rates were calculated by comparing the number of low birthweight babies to the number of total births per community within the time period of 2000-2005.

A rate per 10,000 live births was then calculated to allow for easier comparison to the state. A 95% confidence interval (CI) was also calculated for each rate. As discussed earlier, a confidence interval indicates the precision of a rate; the wider the interval, the less certain or stable the rate. Statistically, the width of the interval reflects the size of the population and the number of events; smaller populations yield less precise estimates which have wider confidence intervals. The results of the analysis of low birthweight data can be seen in Table 7.

As illustrated in Table 7, both Northampton and Easthampton had prevalence estimates for low birthweight births that were statistically significantly lower than the state of Massachusetts as a whole. In the six-year time period analyzed, 46 babies were born in Northampton and 48 were born in Easthampton that were considered to be of low birthweight. Because these numbers are relatively small, the calculated prevalence estimates produced wide confidence intervals, illustrating their statistical instability. However, because the estimates are considerably lower than that of the state, it can reasonably be concluded that low birthweight prevalence is not elevated in either community.

IV. ASTHMA SURVEILLANCE

Asthma prevalence was included as a concern of residents living near the Northampton Regional Landfill. According to the U.S. Centers for Disease Control (CDC) definition, asthma is a reversible obstructive lung disease caused by increased reaction of the airways to various stimuli (Akinbami 2006). When an individual suffers from asthma, the

muscles of the airways constrict, causing an interruption in normal breathing. This constriction is a result of an environmental or biological agent which irritates the airway.

Although there is medical agreement on agents that can trigger asthma, such as cold air, allergens, and exposure to fine particles in air pollution, current information in the medical literature does not provide firm conclusions about the causes of or risk factors for the initial onset of asthma. Many researchers have sought to identify specific environmental exposures associated with the development of asthma. In a report by the National Institute of Medicine for the U.S. Environmental Protection Agency on exposures related to both asthma onset and the exacerbation of established asthma, exposure to house dust mites was reported to cause asthma onset while exposure to environmental tobacco smoke among preschool age children was reported to be associated with asthma onset (meaning the weight of the evidence for causation was less for environmental tobacco smoke than for house dust mites) (Redd 2002). It is well-documented that outdoor exposures to ozone and particulate matter can exacerbate asthma. However, whether outdoor pollution is associated with asthma onset is not yet known.

Children are more likely to be diagnosed with asthma than adults. Approximately 12.5% of children in the US have been diagnosed with asthma (Dey and Bloom 2005). In the last few decades, the prevalence of asthma has increased, especially among children. The reason for this increase has yet to be explained. Male children are more likely to be diagnosed with asthma than female children. This gender inequality changes once individuals reach adulthood, when women become more likely than men to develop asthma (ALA 2005). Asthma which occurs in childhood will sometimes resolve before

adulthood. Currently about 7% of the entire U.S. population suffers from asthma, however rates in Massachusetts and New England are among the highest in the U.S.

Statewide pediatric asthma surveillance has been conducted by the BEH for school years 2003-2004, 2004-2005, 2005-2006, and 2006-2007 (MDPH 2004, 2005a, 2006, 2007). As part of a cooperative agreement with the CDC, MDPH's BEH established a statewide tracking system for pediatric asthma. All public and private schools in Massachusetts with any grade kindergarten through 8 report the number of enrolled students with a diagnosis of asthma. This information is provided by school nurses and/or administrative staff through a standardized report form. School enrollment data were collected from the Massachusetts Department of Education (MDOE) or from a school's administrative staff. Data available from the tracking program indicate that the statewide prevalence of asthma among children in grades K through 8 is approximately 10%.

Table 8 contains prevalence estimates for all of the Northampton K-8 schools that have participated in the MDPH pediatric asthma surveillance program. The prevalence of asthma in the Bridge School was statistically significantly higher than the statewide prevalence for 3 of the 4 years reported. At the John F. Kennedy Middle School, asthma prevalence was statistically significantly elevated in the first two years of surveillance, but not in the subsequent two years. At the Jackson Street School, asthma prevalence was similar to the state as a whole for the first three years of surveillance and then statistically significantly elevated in the most recent year of surveillance. In three different schools – the Leeds, the Montessori School of Northampton, and the Solomon Schechter Day School – pediatric asthma prevalence was either similar to the state as a whole or statistically significantly lower than the statewide prevalence, depending on the

school year. For the remaining Northampton K-8 schools, the prevalence of pediatric asthma was similar to that of the state as a whole.

Table 9 contains asthma prevalence estimates for all of the participating Easthampton K-8 schools. The prevalence of pediatric asthma in the Center School was statistically significantly higher than that of the state as a whole for the years 2005-2006 and 2006-2007 and at the Neil A. Pepin School for the year 2005-2006. For the other school years, the prevalence of pediatric asthma in both schools was either lower than or consistent with the statewide pediatric asthma experience. The White Brook Middle School had statistically significantly lower pediatric asthma prevalence in 2003-2004 and 2004-2005 than the state as a whole. For the remaining Easthampton K-8 schools, the prevalence of pediatric asthma was similar to that of the state as a whole.

The Robert K. Finn School on Ryan Road in Northampton is in closest proximity to the Northampton Regional Landfill (see Figure 3); it is approximately a mile and a half from the Northampton Regional Landfill. For three school years, 2003-2004, 2004-2005, and 2005-2006, the prevalence of asthma in the R.K. Finn School was similar (i.e., not statistically significantly different) compared to the state as a whole. It ranged from 8.2% in the 2005-2006 school year to 10.3% in the 2004-2005 school year. This is consistent with the statewide pediatric asthma prevalence. In the latest school year of surveillance, 2006-2007, asthma prevalence was statistically significantly elevated at the R.K. Finn School (at approximately 16%). There are no schools in Easthampton within 2 miles of the landfill.

Hospital discharge data were also analyzed to assess hospitalizations for asthma-related conditions for Northampton and Easthampton. The Massachusetts Division of Health Care Finance and Policy (DHCFP) collects patient-level information on all patients who are discharged from hospitals in Massachusetts. The collection of data is mandated by regulation 114.1 CMR 17.00, *Requirement for the Submission of Hospital Case Mix and Charge Data*. Hospital discharge data are available through the MDPH Massachusetts Community Health Information Profile (MassCHIP). While the MCR collects address at diagnosis for each individual in its database, the hospital discharge database does not include detailed address information. Therefore, an aggregate analysis of hospital discharge data was possible, providing hospitalization rates at the community-level for both Northampton and Easthampton.

Each hospitalization in Massachusetts is coded using the International Classification of Disease 9th Revision codes (ICD-9). The hospital discharge database of MassCHIP was searched using the specific ICD-9 code for asthma-related conditions, where asthma was the primary or associated cause of hospitalization for residents of Northampton and Easthampton.

These data were examined to determine how many times individuals from Northampton and Easthampton were hospitalized with asthma or an asthma-related condition each year during the six-year period of 2000 through 2005. The statistic reported is the Standardized Hospitalization Ratio (SHR). The SHR is analogous to the SIR in its calculation and interpretation. However, instead of counting individuals diagnosed with a given disease, the SHR utilizes the number of hospitalizations for a city or town. In this report, the SHR is the ratio of the number of hospitalizations observed in either

Northampton or Easthampton to the number that would be expected if the community's population had the same age-specific hospitalization rates as the statewide population, multiplied by 100. As with an SIR, if the SHR is above 100, this indicates that more hospitalizations occurred than where expected. Similarly, if the SHR is below 100, fewer hospitalizations occurred than were expected.

There are some limitations to the data on asthma-related hospitalizations. First, unlike cancer data that are available at a smaller geographic level (i.e. census tract level), the data for hospitalizations are only provided by MassCHIP at the city/town level or the zip code level. This makes it impossible to determine if one area of the city has more hospitalizations for a particular condition than another area of the city. The SHR can be used, therefore, to indicate if Northampton or Easthampton is experiencing a higher or lower rate of hospitalizations from asthma-related conditions than the state as a whole. Another limitation of the hospitalization data is that SHRs are based on the numbers of hospitalizations reported, not on the number of individuals hospitalized. For example, when the database is used to identify the number of asthma-related hospitalizations in Northampton or Easthampton during a specified time period, it would count one individual hospitalized ten times the same number of times as it would count ten individuals each hospitalized once. It is important to keep this information in mind when interpreting the hospital discharge data presented in this report.

The hospital discharge data for asthma-related hospitalizations in Northampton, Easthampton, and the state as a whole from 2000-2005 can be seen in Table 10. With the exception of one year in Northampton (2001), there were no statistically significant elevations in the Standardized Hospitalization Ratios (SHRs) for asthma-related

hospitalizations for residents of either Northampton or Easthampton. This means that rates of hospitalization for asthma and asthma-related conditions were at or near expected rates in both communities, based on the statewide experience of hospitalizations for these conditions during the same years. In 2001 in Northampton, 215 asthma-related hospitalizations were reported compared to approximately 187 expected; this difference was of borderline statistical significance.

V. CHILDHOOD LEAD POISONING

As part of this review, childhood lead poisoning data were also reviewed. Data on childhood blood lead levels are available from the MDPH Bureau of Environmental Health's Childhood Lead Poisoning Prevention Program (CLPPP).

Children exposed to lead through inhalation or ingestion may experience effects on their nervous system which could lead to slowed development, learning disabilities, and/or behavioral problems. Lead is a toxic soft metal which is used in construction, batteries, and bullets, as well as in solder and many alloys. It can be found in the environment due to its historic and widespread use in industry. Lead can be present in soil because of natural deposits, improper disposal by industry, or leaks from underground leaded gasoline tanks. A 1973 mandate implemented standards to phase out leaded gasoline in the United States, and in 1996 it became illegal to use leaded gasoline in an on-road vehicle. These measures helped to decrease blood-lead levels in the U.S. However, the most significant source of children's exposure relates to lead present in older homes and apartments, particularly those built before 1978, due to the use of lead in paint. Massachusetts law requires the removal of lead paint hazards in any home built before

1978 where children under the age of six live. Lead hazards include loose lead paint and lead paint on windows and other surfaces accessible to children.

Young children are particularly sensitive to the health effects of lead as they are more likely to display hand to mouth behavior, or pica. This can lead to increased ingestion of lead via contaminated paint chips or soil. The Bureau of Environmental Health's CLPPP manages the prevention, screening, diagnosis, and treatment of lead poisoning among children. By regulation, Massachusetts has a universal screening program requiring all children ages 1-3 to have their blood lead level analyzed annually and the results must be reported to the CLPPP program. Children who live in communities designated as "high risk" are required to have their blood tested for lead through age 4.

According to the U.S. Centers for Disease Control (CDC), a concentration of 10 micrograms per deciliter of lead in blood is considered to be a threshold level associated with potentially detrimental effects. A blood lead level below this concentration is considered to be a non-case. In Massachusetts, if a blood lead level falls between 10 and 15 μg/dL, education and outreach materials are sent to parents to assist in reducing potential exposures in the home. When the level exceeds 15 μg/dL, CLPPP offers case management services to the family, and if it exceeds 25 μg/dL, the level defined as lead poisoned in Massachusetts, CLPPP must provide case management, monitor medical care, and oversee environmental remediation in the home.

CLPPP screening data were analyzed for Northampton as a whole and CT 8222.00 as well as Easthampton as a whole and CT 8224.01. Percentages of screening tests for children in these areas whose blood lead levels fall into the five categories described

above are presented in Table 11. (It should be noted that these percentages are based on the number of screening tests performed, not the number of children tested; some children undergo multiple tests depending on the levels detected in their blood.) The distribution of screening test results in each CT in the respective communities was compared to the community-wide screening test results as well as statewide screening results, to assess whether the blood lead levels in children residing in the two census tracts containing the Northampton Regional Landfill appear different from the communities as a whole or the state as a whole.

The majority of children screened in the four geographic areas examined fall into the less than 10 ug/dL category. For Northampton, the distribution of blood lead level screening tests results is similar (but slightly lower) in CT 8222.00 as for the city as a whole or the state as a whole. For Easthampton, CT 8224.01 had consistently lower blood lead levels than the town as a whole and the state as whole. Therefore, it appears that children living in the census tract containing the Northampton Regional Landfill, and children in the Easthampton census tract bordering the Landfill, have blood lead levels similar to the communities of Northampton and Easthampton as a whole as well as the state.

CAP also evaluated the geographic distribution of residences of those children in both communities with blood lead screening tests results that exceeded 10 ug/dL. Their residential addresses were geocoded and the spatial distribution of their residences was examined using Geographic Information System (GIS) software. The patterns followed the population density patterns within each community, indicating that there is no particular area in either Northampton or Easthampton that appears to be experiencing a higher prevalence of blood lead levels that exceed the CDC threshold level.

VI. AUTISM

Massachusetts does not have a population-based registry for learning disabilities or developmental disorders such as autism. This means that there is no coordinated or systematic reporting on these disabilities, and as such, there is no way to estimate with precision the incidence or prevalence of such conditions, or whether they are occurring more frequently than might be expected in Northampton, Easthampton, or any other Massachusetts community. However, the Massachusetts Department of Education (MDOE) began collecting special education enrollment data on an individual level in late 2001. Using MDOE data, the BEH Environmental Epidemiology Program (EEP) issued a report in December 2005 on the estimated prevalence of autism in school districts across the state (MDPH 2005b). The report is entitled *Prevalence Estimates of Autism and Autism Spectrum Disorder in Massachusetts – Final Report.* Autism is a pervasive developmental disorder characterized by neurobehavioral traits including social disability, communication impairment, repetitive behaviors, and restricted interest. These prevalence estimates were based on educationally categorized reports of autism as recorded in a student's Individualized Education Program (IEP) plan prepared by special education departments in municipalities across the Commonwealth. While IEP data provide an estimate of autism prevalence in Massachusetts schools, they do not account for all diagnoses within the autism spectrum.

In its 2005 report, MDPH reported that the range of prevalence estimates for autism in Massachusetts, using statewide totals and IEP data, was 41 to 55 cases per 10,000 children, depending upon the school year (MDPH 2005b). The estimates were based on enrollment data from the three school years of 2002-2003, 2003-2004, and 2004-2005.

Autism counts and school enrollment totals include all students who are enrolled either at a public school, private school, collaborative, or out-of-state educational placement and receive some public funding for their special educational services from their home district. Not included in school enrollment are private school students who do not receive public funds to attend their school and children who are home-schooled.

For Northampton, Easthampton, and the state as a whole, autism prevalence estimates are presented in Table 12. In Northampton, the prevalence of autism was lower than that of the state as a whole during 2002-2003, higher during 2003-2004, and then lower in 2004-2005. In Easthampton, the prevalence of autism was higher than that of the state in each of the three years reported. However, for both Northampton and Easthampton, the prevalence estimates are not statistically significantly different from those for the state, meaning that they most likely represent natural or random variation in autism prevalence. Also, it is important to note that the prevalence estimates for both Northampton and Easthampton are relatively unstable, as indicated by their relatively large confidence intervals. In Northampton and Easthampton, between 10 and 14 and 9 and 14 students, respectively, were reported to have autism during the three school years for which data were reported.

VII. DISCUSSION

According to ACS statistics, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of three women and one out of two men develop cancer in their lifetime, but cancer will 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 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. These types of clusters may warrant further public health investigation.

Descriptive epidemiological analyses such as this can be useful in evaluating 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 non-environmental) 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 was to report on the patterns of certain types of cancer in the communities of Northampton and Easthampton and two of the census tracts closest to the Northampton

Regional Landfill (CT 8222.00 in Northampton and CT 8224.01 in Easthampton) to determine whether such patterns appear unusual.

Based on the information reviewed in this report, there does not seem to be an atypical pattern of the cancer types evaluated in Northampton, Easthampton, in either of the two CTs (8222.00 in Northampton or 8224.01 in Easthampton), or in the one-mile radius surrounding the Northampton Regional Landfill. As mentioned previously, the nine cancer types evaluated were chosen based on potential associations with contaminants of concern at the Northampton Regional Landfill and/or residents' concerns over suspected elevations of some cancer types. Although there were elevations in some cancer types during certain time periods, in most instances, the elevations represented one or two additional diagnoses of a particular type of cancer over what would be expected and the differences were not statistically significant, meaning that they could be due to chance and represent natural variability in rates.

Although there were three types of cancer in Northampton that were statistically significantly elevated in one time period – kidney cancer in females in Northampton during 1997-2004, leukemia in females in Northampton CT 8222.00 during 1992-1996, and breast cancer in females in Northampton in 1997-2004 (including CT 8222.00) – these cancer types were not elevated during the other three time periods evaluated. A review of risk factor information for Northampton females diagnosed with kidney cancer during the last time period evaluated showed that their ages at diagnosis and kidney cancer subtypes were consistent with what would be expected based on the epidemiological literature, and that smoking and occupation could have contributed to the incidence of this cancer type. In addition, MDPH accessed the MCR database to assess

the number of new kidney cancer diagnoses in Northampton for the years 2005 to the present, and learned that the incidence of kidney cancer appears to be about as expected in both genders, based on a crude estimation of the number of diagnoses expected. Similarly, a review of risk factor information for CT 8222.00 females diagnosed with leukemia during 1992-1996 did not show any unusual patterns. Four of the six women were diagnosed with the most common subtype of adult leukemia, acute myeloid leukemia (AML). One woman's treatment for a previous cancer may have contributed to her diagnosis, and smoking may also have contributed to the incidence of this cancer type among some women in CT 8222.00. The geographic distribution of those individuals diagnosed with leukemia in CT 8222.00 closely followed the patterns of population density in the CT.

MDPH reviewed cancer staging information for women diagnosed with breast cancer in Northampton for the period 1997-2004. Staging describes the extent of spread of an individual's cancer; from a public health perspective, earlier breast cancer staging reflects to some extent whether women are being screened early and regularly for breast cancer. In Northampton, approximately 33% of the women diagnosed with breast cancer between 1997 and 2004 were diagnosed with in-situ (non-invasive) breast cancer compared to 24% statewide. In Northampton CT 8222.00, approximately 43% of the women diagnosed with breast cancer between 1997 and 2004 were diagnosed with in-situ breast cancer.

According to the ACS and the medical literature, women who have had no children or who had their first child after age 30 have a slightly higher breast cancer risk. Having multiple pregnancies and becoming pregnant at an early age reduces breast cancer risk.

MDPH reviewed data on maternal age at first birth, available through MassCHIP, for Northampton. For 1990, 45% of the women in Northampton had their first child at age 30 or older compared to 28% statewide. Similarly, for 2006, 54% of the women in Northampton had their first child at age 30 or older compared to 39% statewide. The medical literature also reports that women of higher socioeconomic status tend to have a higher risk of breast cancer. According to the 2000 U.S. Census, approximately 49% of Northampton women age 25 or older have at least a Bachelor's or graduate-level degree compared to 31% statewide. The higher than expected breast cancer incidence in Northampton, particularly during the latest time period, appears to be in part correlated with educational level (a measure of socioeconomic status) as well as maternal age at first birth.

In Easthampton, the incidence of brain and CNS cancer fluctuated in males and females over the four time periods evaluated. None of the differences between the number of observed and expected diagnoses were statistically significant, meaning that the elevations may very likely represent natural variation in incidence rates. For the other eight cancer types evaluated, any differences noted between the numbers of observed diagnoses compared to the numbers expected most likely are due to chance. Although some elevations were noted in CT 8224.01, for three of the eight cancer types during different time periods, the elevations did not persist during the other three time periods evaluated and were not statistically significant.

The analysis of the geographic distribution of residence at diagnosis for individuals diagnosed with the nine cancer types evaluated in Northampton or Easthampton did not reveal any atypical spatial patterns, any patterns that would suggest a common factor

(environmental or non-environmental) played a primary role in the incidence of cancer in either community during the 23-year time period 1982-2004. Moreover, no unusual concentrations of individuals diagnosed with any of the cancer types were observed in the vicinity of the Northampton Regional Landfill.

Overall, a review of other readily available health outcome data, specifically for reproductive outcomes, asthma, and childhood blood lead poisoning, did not show any unusual patterns when the experiences of Northampton and Easthampton were compared to those of the state as a whole. While the numbers of children diagnosed with autism in Easthampton were consistently higher than the statewide rates, the differences were not statistically significant and were based on a small number of diagnoses.

Although the prevalence estimates of birth defects in Northampton and Easthampton were higher than the state, the differences were not statistically significant and the evaluation by the MDPH clinical geneticist did not identify any unusual spatial patterns or clustering of specific birth defects in either community. The prevalence of low birthweight babies in both communities was statistically significantly lower than the statewide prevalence for this outcome. The prevalence of pediatric asthma in the Robert K. Finn School in Northampton, the closest school to the Northampton Landfill, was similar to that of the state as a whole for 3 of the 4 years of surveillance. In the latest school year of surveillance, 2006-2007, asthma prevalence was statistically significantly elevated at the Finn School. MDPH followed up with the Northampton school nurse leader and Finn School nurse, to determine if there has been a change in reporting or enrollment, which might account for the change in prevalence estimates in later years. Both nurses felt that better parental reporting is most likely responsible, which can be

attributed to changes made to the health and medication forms completed by parents as well as an increased awareness of asthma among parents and their willingness to report. No elevations were seen among hospital discharge data for asthma-related hospitalizations among residents of Northampton or Easthampton, where asthma was the primary or secondary cause of admission. Childhood blood lead levels for the census tract containing the Northampton Regional Landfill as well as the Easthampton census tract that borders the landfill were consistent with those of the communities of Northampton and Easthampton as a whole as well as the state.

The purpose of this review of readily-available health outcome data was to assess if there were any unusual patterns of health outcomes in either Northampton or Easthampton or the census tracts containing the Northampton Regional Landfill. The health outcomes examined vary in nature and have different risk factors. This type of descriptive epidemiologic investigation serves as a screening-level assessment that can be used to determine the need for future public health actions.

VIII. CONCLUSIONS AND RECOMMENDATIONS

Overall, a review of cancer incidence data and other readily available health outcome data did not reveal any unusual patterns in either Northampton or Easthampton, in the census tracts in closest proximity to the Northampton Regional Landfill, or in the one-mile radius surrounding the Northampton Regional Landfill. While some exceptions were noted, when a particular incidence rate or the prevalence of a particular health outcome was higher than the statewide rate, the elevations did not persist over time and when examined closely, no unusual patterns emerged with respect to their spatial or

temporal distribution or available risk factor information. The MDPH/BEH will continue to monitor the incidence of cancer in the city of Northampton and town of Easthampton through the Massachusetts Cancer Registry and, as a separate effort, review and evaluate environmental sampling data to assess potential public health impacts.

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Figures

Figure 1
Census Tracts in Northampton and Easthampton, MA

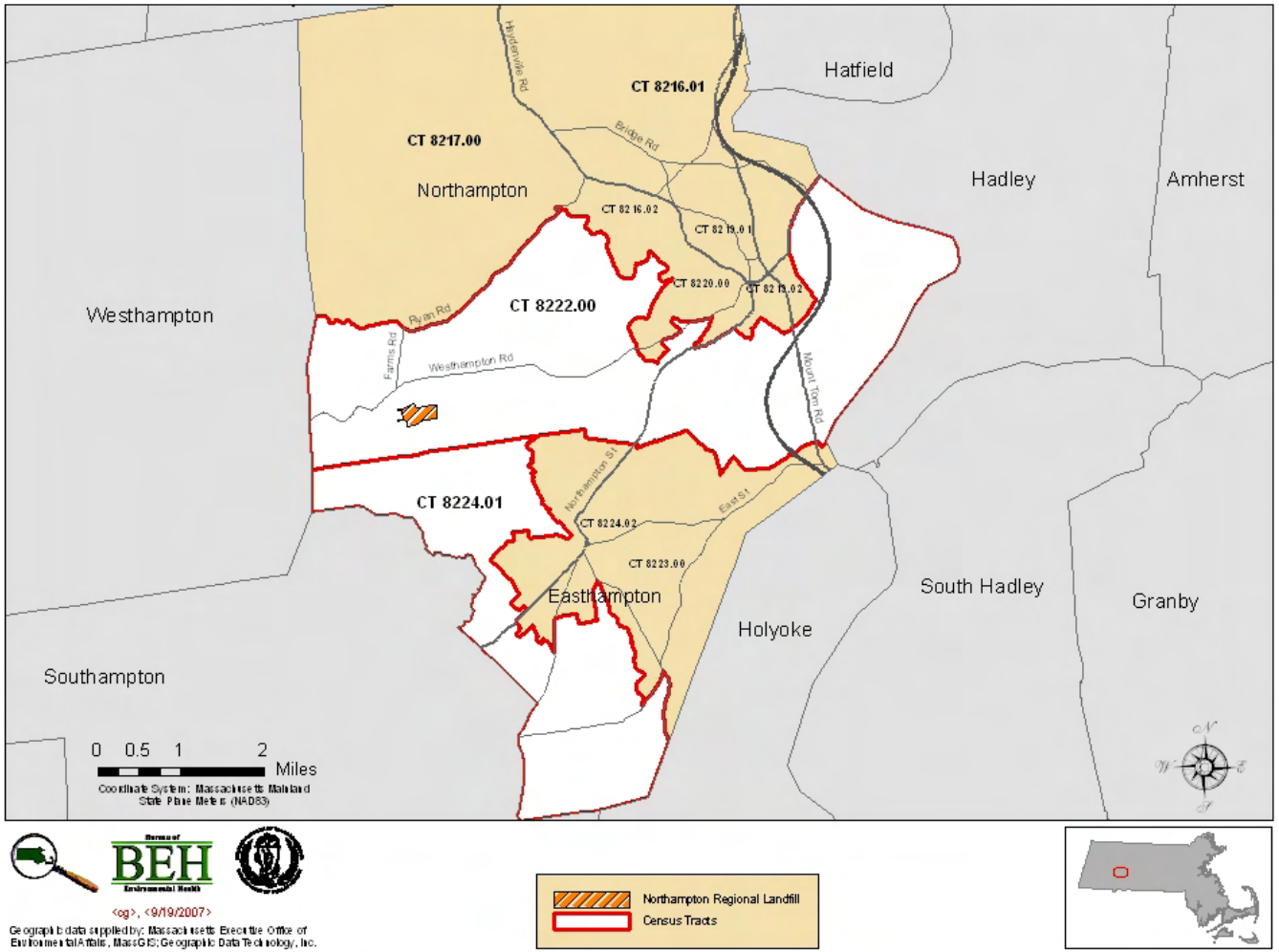


Figure2
One-mile surrounding the Northampton Regional Landfill

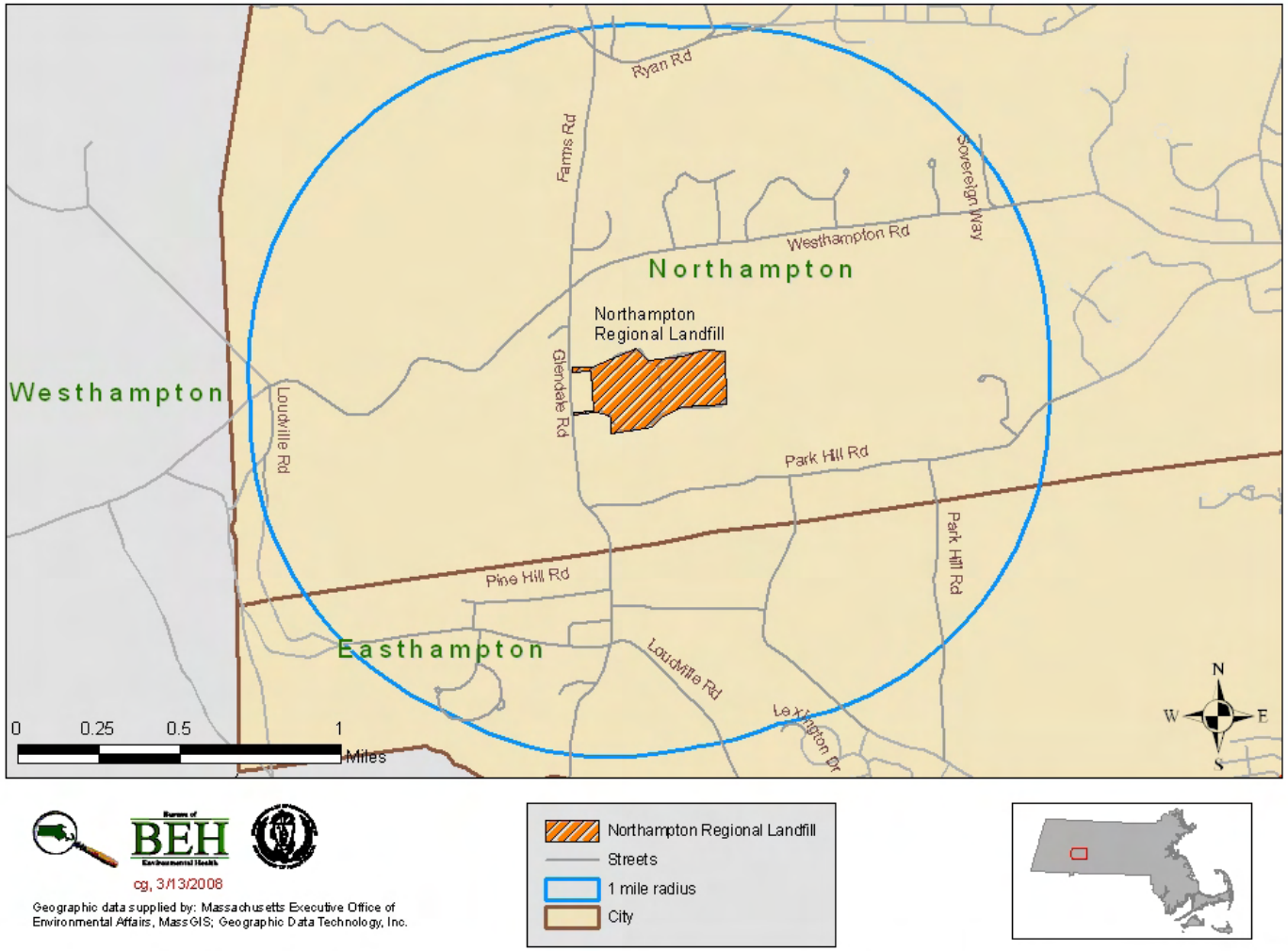
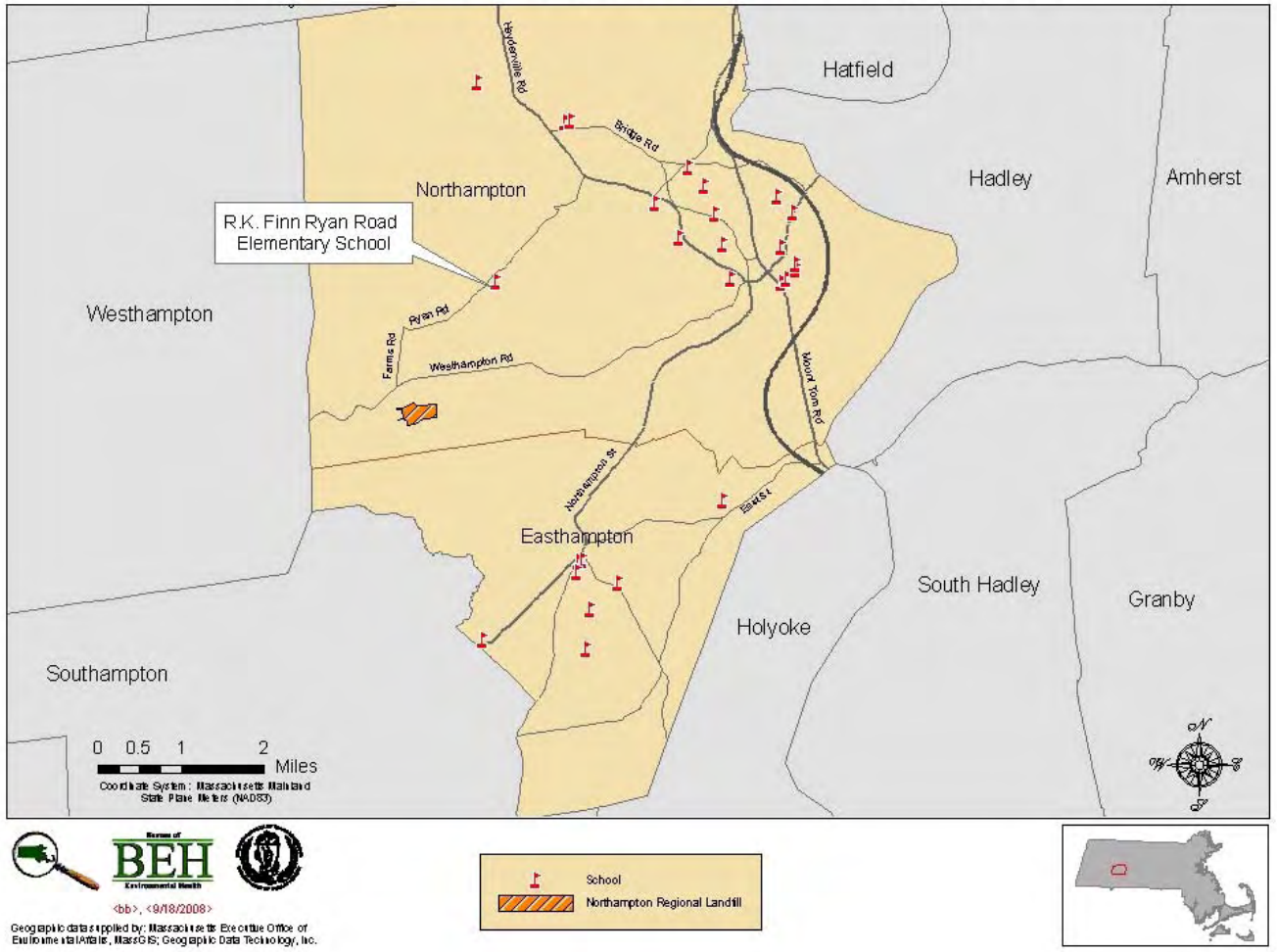


Figure 3
Schools in Northampton and Easthampton, MA



Tables

TABLE 1A
Cancer Incidence
Northampton, Massachusetts
1982-1986

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	19	29.1	65	39 -- 102	14	20.5	68	37 -- 115	5	8.7	57	19 -- 135
Brain and CNS[2]	5	13.8	36 *	12 -- 84	3	6.3	NC	NC -- NC	2	7.5	NC	NC -- NC
Breast[1]	112	94.1	119	98 -- 143	0	0.6	NC	NC -- NC	112	93.5	120	99 -- 144
Hodgkin lymphoma	6	5.9	103	37 -- 223	4	2.8	NC	NC -- NC	2	3.1	NC	NC -- NC
Kidney	14	11.6	121	66 -- 202	10	6.8	147	70 -- 269	4	4.8	NC	NC -- NC
Leukemia	9	12.7	71	32 -- 134	6	6.8	88	32 -- 192	3	6.0	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	3.1	NC	NC -- NC	0	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC
Lung and Bronchus	74	89.1	83	65 -- 104	45	58.2	77	56 -- 103	29	30.9	94	63 -- 135
Non-Hodgkin lymphoma	25	19.6	128	83 -- 189	12	9.6	125	65 -- 219	13	10.0	130	69 -- 223

[1] Includes In situ and Invasive cancers

[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1B
Cancer Incidence
Northampton, Massachusetts
1987-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	22	27.4	80	50 -- 122	11	19.5	57	28 -- 101	11	7.9	139	69 -- 248
Brain and CNS[2]	13	14.7	88	47 -- 151	9	6.7	135	62 -- 256	4	8.0	NC	NC -- NC
Breast[1]	111	108.0	103	85 -- 124	1	0.7	NC	NC -- NC	110	107.3	103	84 -- 124
Hodgkin lymphoma	5	6.2	81	26 -- 189	2	2.9	NC	NC -- NC	3	3.3	NC	NC -- NC
Kidney	17	15.5	110	64 -- 176	11	9.3	118	59 -- 211	6	6.1	98	36 -- 213
Leukemia	3	12.0	NC	NC -- NC	3	6.5	NC	NC -- NC	0	5.5	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	4	3.6	NC	NC -- NC	4	2.4	NC	NC -- NC	0	1.2	NC	NC -- NC
Lung and Bronchus	77	93.8	82	65 -- 103	48	56.9	84	62 -- 112	29	36.8	79	53 -- 113
Non-Hodgkin lymphoma	18	23.5	77	45 -- 121	11	11.8	94	47 -- 168	7	11.8	59	24 -- 122

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1C
Cancer Incidence
Northampton, Massachusetts
1992-1996

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	33	37.8	87	60 -- 123	19	26.6	71	43 -- 111	14	11.2	125	68 -- 210
Brain and CNS[2]	8	14.5	55	24 -- 109	4	6.8	NC	NC -- NC	4	7.6	NC	NC -- NC
Breast[1]	141	135.8	104	87 -- 122	0	1.0	NC	NC -- NC	141	134.8	105	88 -- 123
Hodgkin lymphoma	8	5.6	144	62 -- 284	4	2.8	NC	NC -- NC	4	2.7	NC	NC -- NC
Kidney	12	16.7	72	37 -- 126	5	10.0	50	16 -- 116	7	6.6	105	42 -- 217
Leukemia	15	14.2	106	59 -- 174	6	7.6	79	29 -- 173	9	6.6	136	62 -- 258
Liver and Intrahepatic Bile Ducts	0	4.7	NC	NC -- NC	0	3.1	NC	NC -- NC	0	1.5	NC	NC -- NC
Lung and Bronchus	84	97.4	86	69 -- 107	49	54.9	89	66 -- 118	35	42.5	82	57 -- 115
Non-Hodgkin lymphoma	23	27.5	84	53 -- 125	9	14.0	64	29 -- 122	14	13.6	103	56 -- 173

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 1D
Cancer Incidence
Northampton, Massachusetts
1997-2004

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	70	66.6	105	82 -- 133	42	47.3	89	64 -- 120	28	19.3	145	97 -- 210
Brain and CNS[2]	15	32.4	46	* 26 -- 76	6	13.8	43	* 16 -- 95	9	18.6	49	* 22 -- 92
Breast[1]	285	250.3	114	* 101 -- 128	0	1.6	NC	NC -- NC	285	248.7	115	* 102 -- 129
Hodgkin lymphoma	7	8.5	83	33 -- 171	4	3.9	NC	NC -- NC	3	4.5	NC	NC -- NC
Kidney	42	32.3	130	94 -- 176	19	19.2	99	60 -- 154	23	13.0	176	* 112 -- 265
Leukemia	20	28.8	70	42 -- 107	13	15	87	46 -- 148	7	13.8	51	20 -- 105
Liver and Intrahepatic Bile Ducts	7	12.9	54	22 -- 112	5	9.0	55	18 -- 129	2	3.8	NC	NC -- NC
Lung and Bronchus	175	175.8	100	85 -- 115	88	90	98	78 -- 120	87	85.8	101	81 -- 125
Non-Hodgkin lymphoma	42	49.1	85	62 -- 116	22	24.3	90	57 -- 137	20	24.8	81	49 -- 125

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2A
Cancer Incidence
Census Tract 8222.00 in Northampton, Massachusetts
1982-1986

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	4	4.0	NC	NC -- NC	4	3.0	NC	NC -- NC	0	1.1	NC	NC -- NC
Brain and CNS[2]	1	2.3	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.2	NC	NC -- NC
Breast[1]	13	14.3	85	48 -- 155	0	0.1	NC	NC -- NC	13	14.2	92	49 -- 156
Hodgkin lymphoma	2	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC
Kidney	5	1.8	282	91 -- 659	4	1.1	NC	NC -- NC	1	0.7	NC	NC -- NC
Leukemia	1	2.0	NC	NC -- NC	1	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung and Bronchus	10	13.4	75	36 -- 138	8	8.8	91	39 -- 179	2	4.5	NC	NC -- NC
Non-Hodgkin lymphoma	5	2.9	171	55 -- 398	3	1.6	NC	NC -- NC	2	1.3	NC	NC -- NC

[1] Includes In situ and Invasive cancers

[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2B
Cancer Incidence
Census Tract 8222.00 in Northampton, Massachusetts
1987-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	4	3.9	NC	NC -- NC	4	2.8	NC	NC -- NC	0	1.1	NC	NC -- NC
Brain and CNS[2]	1	2.5	NC	NC -- NC	0	1.2	NC	NC -- NC	1	1.3	NC	NC -- NC
Breast[1]	23	17.6	131	83 -- 196	1	0.1	NC	NC -- NC	22	17.5	126	79 -- 190
Hodgkin lymphoma	3	1.1	NC	NC -- NC	1	0.6	NC	NC -- NC	2	0.5	NC	NC -- NC
Kidney	2	2.5	NC	NC -- NC	2	1.6	NC	NC -- NC	0	0.9	NC	NC -- NC
Leukemia	0	1.9	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung and Bronchus	9	14.5	62	28 -- 118	7	9.0	78	31 -- 160	2	5.5	NC	NC -- NC
Non-Hodgkin lymphoma	1	3.8	NC	NC -- NC	1	2.1	NC	NC -- NC	0	1.7	NC	NC -- NC

[1] Includes In situ and Invasive cancers

[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2C
Cancer Incidence
Census Tract 8222.00 in Northampton, Massachusetts
1992-1996

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	9	6.0	149	68 -- 284	6	4.4	137	50 -- 298	3	1.6	NC	NC -- NC
Brain and CNS[2]	3	2.7	NC	NC -- NC	1	1.4	NC	NC -- NC	2	1.4	NC	NC -- NC
Breast[1]	18	25.3	71	42 -- 112	0	0.2	NC	NC -- NC	18	25.1	72	42 -- 113
Hodgkin lymphoma	2	1.1	NC	NC -- NC	2	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
Kidney	2	3.0	NC	NC -- NC	2	1.9	NC	NC -- NC	0	1.1	NC	NC -- NC
Leukemia	7	2.5	277	* 111 -- 571	1	1.4	NC	NC -- NC	6	1.1	555	* 203 -- 1208
Liver and Intrahepatic Bile Ducts	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung and Bronchus	13	16.5	79	42 -- 134	7	9.7	72	29 -- 149	6	6.9	87	32 -- 190
Non-Hodgkin lymphoma	5	4.8	104	33 -- 242	2	2.7	NC	NC -- NC	3	2.1	NC	NC -- NC

[1] Includes In situ and Invasive cancers

[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 2D
Cancer Incidence
Census Tract 8222.00 in Northampton, Massachusetts
1997-2004

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	12	12.2	98	51 -- 172	8	9.0	89	38 -- 175	4	3.2	NC	NC -- NC
Brain and CNS[2]	4	6.7	NC	NC -- NC	1	3.1	NC	NC -- NC	3	3.6	NC	NC -- NC
Breast[1]	68	52.9	128	* 100 -- 163	0	0.3	NC	NC -- NC	68	52.6	129	* 101 -- 164
Hodgkin lymphoma	1	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC
Kidney	12	6.6	183	94 -- 320	8	4.1	194	84 -- 383	4	2.4	NC	NC -- NC
Leukemia	4	5.5	NC	NC -- NC	4	3.1	NC	NC -- NC	0	2.4	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	1	2.6	NC	NC -- NC	1	2.0	NC	NC -- NC	0	0.6	NC	NC -- NC
Lung and Bronchus	38	33.2	114	81 -- 157	17	17.9	95	55 -- 152	21	15.4	136	85 -- 209
Non-Hodgkin lymphoma	9	9.4	96	44 -- 182	5	5.1	99	32 -- 231	4	4.3	NC	NC -- NC

[1] Includes In situ and Invasive cancers

[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3A
Cancer Incidence
Easthampton, Massachusetts
1982-1986

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	12	14.1	85	44 -- 148	7	10.2	68	27 -- 141	5	3.9	128	41 -- 299
Brain and CNS[2]	11	7.2	152	76 -- 272	6	3.5	172	63 -- 375	5	3.8	133	43 -- 310
Breast[1]	44	47.1	93	68 -- 125	1	0.3	NC	NC -- NC	43	46.8	92	66 -- 124
Hodgkin lymphoma	3	2.8	NC	NC -- NC	1	1.6	NC	NC -- NC	2	1.3	NC	NC -- NC
Kidney	10	5.9	168	81 -- 310	4	3.6	NC	NC -- NC	6	2.4	253	93 -- 551
Leukemia	5	6.4	78	25 -- 183	4	3.6	NC	NC -- NC	1	2.8	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	1.5	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.5	NC	NC -- NC
Lung and Bronchus	42	46.2	91	66 -- 123	27	30.1	90	59 -- 130	15	16.0	94	52 -- 155
Non-Hodgkin lymphoma	7	9.7	72	29 -- 149	5	5.0	100	32 -- 233	2	4.7	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3B
Cancer Incidence
Easthampton, Massachusetts
1987-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	12	14.0	86	44 -- 150	9	10.3	87	40 -- 166	3	3.7	NC	NC -- NC
Brain and CNS[2]	11	7.8	141	70 -- 252	7	3.8	185	74 -- 382	4	4.0	NC	NC -- NC
Breast[1]	50	55.9	89	66 -- 118	0	0.4	NC	NC -- NC	50	55.5	90	67 -- 119
Hodgkin lymphoma	3	3.0	NC	NC -- NC	2	1.6	NC	NC -- NC	1	1.4	NC	NC -- NC
Kidney	6	8.3	73	26 -- 158	5	5.1	98	31 -- 228	1	3.2	NC	NC -- NC
Leukemia	4	6.2	NC	NC -- NC	2	3.6	NC	NC -- NC	2	2.6	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	3	1.9	NC	NC -- NC	3	1.3	NC	NC -- NC	0	0.6	NC	NC -- NC
Lung and Bronchus	40	51.0	78	56 -- 107	22	31.4	70	44 -- 106	18	19.6	92	54 -- 145
Non-Hodgkin lymphoma	11	12.1	91	45 -- 163	4	6.4	NC	NC -- NC	7	5.7	123	49 -- 254

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3C
Cancer Incidence
Easthampton, Massachusetts
1992-1996

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	16	20.4	79	45 -- 128	11	14.7	75	37 -- 134	5	5.6	89	29 -- 207
Brain and CNS[2]	4	8.1	NC	NC -- NC	1	3.9	NC	NC -- NC	3	4.1	NC	NC -- NC
Breast[1]	64	73.6	87	70 -- 111	0	0.6	NC	NC -- NC	64	73.0	88	68 -- 112
Hodgkin lymphoma	4	2.9	NC	NC -- NC	3	1.7	NC	NC -- NC	1	1.3	NC	NC -- NC
Kidney	10	9.3	107	51 -- 197	6	5.7	104	38 -- 227	4	3.6	NC	NC -- NC
Leukemia	6	7.7	78	28 -- 169	5	4.3	115	37 -- 269	1	3.4	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	2	2.6	NC	NC -- NC	2	1.8	NC	NC -- NC	0	0.8	NC	NC -- NC
Lung and Bronchus	58	54.5	106	81 -- 138	29	31.1	93	63 -- 134	29	23.4	124	83 -- 178
Non-Hodgkin lymphoma	7	14.9	47 *	19 -- 97	4	7.9	NC	NC -- NC	3	7.0	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 3D
Cancer Incidence
Easthampton, Massachusetts
1997-2004

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	30	38.0	79	53 -- 113	24	27.4	88	56 -- 130	6	10.6	57	21 -- 123
Brain and CNS[2]	23	18.4	125	79 -- 187	8	8.2	97	42 -- 192	15	10.2	147	82 -- 242
Breast[1]	138	142.4	97	81 -- 114	1	0.9	NC	NC -- NC	137	141.5	97	81 -- 114
Hodgkin lymphoma	5	4.4	113	37 -- 265	3	2.4	NC	NC -- NC	2	2.0	NC	NC -- NC
Kidney	18	18.6	97	57 -- 153	13	11.2	116	62 -- 198	5	7.4	68	22 -- 158
Leukemia	16	16.2	99	56 -- 160	9	8.8	102	47 -- 194	7	7.4	95	38 -- 195
Liver and Intrahepatic Bile Ducts	7	7.4	95	38 -- 196	6	5.3	113	41 -- 246	1	2.1	NC	NC -- NC
Lung and Bronchus	92	101.4	91	73 -- 111	59	52.2	113	86 -- 146	33	49.1	67 *	46 -- 94
Non-Hodgkin lymphoma	32	28.0	114	78 -- 162	17	14.3	119	69 -- 191	15	13.7	109	61 -- 181

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 4A
Cancer Incidence
Census Tract 8224† in Easthampton, Massachusetts
1982-1986

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	5	7.7	65	21 -- 151	2	5.7	NC	NC -- NC	3	2.0	NC	NC -- NC
Brain and CNS[2]	8	4.3	185	80 -- 365	4	2.1	NC	NC -- NC	4	2.2	NC	NC -- NC
Breast[1]	23	26.4	87	55 -- 131	0	0.2	NC	NC -- NC	23	26.2	88	56 -- 131
Hodgkin lymphoma	2	1.9	NC	NC -- NC	0	1	NC	NC -- NC	2	0.8	NC	NC -- NC
Kidney	7	3.4	206	82 -- 423	3	2.1	NC	NC -- NC	4	1.3	NC	NC -- NC
Leukemia	4	3.8	NC	NC -- NC	3	2.2	NC	NC -- NC	1	1.6	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung and Bronchus	22	25.9	85	53 -- 129	14	17.1	82	45 -- 137	8	8.8	91	39 -- 180
Non-Hodgkin lymphoma	6	5.5	109	40 -- 237	4	3	NC	NC -- NC	2	2.6	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

†The CT in Easthampton in closest proximity to the Northampton Regional Landfill during this time period was CT 8224. In the 1990 U.S. Census, Easthampton CT 8224 was split into two CTs: 8224.01 and 8224.02. CT 8224.01 is the closer of the two CTs to the landfill.

TABLE 4B
Cancer Incidence
Census Tract 8224† in Easthampton, Massachusetts
1987-1991

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	6	7.8	77	28 -- 167	3	5.8	NC	NC -- NC	3	2.0	NC	NC -- NC
Brain and CNS[2]	8	4.8	168	72 -- 331	6	2.4	253	92 -- 551	2	2.4	NC	NC -- NC
Breast[1]	33	32.6	101	70 -- 142	0	0.2	NC	NC -- NC	33	32.4	102	70 -- 143
Hodgkin lymphoma	1	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC	1	0.9	NC	NC -- NC
Kidney	5	4.8	103	33 -- 241	4	3.1	NC	NC -- NC	1	1.8	NC	NC -- NC
Leukemia	2	3.7	NC	NC -- NC	1	2.2	NC	NC -- NC	1	1.5	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	3	1.1	NC	NC -- NC	3	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung and Bronchus	22	29.5	75	47 -- 113	10	18.5	54	26 -- 100	12	11.0	109	56 -- 190
Non-Hodgkin lymphoma	5	7.2	70	22 -- 163	2	4.0	NC	NC -- NC	3	3.2	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

†The CT in Easthampton in closest proximity to the Northampton Regional Landfill during this time period was CT 8224. In the 1990 U.S. Census, Easthampton CT 8224 was split into two CTs: 8224.01 and 8224.02. CT 8224.01 is the closer of the two CTs to the landfill.

TABLE 4C
Cancer Incidence
Census Tract 8224.01† in Easthampton, Massachusetts
1992-1996

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	7	4.9	143	57 -- 295	4	3.6	NC	NC -- NC	3	1.3	NC	NC -- NC
Brain and CNS[2]	3	2.4	NC	NC -- NC	1	1.2	NC	NC -- NC	2	1.2	NC	NC -- NC
Breast[1]	20	21.8	92	56 -- 142	0	0.1	NC	NC -- NC	20	21.7	92	56 -- 143
Hodgkin lymphoma	1	0.9	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
Kidney	2	2.5	NC	NC -- NC	1	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC
Leukemia	2	2.1	NC	NC -- NC	2	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung and Bronchus	17	13.8	123	72 -- 197	12	7.9	152	78 -- 265	5	5.9	84	27 -- 197
Non-Hodgkin lymphoma	0	4.0	NC	NC -- NC	0	2.3	NC	NC -- NC	0	1.8	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

† In the 1990 U.S. Census, Easthampton CT 8224 was split into two CTs: 8224.01 and 8224.02. The CT in closest proximity to the Northampton Regional Landfill during this time period was CT 8224.01.

TABLE 4D
Cancer Incidence
Census Tract 8224.01† in Easthampton, Massachusetts
1997-2004

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder[1]	10	10.5	95	45 -- 175	9	7.7	116	53 -- 221	1	2.8	NC	NC -- NC
Brain and CNS[2]	5	6.0	83	27 -- 195	3	2.7	NC	NC -- NC	2	3.3	NC	NC -- NC
Breast[1]	47	47.9	98	72 -- 130	0	0.3	NC	NC -- NC	47	47.6	99	73 -- 131
Hodgkin lymphoma	2	1.4	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.6	NC	NC -- NC
Kidney	7	5.8	120	48 -- 247	4	3.6	NC	NC -- NC	3	2.2	NC	NC -- NC
Leukemia	5	4.7	106	34 -- 248	5	2.7	187	60 -- 437	0	2.0	NC	NC -- NC
Liver and Intrahepatic Bile Ducts	1	2.3	NC	NC -- NC	1	1.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Lung and Bronchus	26	29	90	59 -- 131	15	15.3	98	55 -- 162	11	13.6	81	40 -- 144
Non-Hodgkin lymphoma	8	8.2	97	42 -- 191	4	4.4	NC	NC -- NC	4	3.8	NC	NC -- NC

[1] Includes In situ and Invasive cancers
[2] Includes Benign and Malignant tumors

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, Bureau of Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

† In the 1990 U.S. Census, Easthampton CT 8224 was split into two CTs: 8224.01 and 8224.02. The CT in closest proximity to the Northampton Regional Landfill during this time period was CT 8224.01.

Table 5:	
Streets Included in the One-Mile Radius Surrounding the Northampton Regional Landfill:	
Northampton:	Easthampton:
Bayberry Lane Brisson Drive Drury Lane Easthampton Road Farms Road Glendale Road Ladyslipper Lane Loudville Road Oliver Street Park Hill Pine Valley Road Ryan Road Sovereign Way Sylvan Lane Tiffany Lane Unnamed Street Westhampton Road Woodland Drive	Ashley Circle Clark Lane Loudville Road Oliver Street Park Hill Road Pine Hill Road Russell Lane Torrey Street Torrey Way Upland Meadow Way

Table 6: Birth Defect Prevalence Rates: Northampton/Easthampton Compared to Massachusetts, 2000-2003				
2000-2003	#	Rate*	CIs	
Northampton	16	186.1	106.3	302.1
Easthampton	12	177.5	91.7	310.1
MA	4813	148.8	144.7	153.1
*Rate per 10,000 live births				

Table 7: Low Birth Weight* Prevalence Rates: Northampton/Easthampton Compared to Massachusetts, 2000-2005				
2000-2005	#	Rate**	CIs	
Northampton	46	360	260.3	457.3
Easthampton	48	490	358.3	645.8
MA	35,853	750	742.52	757.48

*Low Birth Weight is considered to be below 2500 grams (5 lbs.)
**Rate per 10,000 births

Table 8: Pediatric Asthma Prevalence by School, Northampton				
	2003-2004	**2004-2005**	**2005-2006**	**2006-2007**
Northampton Schools				
Bridge Street	15.6 (11.6-19.6)*	13.7 (9.8-17.6)	15 (11.0-19.0)*	16.0 (11.8-20.3)*
Jackson Street	11.5 (8.0-15.0)	8.4 (5.5-11.3)	13.7 (10.2-17.3)	18.2 (14.2-22.2)*
Leeds	5.2 (2.9-7.6)*	8.3 (5.3-11.3)	7.4 (4.6-10.2)*	11.4 (7.9-14.8)
Robert K Finn	9.7 (6.3-13.1)	10.3 (6.8-13.9)	8.2 (5.0-11.4)	15.8 (11.4-20.2)*
John F Kennedy Jr High	14.5 (11.9-17.0)*	14.6 (12.1-17.1)*	11.8 (9.4-14.2)	11.1 (8.7-13.5)
Clarke School for the Deaf	9.5 (2.3-16.8)	16.4 (7.1-25.7)	7.3 (0.4-14.1)	16.4 (6.6-26.1)
Montessori School of Northampton	n/r	3.7 (0.0-8.7)*	8.5 (0.5-16.5)	8.3 (0.51-16.1)
Smith College Campus School	11.4 (7.6-15.2)	n/r	9.4 (5.9-12.8)	10.4 (6.8-14.0)
Solomon Schechter Day School	n/r	4.1 (0.2-8.0)*	6.3 (1.4-11.1)	6.4 (0.97-11.8)
Community Prevalence	**11.5 (10.2-13.0)***	**11.6 (10.3-12.9)***	**10.8 (9.6-12.0)**	**n/a**
Statewide Prevalence	**9.5 (9.4-9.6)**	**10 (9.9-10.1)**	**10.6 (10.5-10.7)**	**10.8 (10.7-10.8)**
n/r = not reported; n/a = not available				
***indicates a statistically significant difference from the state rate for that school year**				
Schools with enrollment < 25 were not included for analysis				

Table 9: Pediatric Asthma Prevalence by School, Easthampton				
	2003-2004	**2004-2005**	**2005-2006**	**2006-2007**
Easthampton Schools				
Center School	7.5 (4.0-11.1)	6.0 (2.7-9.3)*	16.7 (11.4-21.9)*	16.2 (11.0-21.5)*
Maple	8.3 (4.5-12.0)	8.4 (4.7-12.1)	7.5 (4.0-11.1)	11.8 (7.6-16.1)
Neil A Pepin	6.2 (2.6-9.7)	8.0 (4.0-12.0)	18.3 (12.4-24.2)*	15.8 (10.2-21.3)
White Brook Middle School	4.2 (2.6-5.8)*	5.0 (3.2-6.8)*	n/r	n/r
Calvary Baptist	n/r	0	7.9 (0.0-16.5)	5.7 (0-13.4)
Notre Dame- Immaculate Conception	5.9 (1.9-9.8)	0	8.0 (3.5-12.6)	11.7 (5.5-17.8)
Williston Northampton	12.1 (5.4-18.8)	9.8 (3.7-15.9)	13.8 (6.2-21.3)	11.5 (4.4-18.6)
Community Prevalence	**5.6 (4.5-6.9)***	**5.6 (4.5-6.8)***	**12.5 (10.2-14.8)**	**n/a**
Statewide Prevalence	**9.5 (9.4-9.6)**	**10 (9.9-10.1)**	**10.6 (10.5-10.7)**	**10.8 (10.7-10.8)**

n/r = not reported; n/a = not available

***indicates a statistically significant difference from the state rate for that school year**

Schools with enrollment < 25 were not included for analysis

TABLE 10
Asthma-Related Hospitalization Incidence**
Easthampton and Northampton, Massachusetts
2000-2005

	Easthampton				Northampton			
	Obs	**Exp**	**SHR**	**95% CI**	**Obs**	**Exp**	**SHR**	**95% CI**
2000	110	98.0	112	92 — 135	171	176.8	97	83 — 112
2001	112	104.8	107	88 — 129	215	187.3	115	100 — 131
2002	133	113.7	117	98 — 139	215	202.5	106	92 — 121
2003	134	130.5	103	86 — 122	230	231.1	99	87 — 113
2004	122	132.9	92	76 — 220	230	235.7	98	85 — 111
2005	126	140.6	90	75 — 107	200	248.4	81	70 — 92

Note: SHRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
**indicates that asthma is the primary or secondary cause of hospitalization

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

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

Table 11: Childhood Blood Lead Level Test Results*
Northampton, Easthampton,Census Tracts 8222.00, 8224.01, & Massachusetts
1990-2006

	Northampton		CT 8222.00	
Blood Lead Level (ug/dL)	**Count of Tests**	**Percent of Total Tests**	**Count of Tests**	**Percent of Total Tests**
<10	6216	86.70%	1541	89.60%
10-14	682	9.50%	120	6.90%
15-19	159	2.20%	30	1.70%
20-24	50	0.70%	12	0.70%
25+	61	0.80%	16	0.90%
Total:	7168		1719	

	Easthampton		CT 8224.01	
Blood Lead Level (ug/dL)	**Count of Tests**	**Percent of Total Tests**	**Count of Tests**	**Percent of Total Tests**
<10	4412	86.30%	1453	94.70%
10-14	465	9.10%	61	3.90%
15-19	130	2.50%	16	1%
20-24	48	0.90%	0	n/a
25+	54	1.10%	4	0.20%
Total:	5109		1534	

	Massachusetts	
Blood Lead Level (ug/dL)	**Count of Tests**	**Percent of Total Tests**
<10	2281212	87.70%
10-14	205725	7.90%
15-19	63161	2.40%
20-24	25621	1.00%
25+	25707	1.00%
Total:	2601426	

* All venous and capillary tests including non-cases, cases, and unconfirmed cases

Table 12: Autism Prevalence: Northampton, Easthampton, & Massachusetts
School Years 2002-2003, 2003-2004, and 2004-2005

	Northampton				Easthampton				Massachusetts			
	Autism Count	Total Enrollment	Rate*	95% CI	Autism Count	Total Enrollment	Rate*	95% CI	Autism Count	Total Enrollment	Rate*	95% CI
2002-2003	10	2939	34	(13, 55)	9	1723	52	(18, 86)	4080	991,641	41	(40, 42)
2003-2004	16	3016	53	(27, 79)	14	1663	84	(40, 128)	4876	991,478	49	(48, 51)
2004-2005	14	3024	46	(22, 70)	11	1655	66	(27, 106)	5467	986,662	55	(54, 57)

*Prevalence per 10,000 students based on IEP data and enrollment

APPENDIX A

Cancer Incidence Coding Definitions

APPENDIX A

ICD CODES USED FOR THIS REPORT

Cancer Site / Type	ICD-O-3[1]	
	Primary Site Codes	**Histology Type Codes[2]**
Urinary Bladder	C67.0 - C67.9	all except 9590 - 9989
Brain & Other Nervous System	C70.0 - C72.9	all except 9590 - 9989
Breast	C50.0 - C50.9	all except 9590 - 9989
Hodgkin Lymphoma	C00.0 - C80.9	includes 9650 – 9667
Kidney & Renal Pelvis	C64.9, C65.9	all except 9590 - 9989
Leukemia	C00.0 - C80.9	includes 9733, 9742, 9800-9820, 9826,
	C42.0, C42.1, C42.4	9831-9948, 9963-9964 includes 9823, 9827
Liver and Intrahepatic Bile Ducts	C22.0, C22.1	all except 9590 - 9989
Lung and Bronchus	C34.0 - C34.9	all except 9590 - 9989
Non-Hodgkin('s) Lymphoma	C00.0 - C80.9	includes 9590 - 9595, 9670 – 9729
	all sites except C42.0, C42.1, C42.4	includes 9823, 9827

[1] *International Classification of Diseases for Oncology, 3d Ed.* (2) (includes codes added since publication)

[2] Except where noted, only invasive cancers (those with invasive behaviors) are included in this report.

APPENDIX B

Risk Factor Information for Selected Cancer Types

The American Cancer Society estimates that bladder cancer will affect 61,420 people in the U.S. in 2006, accounting for 6% of all cancers diagnosed in the United States among men and 2% among women. In Massachusetts, bladder cancer accounts for approximately 5% of all cancers diagnosed among males and females combined (ACS 2006a). Males are four 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 nearly 90% of people with this cancer are over the age of 55 at the time of diagnosis (ACS 2006b).

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 2006a). 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 beta-naphthylamine, increases the risk of bladder cancer (ACS 2006b). 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 2006a; 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 2006b).

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 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 2006a; Silverman et al. 1996). Long term exposure to chlorinated by-products in drinking water

has also been suggested to increase the risk of developing bladder cancer, particularly among men (Villanueva 2003).

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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 25% of all reported primary brain tumors and the majority of spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the neurons 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 2006a). The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,820 people (7,260 men and 5,560 women) will die from this disease in 2006 (ACS 2006).

Brain and spinal cord cancers account for over 20% of malignant tumors diagnosed among children aged 0-14 (ACS 2006b). About half of all childhood brain tumors are astrocytomas and 25% are primitive neuroectodermal tumors (PNET), which spread along the spinal cord and the meninges (ACS 2006b). 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 2006a). 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 2006a). 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 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 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 2006a). 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 2006b). 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 2006b). 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).

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Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the American Cancer Society, female breast cancer incidence in Massachusetts is the fourth highest among all states (ACS 2006). The breast cancer incidence rate has been rising in the United States since the 1980s. However, the rate of increase slowed in the 1990s compared to the 1980s. Most recently, breast cancer incidence has only increased in women over 50 years of age (ACS 2006a). A similar trend occurred in Massachusetts and there was even a significant decrease in incidence (2.5%) between 1998 and 2002 (MCR 2005).

In the year 2006, approximately 212,920 women in the U.S. will be diagnosed with breast cancer (ACS 2006). 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 et al. 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 three-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 5 to 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. Women who inherit BRCA1 or BRCA2 gene mutations have up to an 80% chance of developing breast cancer at some point in their lifetimes (ACS 2006).

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; ACS 2006). Women who experience menopause at a later age (after the age of 55) have a slightly elevated risk for developing the disease (ACS 2006). 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 and Leake 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 2006; 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. 1997; ACS 2006). 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 2006). Despite this

association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al. 1997).

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 lymphoma and non-Hodgkin's Lymphoma) (ACS 2006). 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 suggest 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). In particular, 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 occupational exposures 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 et al. 1999; Aschengrau et al. 1998; Lewis-Michl et al. 1996). This

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, 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 lymphoma is a form of cancer that occurs in the lymphatic system, which accounts for a major part of our immune defense. Hodgkin lymphoma occurs specifically in the *Reed-Sternberg* cells, a type of B lymphocyte, or white blood cell; while other lymphomas (non-Hodgkin types) occur in different cells. The most common type of the disease is nodular sclerosis Hodgkin's, which accounts for about 70-80% of diagnoses in developed countries; other types include mixed cellularity Hodgkin's, lymphocyte-rich Hodgkin's, and lymphocyte-poor Hodgkin's. These four types of the disease make up what is known as classical Hodgkin lymphoma, and collectively represent 95% of all diagnoses of the disease. The other 5% of diagnoses fall into the category of nodular lymphocyte predominance Hodgkin lymphoma (ACS 2007). The American Cancer Society estimates that there will be approximately 8,190 new cases of Hodgkin lymphoma in the U.S. in 2007, accounting for less than 1% of all cancer types, and approximately 1,070 deaths (ACS 2007). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS 2007).

Epidemiologic studies have shown that Hodgkin lymphoma is more common among men than women and more common among white people than African-Americans. People of Jewish descent appear to be at higher risk of Hodgkin lymphoma compared to people of non-Jewish descent (Mueller 1996). Although the disease is relatively rare among children, two peaks in the bimodal 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 (ACS 2007).

Slightly higher rates of Hodgkin lymphoma 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 lymphoma occurs at a much lower rate than non-Hodgkin lymphomas among this group of individuals (ACS 2007).

Scientists have identified few risk factors that may make a person more likely to develop Hodgkin lymphoma. The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin lymphoma may be involved for each group. Hodgkin lymphoma 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 lymphoma is associated with an infectious agent (Mueller 1996).

A four times higher rate of Hodgkin lymphoma has been observed in individuals who have had infectious mononucleosis, an infection that is caused by the Epstein-Barr virus (EBV). The virus is present in the lymph nodes of approximately half of the individuals diagnosed with Hodgkin lymphoma the other half have no evidence of EBV in their Hodgkin cells (ACS 2007). 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 lymphoma, results of antibody studies are inconsistent and these viruses do not appear to be related to the risk of Hodgkin lymphoma (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 lymphoma. 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 lymphoma does not seem to be caused by lifestyle (e.g., diet, smoking status), or environmental factors (ACS 2007).

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Risk Factor Information for 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 2006). The American Cancer Society estimates that there will be approximately 38,890 cases of kidney and upper urinary tract cancer, resulting in more than 12,840 deaths in 2006 (ACS 2006). Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in individuals between 55 and 84 years of age (ACS 2006). 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, 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 about 40% (ACS 2006). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed (Yuan et al. 1998).

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 2006). 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 (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 2000). 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 (McLaughlin et al. 1996).

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 2006; 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 2006; Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS 2006). 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 7 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 2006a).

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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 2006, leukemia is expected to affect approximately 35,070 individuals in the United States (20,000 males and 15,070 females) in the United States, resulting in 22,280 deaths. Acute cases of leukemia are slightly more common that chronic, 15,860 and 14,520 respectively. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, 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 (approximately 11,700 cases) and CLL (approximately 9,560 cases). Incidences of ALL have increased approximately 1.8% per year since 1988 while incidences of CLL have decreased approximately 1.9% each year since 1988. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority (74%) of these cases are of the ALL type (ACS 2006a).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals, and 1300 (one-third) of total cases of ALL will occur in adults. ALL risk is lowest for adults aged 25 through 50 and then begins to pick up (ACS 2006b). 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 (ACS 2006b). Significant radiation exposure (e.g., diagnostic x-rays) within the first few months of development may carry up to a 5-fold increased risk of developing ALL (ACS 2006b). 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). There is conflicting evidence about 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 2006b).

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 2006c). 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 2006c). 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). 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 2006d). 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 (a chemical in gasoline and cigarette smoke), 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 2006d). 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). 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 (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, such as Down's syndrome (ACS 2006d). 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 2006e). 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). 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 2006f). 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 2006f). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell 1997).

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An estimated 18,510 people in the U.S. (12,600 men and 5,910 women) will be diagnosed with liver and intrahepatic bile duct cancer in 2006, accounting for approximately 1% of all new cancers (ACS 2006). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver and accounts for about 75% of all cases. Rarer forms of malignant liver cancer include the fibrolamellar subtype of HCC, cholangiocarcinoma, and angiosarcomain adults and hepatoblastoma in children. Cholangriocarcinomas account for approximately 10% to 20% of all primary liver cancers and people with gallstones, gall bladder inflammation, chronic ulcerative colitis (long-standing inflammation of the large bowel) or chronic infection with certain types of parasitic worms are at an increased risk for developing this cancer. Hepatoblastoma is a rare cancer that forms usually in children under age 4 and has a 90% survival rate with early detection (ACS 2006a).

In some developing countries, HCC is most common type of cancer diagnosed particularly in East Asia and Africa. Incidence in the United States had been increasing up to 1999. Recently, the rate has become more stable (ACS 2006a). Rates of HCC in the U.S. had increased by 70% during the 1980s and 1990s (Yu et al. 2000). Similar trends were observed in Canada and Western Europe. The primary reason for the higher rates observed during those years was 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 three times more likely to develop HCC than women. Much of this is likely due to differences in lifestyle factors which increase a person's risk for developing liver cancer (ACS 2006a). Although 85% of individuals diagnosed with liver cancer are between 45 and 85 years of age, the disease can occur in persons of any age (ACS 2006a).

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 2006a). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al. 2000). In the United States, HBV accounts for less than a quarter of the cases 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 2006a).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that is the result of scar tissue formation on the liver, which can 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 2006a). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS 2006a). 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 2006a; London and McGlynn 1996). These chemicals may also increase the risk of cholangiocarcinoma and 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 2006a). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS 2006a; 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 2006a; London and McGlynn 1996). Long-term anabolic steroid use may slightly increase the risk of HCC (ACS 2006a). 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 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 2005). 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 and bronchus cancer will be diagnosed in 174,470 people (92,700 cases in men and 81,770 in women) in the U.S. in 2006, accounting for about 12% of all new cancer diagnoses. For purposes of treatment, lung cancer is divided into two clinical groups: small cell lung cancer (13%) and non-small cell lung cancer (87%) (ACS 2006). 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 2005). In Massachusetts, an estimated 4,070 individuals will be diagnosed with lung and bronchus cancer in 2006. Incidence rates for lung and bronchus cancer in Massachusetts from 1998 through 2002 were 86.5 per 100,000 and 60.4 per 100,000 for males and females, respectively (ACS 2006). 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 are approaching a plateau, after a long period of increase. This is likely because decreasing smoking patterns among women have lagged behind those of men (ACS 2006). 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).

Approximately 87% of all lung cancers are caused directly by smoking cigarettes and some 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 and ten years after smoking cessation the risk is reduced by one-third of what it would have been had smoking continued. However, former smokers still carry a greater risk than those who have never smoked. There is no evidence that smoking low tar or "light" cigarettes reduces the risk of lung cancer and mentholated cigarettes are thought to increase the risk of lung cancer. Additionally, breathing secondhand smoke also increases an individual's risk of developing lung cancer. A nonsmoking spouse of a smoker has a 30% greater risk of developing lung cancer than the spouse of a nonsmoker (ACS 2005).

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 2005). Underground miners exposed to

radon and uranium are at an increased risk for developing lung cancer (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; Pohlabeln 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 2005; 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).

Some types of pneumonia may increase the risk of lung cancer due to scarred lung tissue (ACS 2002). In addition, people who have had lung cancer have a higher risk of developing another tumor. A family history of lung cancer also increases an individual's risk this is due to an abnormality on chromosome 6 (ACS 2005).

Air pollution may increase the risk of developing lung cancer in some cities. However, this risk is much lower than that due to cigarette smoking (ACS 2005).

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 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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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 lymphoma (NHL) is a classification of all lymphomas except Hodgkin lymphoma. 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 2006a). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller 1996). NHL can occur at any age. 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 2006a). The American Cancer Society estimates that approximately 58,870 Americans will be diagnosed with NHL in 2006 with 30,680 diagnoses occurring among males and 28,190 diagnoses occurring among females (ACS 2006b).

Overall, between 1973 and 1997, the incidence of NHL in the U.S. grew 81% (Garber 2001), although over the past 20 years, the incidence rate appears to have stabilized (ACS 2006b). 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, 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).

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 herpes virus 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. 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 2006). 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, 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 2006a). 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 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 2006a), there is little evidence for an increased risk of lymphoma due to radiation (Scherr and Mueller 1996).

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 review of five 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 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).

Recent studies have also linked Hepatitis C virus infection with increased risk for NHL, particularly the subtype of Waldenstrom's macroglobulinemia. However, these studies focused on individuals within the Veteran's Administration healthcare system, a limited group which does not reflect the general population (Giordano et al., 2007).

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 2006a).

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