Appendix F

Additional Analyses in Response to Public Comments

Assessment of Cancer Incidence in Belmont, Massachusetts, and Surrounding Communities

TABLE OF CONTENTS

List of Tables

I. Introduction and Background

On October 20, 2005, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH) released for public comment the Public Health Assessment (PHA) report titled *Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999* (MDPH 2005). This investigation was conducted in response to requests by the Belmont Department of Health, community residents, and in response to a legislative directive, to address concerns about suspected increases in cancer incidence in the vicinity of the Cambridge Plating Company and a possible relationship to environmental contamination. The 2005 report describing that investigation provided an evaluation of six cancer types in the town of Belmont and the surrounding communities of Cambridge, Arlington, and Watertown. Cancers of the kidney, liver, lung and bronchus, and pancreas as well as leukemia and non-Hodgkin's lymphoma (NHL) were evaluated based on potential associations with contaminants of concern at the Cambridge Plating site and/or residents' concerns over suspected elevations in some cancer types.

The findings of the 2005 PHA report indicated that, in general, the six cancer types evaluated occurred near or below the expected rates for Belmont census tracts (CTs) 3572, where Cambridge Plating is located, and the adjacent CT 3571 and for the town of Belmont as a whole during the 18-year time period, 1982–1999. Similar trends were observed in the surrounding communities of Cambridge, Arlington, and Watertown.

In addition to calculating cancer incidence rates, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer did not reveal any atypical spatial patterns that would suggest a common factor (environmental or nonenvironmental) in Belmont as a whole, CTs 3571 and 3572, or the surrounding communities of Cambridge, Arlington, and Watertown is related to the incidence of cancer. No unusual concentrations of individuals diagnosed with cancer were observed in the vicinity of the Cambridge Plating site or in any other area of Belmont, Cambridge, Arlington, or Watertown.

This current assessment contains additional analyses requested by concerned residents and received during the public comment period for the report released on October 20, 2005 (MDPH 2005). Public comments on the October 20, 2005 report were accepted through December 15, 2005. Two additional analyses were performed in response to public comments and results are provided below. Requests for additional analyses included further evaluation of the six cancer types analyzed in the public comment release report for the most recent time period, 2000-2003, and well as requests for analyses of bladder, brain and central nervous system (CNS) cancer, breast cancer and stomach cancer in Belmont.

II. Cancer Incidence, 2000–2003, for Six Cancer Types Analyzed in 2005 Public Health Assessment (PHA) for Belmont and Surrounding Communities

Cambridge Plating is located in CT 3572 close to the border of CT 3571 (see Figure 1 in the main report). This section presents cancer incidence rates for the town of Belmont, with particular focus on Belmont CTs 3571 and 3572, during the most recent time period for which data were available at the time of this writing, 2000–2003. A summary of the cancer experience in the communities of Cambridge, Arlington, and Watertown is also provided. Cancer incidence results for Belmont as a whole and the census tracts immediately surrounding Cambridge Plating are presented in Tables 1-3, while results for Cambridge, Arlington, and Watertown are presented in Tables 4, 5, and 6, respectively. Standardized Incidence Ratios (SIRs) were not calculated for some cancer types due to the small number of observed cases (less than five). However, the expected number of cases was calculated during each time period, and the observed and expected numbers of cases were compared to determine whether more cancer cases were occurring than expected.

A. Results

1. Town of Belmont

The cancer types evaluated in this section generally occurred approximately near or below expected rates in the town of Belmont as a whole during the 4-year time period,

2000–2003, although one statistically significant elevation was observed (i.e., NHL among females) (Table 1).

NHL occurred more often than expected in Belmont during 2000–2003 (32 diagnoses observed vs. 23.6 expected, SIR = 136, 95% CI = 93-191). Eleven males were diagnosed with NHL during 2000–2003 compared to about 12 males expected. The elevation was due to approximately nine additional diagnoses among females (21 diagnoses observed versus 11.7 expected, SIR = 180, 95% CI = 111-275) and was statistically significant. This statistically significant elevation among females diagnosed with NHL is discussed later, when risk factor information is summarized for these females diagnosed with NHL.

Overall, kidney cancer, liver cancer, and lung and bronchus cancer occurred less often than expected during 2000–2003. Kidney cancer occurred statistically significantly less often than expected in Belmont during 2000–2003 (7 diagnoses observed vs. 16.1 expected, SIR = 44, 95% CI = 17–90). Three males were diagnosed with kidney cancer compared to about ten expected. Among females, there were four diagnoses observed compared to about seven diagnoses expected. Liver cancer occurred slightly less often than expected in Belmont during 2000–2003 (4 diagnoses observed vs. 5.8 expected). Lung and bronchus cancer occurred statistically significantly less often than expected in Belmont (56 diagnoses observed vs. 87.5 expected, SIR = 64, 95% CI = 48–83) during the 4-year time period, 2000–2003.

Leukemia occurred at approximately the expected rate for males and females evaluated together during 2000–2003. Fifteen diagnoses of leukemia were observed in Belmont versus about 14 expected. Males in Belmont experienced fewer diagnoses of leukemia than expected during 2000–2003 (4 observed vs. 7.2 expected). However, among females, more diagnoses occurred during 2000–2003 than expected (11 diagnoses observed vs. 6.5 expected), but this elevation was not statistically significant.

Pancreatic cancer occurred more often than expected during 2000–2003 based on the statewide cancer experience. Nineteen diagnoses of pancreatic cancer were observed in Belmont during 2000–2003 versus about 15 expected. The increase was based on

approximately one additional diagnosis for males and approximately three additional diagnoses for females over the expected number and was not statistically significant.

2. Belmont CTs 3571 and 3572

Rates of kidney cancer, leukemia, liver, lung and bronchus cancer, and pancreatic cancer were approximately near or below the expected rates among males and females combined in Belmont CT 3571 during the 4-year time period, 2000–2003 (Table 2). Lung and bronchus cancer occurred statistically significantly less often than expected among males and females combined during this time (8 diagnoses observed vs. 19.3 expected, SIR = 42, 95% CI = 18-82).

Of the six cancer types evaluated in this section, one statistically significant elevation was observed (i.e., NHL) during the time period 2000–2003 in Belmont CT 3571. NHL occurred statistically significantly more often than expected (12 diagnoses observed versus 5.1 expected, SIR = 237, 95% CI = 123-415). This elevation was due to approximately one additional diagnosis among males in CT 3571, and approximately five additional diagnoses among females in CT 3571. The elevation observed among females in CT 3571 was statistically significant and is further discussed below in section B.1 (see page 261).

Rates of kidney cancer, leukemia, liver, lung and bronchus cancer, and NHL were approximately near or below the expected rates among males and females combined in Belmont CT 3572, where Cambridge Plating is located, during the 4-year time period 2000–2003 (Table 3).

Pancreatic cancer occurred statistically significantly more often among males and females in Belmont CT 3572 during 2000–2003. Six individuals were diagnosed with pancreatic cancer among males and females combined while approximately 1.8 would have been expected. About one excess case occurred among males (2 diagnoses observed vs. 0.9 expected), while about 3 excess cases of pancreatic cancer occurred among females (4 diagnoses observed vs. 0.9 expected). Cancer risk factor information related to pancreatic cancer is discussed below in section B.2 (see page 263).

3. City of Cambridge

Cancer incidence rates were evaluated for the city of Cambridge. The results of these analyses are summarized for each of the six cancer types in Table 4. The evaluation indicates that rates during the 2000–2003 time period were very similar to rates in the previously evaluated time period, 1982–1999. Citywide incidence rates in Cambridge during 2000–2003 were lower than expected for kidney cancer, leukemia, lung and bronchus cancer, and NHL. Rates were about as expected for pancreatic cancer and higher than expected for liver cancer. Overall, 21 individuals were diagnosed with liver cancer compared to 15.3 expected (SIR = 137, 95% CI = 85-209). The elevation in liver cancer was not statistically significant for males and females evaluated together. However, among females in Cambridge, nine individuals were diagnosed with liver cancer, compared to about four expected and the elevation was statistically significant.

4. Town of Arlington

In general, residents of Arlington experienced the six cancer types evaluated approximately at or below the rates expected during 2000–2003 (Table 5). Leukemia, liver cancer, lung and bronchus cancer, and pancreatic cancer all occurred less often than expected during 2000–2003. Overall, the incidence of lung and bronchus cancer in Arlington continued to be statistically significantly decreased with respect to the state rate during 2000–2003 (117 diagnoses observed vs. 150.5 expected, SIR = 78, 95% CI = 64–93). Kidney cancer occurred near expected rates during this time period (29 diagnoses observed vs. 27.5 expected, SIR = 105, 95% CI = 71–151), while NHL was diagnosed at slightly higher rates than expected during 2000–2003. Overall, 45 individuals were diagnosed with NHL compared to 40.8 expected (SIR = 110, 95% CI = 80-148). The elevation in NHL was not statistically significant.

5. Town of Watertown

With one exception, Watertown residents experienced the six cancer types approximately at or below the rates expected during 2000–2003 (Table 6). Specifically, the incidence of leukemia, liver cancer, lung and bronchus cancer, NHL, and pancreatic cancer were all

lower than expected during the time period 2000–2003. A slight elevation in kidney cancer continued to be observed during this time period, however the elevation was not statistically significant. The incidence of kidney cancer among males and females combined was slightly higher than expected based on the state rate (23 diagnoses observed vs. 20.1 expected, SIR = 114, 95% CI = 72-171).

B. Review of Cancer Risk Factor Information

As previously mentioned in the report, cancer is not just one disease, but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (the time between exposure and development of disease), characteristics, and trends in survival. Available information from the Massachusetts Cancer Registry (MCR) related to age and gender, as well as other factors related to the development of cancer such as smoking and occupation, was reviewed for individuals diagnosed with cancer in Belmont. Information for the two cancer types (i.e., NHL and pancreatic cancer) that were statistically significantly elevated was compared to known or established incidence trends to assess whether any unexpected patterns exist among these cases. It is important to note, however, that personal risk factors such as family history, pre-existing medical conditions, hormonal events, diet, and other factors also influence the development of these cancer types. This information is not collected by the MCR or any other readily accessible source. Therefore, it was not possible to evaluate the role these types of risk factors may have played in the incidence of cancer in Belmont in this investigation.

Age and gender are risk factors in many types of cancers, including kidney cancer, liver cancer, lung and bronchus cancer, leukemia, NHL, and pancreatic cancer. Therefore, a review of age-group specific SIRs was conducted for the cancer types with statistically significant elevations. Where numbers of cases in each age group were too small to calculate SIRs, the distribution of cases by age was reviewed.

Tobacco use is also a known or suggested causal risk factor in several types of cancer, including kidney cancer, lung and bronchus cancer, and pancreatic cancer. The smoking

history of individuals diagnosed with pancreatic cancer was reviewed to assess the role tobacco smoking may have played in the development of this cancer among residents of Belmont. However, results of smoking history analysis should be interpreted with caution because of the number of individuals for which smoking status was unknown.

In some studies, an association has been found between specific occupational exposures and an increase in the incidence of kidney cancer, leukemia, liver cancer, lung and bronchus cancer, NHL, and pancreatic cancer. Therefore, occupational information as reported to the MCR at the time of diagnosis was reviewed for individuals diagnosed with statistically significantly elevated cancer types to determine the role that occupational factors may have played in the development of these cancers in Belmont. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. In addition, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

1. Non-Hodgkin's Lymphoma (NHL)

Non-Hodgkin's lymphoma (NHL) can occur at all ages; however, the average age at diagnosis is the early 60s, and the incidence of this disease generally increases with age. The American Cancer Society estimates that approximately 63,190 Americans will be diagnosed with NHL in 2007, making it the fifth most common cancer in the United States among both men and women, excluding nonmelanoma skin cancers (ACS 2006a). Although the primary factors related to the development of NHL include viral infections and conditions that suppress the immune system, certain exposures related to occupations involving chemicals or agriculture have been associated with an increased risk of developing NHL. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm et al. 1990 and 1993; Tatham et al. 1997). 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).

a. Age and Gender

In the town of Belmont as a whole, a statistically significant elevation in the incidence of NHL was noted for females only, during the 4-year time period 2000–2003. During this time period males were diagnosed about as expected in Belmont. Among the 32 individuals diagnosed in the town of Belmont, the average age at diagnosis was 71 years, which is consistent with that seen in the general population. Among males, NHL occurred about as expected with 82% of the diagnoses occurring in males age 60 and older. Among females, NHL occurred statistically significantly more often than expected with 15 of the 21 diagnoses (71%) occurring in females age 60 and older.

Among individuals diagnosed with NHL in Belmont CT 3571, a statistically significant elevation was noted for the 4-year time period 2000–2003. The average age at diagnosis for individuals diagnosed with NHL in Belmont CT 3571 during this time period was 67 years, which is consistent with that seen in the general population. The average age for individuals diagnosed with NHL in the state of Massachusetts was 65 years. Among males, NHL occurred about as expected with 75% of the diagnoses occurring in males age 60 and older. Among females, NHL occurred statistically significantly more often than expected with four of the eight diagnoses occurring in females age 60 and older.

b. Occupation

On the basis of a review of job title information as reported to the MCR for individuals in Belmont diagnosed with NHL during 2000–2003, occupational exposures do not appear likely for the majority of those diagnosed. However, two individuals reported working in an occupation in which occupational exposures may have been possible. Information regarding specific job duties that could help to further define exposure potential for these individuals was not available. Occupation was reported as retired, unknown, or at home for 38% of individuals (n = 12).

Review of occupational information for individuals diagnosed with NHL in Belmont CT 3751 revealed that two individuals may have worked jobs in which occupational exposures potentially related to the development of NHL may have been possible. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not available. Occupation was reported as retired, unknown, or at home for 25% of individuals (n = 3). The occupations reported for the remaining seven individuals are not likely related to an increased risk of this cancer type.

c. Previous Cancer Diagnosis

According to the American Cancer Society, individuals treated with radiation therapy for some other cancers have a slightly increased risk of developing NHL later in life (ACS 2006a). Individuals treated with some chemotherapy drugs for other cancers may also have an increased risk of developing NHL later in life; however, a direct cause and effect relationship has not yet been definitely established (ACS 2006a). Among the 12 individuals diagnosed with NHL in Belmont CT 3571, five individuals (42%) had a previous cancer diagnosis. Unfortunately, the data reported to the MCR does not include information regarding specific treatments for the individuals' previous cancer diagnoses. However, it is possible that treatment related to previous cancer diagnoses may have played a role in NHL incidence in this census tract.

2. Pancreatic Cancer in Belmont CT 3572

The risk of developing pancreatic cancer increases with age, and the majority of cases occur between ages 60 and 80. Besides age, the most consistent risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2006b). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in nonsmokers (Anderson et al. 1996).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to

certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS 2006b; Ji et al. 2001; Porta et al. 1999). Exposure to certain dyes and to certain chemicals related to gasoline, in addition to exposure to asbestos and ionizing radiation, have also been associated with the development of pancreatic cancer in some studies. Other studies, however, have found no link between these agents and pancreatic cancer (Anderson et al. 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this finding (Ojajarvi et al. 2000).

a. Age and Gender

A review of individuals diagnosed with pancreatic cancer in Belmont CT 3572 from 2000–2003 revealed that more females (67%) than males (33%) were diagnosed. A review of individuals diagnosed with pancreatic caner in the state of Massachusetts revealed that only slightly more females (54%) than males (46%) were diagnosed. The average age at diagnosis for individuals with pancreatic cancer in Belmont CT 3572 during 2000–2003 was 69 years, consistent with the statewide average age at diagnosis of 71 years. Eighty-three percent of those diagnosed were age 65 or older at the time of diagnosis.

b. Smoking History

Of the six individuals diagnosed with pancreatic cancer in Belmont CT 3572 during 2000–2003, 33% (n = 2) reported being current or former smokers at the time of diagnosis. Another 33% (n = 2) were nonsmokers, and smoking history was unknown for the remaining 33% (n =2). In the state as a whole, 41% of those diagnosed with pancreatic cancer during 2000–2003 were current or former smokers at the time of diagnosis, 30% were nonsmokers, and smoking history was unknown for 29%.

c. Occupation

On the basis of a review of occupational information as reported to the MCR, two of the individuals diagnosed with pancreatic cancer in Belmont CT 3572 during 2000–2003 indicated working in a job likely to be associated with occupational exposures related to an increased risk of pancreatic cancer. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not available. The occupations reported for three individuals are not likely related to an increased risk of this cancer type. Occupation was reported as retired, unknown, or at home for the remaining individual.

C. Analysis of Geographic Distribution of Cancer Incidence

In addition to determining incidence rates for the six cancer types, a qualitative evaluation of the geographic pattern of cancer diagnoses was conducted for the town of Belmont and surrounding communities. Place of residence at the time of diagnosis was mapped for each individual diagnosed with the cancer types evaluated in this report to assess any possible geographic concentrations of cases in relation to each other or in the vicinity of Cambridge Plating. As previously mentioned, cancer is one word that describes many different diseases. Therefore, for the purposes of this evaluation, the geographic distribution of each cancer type was evaluated separately to determine whether an atypical pattern of any one type was occurring. The geographic distributions of some specific types of cancer were also evaluated together because they may have similar etiologies (e.g., leukemia and NHL in children).

Based on a review of address at the time of diagnosis for each individual diagnosed with a cancer type considered in this section, no apparent concentrations of cancer diagnoses (of any type) were observed in the vicinity of the Cambridge Plating site.

No other unusual spatial patterns or concentrations of cases at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents were apparent for any cancer type. Any patterns that were observed were consistent with what would be expected based on the population distribution and areas of higher population density. For example, in Belmont, the

majority of individuals with each type of cancer tended to be located in areas of the town where population and housing density are greater. Although elevations in the incidence of some cancer types were noted in Belmont or Belmont census tracts, in general, the geographic distribution of diagnoses for these cancer types seemed to coincide closely with the pattern of population density. Specifically, in Belmont CTs 3571 and 3572 where elevations in NHL and pancreatic cancer were observed, respectively, there were no apparent spatial patterns at the neighborhood level that could not be attributed to factors such as areas of higher population density (e.g., the presence of multi-unit housing complexes or nursing homes). Further, no apparent concentrations of cancer diagnoses (of any type) were observed in any of the three surrounding communities that would suggest a potential relationship to Cambridge Plating. Thus, it does not appear that exposures to environmental contamination associated with Cambridge Plating are likely to have played a role in the development of cancer among residents of Belmont or surrounding communities.

D. Discussion

In general, the six cancer types (e.g., kidney cancer, leukemia, liver cancer, lung and bronchus cancer, Non-Hodgkin's Lymphoma (NHL), and pancreatic cancer) evaluated during 2000–2003 occurred approximately near or below expected rates in the town of Belmont and the surrounding communities of Cambridge, Arlington, and Watertown. However, some elevations were observed. In Belmont, the incidence of NHL among females was statistically significantly elevated in census tract (CT) 3571 during 2000–2003. Pancreatic cancer was also statistically significantly elevated among males and females evaluated together in Belmont CT 3572 during 2000–2003. This elevation in pancreatic cancer was due to three additional diagnoses among females during this period of study. Citywide incidence ratios for liver cancer among females in Cambridge were statistically significantly elevated during 2000–2003.

Available risk factor information for individuals diagnosed with NHL or pancreatic cancer in Belmont was compared to known or established trends to assess whether any unexpected patterns exist in the town. In general, risk factor trends observed in Belmont were similar to those seen in the general population. Further evaluation of the pattern of

cancer diagnoses revealed that individuals diagnosed with NHL in Belmont CT 3571 during 2000–2003 were diagnosed with histology types of the disease that are consistent with the distribution observed statewide during the same time period. Of the 12 individuals diagnosed with NHL in Belmont CT 3571, 50% were diagnosed with the most common type of NHL diagnosed in the state. The remaining individuals were diagnosed with ## different histology types of NHL. Among the eight females diagnosed with NHL, four were diagnosed with the most common type of NHL diagnosed in the state. Analysis of the geographic distribution of place of residence for individuals diagnosed with cancer, with particular focus on NHL diagnoses in Belmont CT 3571 and pancreatic cancer diagnoses in Belmont CT 3572, did not reveal any atypical spatial patterns that would suggest a common factor (environmental or nonenvironmental) related to the incidence of cancer in Belmont or in surrounding communities.

To evaluate possible trends in cancer incidence over time, data from 1982–1999 was evaluated alongside data from the more recent time period, 2000–2003. Appendix A of this report entitled "*Assessment of Cancer Incidence in Belmont, Massachusetts, and Surrounding Communities: 1982–1999*" provides a detailed review of the pattern of cancer incidence in Belmont and adjacent communities for the years 1982–1999 and for three smaller time periods, 1982–1987, 1988–1993, 1994–1999. By including the results from the most recent time period, 2000–2003, as discussed above, patterns or trends in cancer incidence over four time periods can be evaluated.

In the town of Belmont, an evaluation of cancer incidence over time indicates that kidney cancer rates decreased from 1982 through 2003. Lung and bronchus cancer and liver cancer occurred approximately at or below expected rates across previously evaluated time periods during 1982–1999 and during 2000–2003. Pancreatic cancer and NHL occurred approximately at or below expected rates during three of the four time periods. Rates of pancreatic cancer and NHL were elevated during the 2000–2003 time period. Neither elevation was statistically significant. Incidence of leukemia in the town of Belmont occurred approximately at or below expected rates in two time periods: 1982–1987 and 2000–2003; and occurred slightly above expected rates in two time periods:

1988–1993 and 1994–1999. The slight elevation was not statistically significant and is discussed further in Appendix A.

In Belmont CT 3571, kidney cancer, leukemia, liver cancer, and lung and bronchus cancer occurred approximately at or below expected rates across time periods evaluated in Appendix A of this report, 1982-1999, and during 2000-2003. NHL occurred approximately at or below expected rates during three of the four time periods and was elevated during the 2000-2003 time period. This elevation was statistically significant, however, an evaluation of risk factors (e.g., age, gender, occupation, previous cancer diagnoses), histology types, and spatial and temporal patterns did not reveal any atypical patterns that would suggest a common factor (environmental or nonenvironmental) related to the incidence of NHL in Belmont CT 3571. Pancreatic cancer occurred below expected rates in two time periods, 1988-1993 and 2000-2003, and occurred slightly above expected rates in two time periods, 1982-1987and 1994-1999. No clear trend was apparent in pancreatic cancer over time and no single time period showed a statistically significant elevation.

In Belmont CT 3572, the census tract where Cambridge Plating is located, kidney cancer, leukemia, liver cancer, lung and bronchus cancer, and NHL occurred approximately at or below expected rates across previously evaluated time periods during 1982–1999 and during 2000–2003. Pancreatic cancer occurred approximately at or below expected rates during 1982–1999. However, pancreatic cancer occurred statistically significantly above expected rates during 2000–2003 and is discussed in Sections IIA (see page 2) and IIB (see page 6) of this appendix.

In the city of Cambridge, an evaluation of cancer incidence over time indicates that rates during the 2000–2003 time period were very similar to rates in the previously evaluated time period, 1982–1999. Citywide incidence rates in Cambridge during 1982–1999 and 2000–2003 were approximately at or below expected rates for kidney cancer, leukemia, lung and bronchus cancer, NHL and pancreatic cancer. Liver cancer occurred slightly more often than expected in previously evaluated time periods during 1982–1999 and during 2000–2003.

In the town of Arlington, kidney cancer, leukemia, and lung and bronchus cancer occurred approximately at or below expected rates across previously evaluated time periods during 1982–1999 and during 2000–2003. Liver cancer and pancreatic cancer showed elevations during the 1982–1987 time period and are discussed in Appendix A of this PHA. Rates of liver and pancreatic cancer in the most recent time period evaluated, 2000–2003, are approximately at or below expected. Rates of NHL were approximately at or below expected during the two earlier time periods, 1982–1987, 1988–1993 and were slightly elevated across the two latter time periods, 1994–1999 and 2000–2003. The slight elevation in NHL in Arlington was not statistically significant.

In Watertown, an evaluation of cancer incidence over time indicates that lung and bronchus cancer and pancreatic cancer occurred approximately at or below expected rates across previously evaluated time periods during 1982-1999 and during 2000-2003. Kidney cancer in Watertown occurred below expected rates during 1994–1999, and slightly above expected rates in other time periods evaluated. The slight elevations in kidney cancer were not statistically significant for males and females combined. However, a statistically significant elevation occurred in males during 1988–1993 and is discussed in Appendix A. Rates of leukemia were below expected during two time periods: 1982–1987, 2000–2003; and were slightly elevated across two time periods: 1988–1993 and 1994–1999. Liver cancer occurred approximately at or below expected rates during three of the four time periods. Liver cancer occurred more often than expected during 1994–1999 and is discussed in Appendix A. The slight elevation in leukemia in Watertown was not statistically significant. An evaluation of NHL rates over time indicated that incidence of NHL has been decreasing over time.

III. Cancer Incidence for Four Cancer Types in Belmont Census Tracts 3571 and 3572 in Response to Community Concern

The MDPH selected the six cancer types (e.g., kidney cancer, leukemia, liver cancer, lung and bronchus cancer, NHL, and pancreatic cancer) for evaluation in the 2005 Public Health Assessment (PHA) report based on available environmental data for Cambridge Plating and information in the scientific literature on known or suspected associations with contaminants of concern (e.g., trichloroethylene, chromium). However, due to community concerns about bladder cancer, brain and central nervous system (CNS) cancer, breast cancer, and stomach cancer, the MDPH reviewed cancer incidence data from 1982–2003 for each of these types. To evaluate possible trends over time, cancer incidence data were also analyzed by four smaller time periods, 1982–1987, 1988–1993, 1994–1999, and 2000–2003.

Because the six cancer types evaluated in the 2005 PHA in relation to Cambridge Plating generally occurred near or below the expected rates for the census tracts surrounding the facility (Belmont CTs 3571 and 3572) and the town of Belmont as a whole during the 18-year time period 1982–1999, this evaluation will focus on diagnoses of the four types within census tracts immediately surrounding Cambridge Plating.

The following section presents the results of the cancer incidence analyses for the Belmont CTs 3571 and 3572 during the 22-year time period 1982–2003. Although SIRs and 95% confidence intervals were not calculated for some cancer types in smaller time periods due to small numbers of observed diagnoses (i.e., fewer than five), the expected number of diagnoses was calculated to determine whether excess numbers of diagnoses were occurring. These data are summarized in Tables 7a through 7e and 8a through 8e.

A. Results

1. Belmont Census Tract 3571

Of the cancer types evaluated in this section, three types (bladder cancer, brain/CNS cancer, and stomach cancer) occurred approximately near or below expected rates, while one (breast cancer) occurred slightly more than expected rates in Belmont CT 3571 during the 22-year time period 1982–2003 as well as smaller time periods (i.e., 1982–1987, 1988–1993, 1994–1999, and 2000–2003). No statistically significant elevations were noted during the 22-year time period or during any of the smaller time periods in Belmont CT 3571.

Bladder cancer occurred approximately near expected rates during 1982–2003 based on the statewide cancer experience. Twenty-nine diagnoses of bladder cancer were observed in Belmont during 1982–2003 versus about 27 expected. When examined by smaller time periods, no clear trends emerged among males or females over time. During 1982–1987 and 1994–1999, males were diagnosed with bladder cancer slightly less than expected, while females were diagnosed slightly more often than expected. During 1988–1993, males and females were diagnosed slightly more often than expected based on statewide incidence of bladder cancer. During the most recent time period, 2000–2003, bladder cancer rates were near or below the expected rates for males and females in Belmont CT 3571. None of the slight elevations noted during the earlier time periods were statistically significant.

Brain and CNS cancer occurred generally as expected during the overall time period 1982–2003 and during each of the smaller time periods evaluated. While brain and CNS cancer occurred at about the rate expected based on statewide rates during the three time periods, 1982–1987, 1994–1999, and 2000–2003, one additional diagnosis was noted during the time period 1988–1993.

Females in Belmont CT 3571 were diagnosed with breast cancer more often than expected during the overall time period 1982–2003, and during the first three smaller time periods evaluated. The increases observed during the smaller time periods were

based on approximately four to five additional diagnoses over the expected number among females and were not statistically significant. During the most recent time period, 2000–2003, females were diagnosed with breast cancer less than the expected rates in Belmont CT 3571.

Stomach cancer occurred less often than expected during 1982–2003 and during each of the smaller time periods. The incidence of stomach cancer appears to have remained consistent over time among both male and female residents of Belmont CT 3571, with a slight decrease in incidence in the most recent time period evaluated, 2000–2003. During the most recent time period evaluated, no individuals were diagnosed with stomach cancer compared to about two diagnoses expected. Both males and females experienced lower than expected incidence of stomach cancer during all time periods.

Refer to Tables 7a– 7e for bladder, brain and CNS, breast, and stomach cancer incidence rates in Belmont CT 3571.

2. Belmont Census Tract 3572

Rates of bladder cancer and stomach cancer were near or below the expected rates among males and females combined in Belmont CT 3572 during the 22-year time period, 1982–2003. Slightly more brain and CNS cancer diagnoses were observed during the overall time period; however, the elevation was not statistically significant. Statistically significant elevations in breast cancer were observed among females during the 22-year time period, as well as during two of the smaller time periods, 1988–1993 and 2000–2003.

When examined by smaller time periods, incidence of bladder cancer was generally near or below the expected rates for males and females combined and no clear patterns emerged when males and females were evaluated separately. During 1988–1993, about one additional diagnosis was observed for males and females combined. This was due to fewer diagnoses than expected among males (2 diagnoses observed vs. 2.9 expected) and more diagnoses than expected among females (3 diagnoses observed vs. 1.1 expected).

Brain and CNS cancer occurred slightly more often than expected during the 22-year time period from 1982–2003 (9 diagnoses observed vs. 6.3 expected). The elevation was based on approximately one additional diagnosis for females during 1982–1987 and one additional diagnosis for males during 1994–1999.

Evaluation of breast cancer showed statistically significant elevations from 1988–1993 and 2000–2003 and a higher than expected number of diagnoses during 1994–1999. Breast cancer during 1982–1987 occurred approximately as expected, with 15 diagnoses observed compared to 15.2 diagnoses expected based on statewide cancer incidence.

Stomach cancer was diagnosed approximately near or below the expected rate for three of the four smaller time periods. During the most recent time period, 2000–2003, approximately one additional diagnosis was noted for males and approximately one additional diagnosis was noted for females. This elevation was not statistically significant.

Refer to Tables 8a– 8e for bladder, brain and CNS, breast, and stomach cancer incidence rates in Belmont CT 3572.

B. Review of Risk Factor Information

1. Breast Cancer in Belmont Census Tract 3572

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts and accounts for almost 30% of all newly diagnosed cancers among females (Henderson et al. 1996). Breast cancer has the highest incidence rate of all cancers among women ages 35 and above, with higher incidence rates in the older age groups (Devesa et al. 1995). According to the American Cancer Society, about 17% of new breast cancer diagnoses are among women in their 40s, while approximately 78% occur in women over age 50 (ACS 2006c). Breast cancer incidence and age have been shown to be related because incidence increases from age 35 to 45, increases at a slower rate from age 45 to 50, and at a steeper rate in post-menopausal women after age 50 (Kessler 1992).

The risk of developing breast cancer can be influenced by a number of factors. Epidemiological studies have determined some well-established risk factors for this cancer type. The most well-established risk factors for breast cancer are related to genetic and specific reproductive events in a woman's life, such as age at first pregnancy, number of births, and age at menopause (Kessler 1992). Other factors such as a woman's age and demographic characteristics (e.g., socioeconomic status) are known to increase breast cancer risk. More recent research on breast cancer has included evaluation of the possible contributions of occupation or environmental factors in breast cancer development.

a. Age and Gender

A review of individuals diagnosed with breast cancer in Belmont CT 3572 from 1982–2003 revealed that approximately 17% were in their 40s at time of diagnosis. An additional 75% were age 50 or older at time of diagnoses. This is consistent with established prevalence patterns reported by the American Cancer Society (ACS 2006c). The average age at diagnosis for individuals with breast cancer in Belmont CT 3572 during 1982–2003 was 61 years, which is consistent with established trends of this disease. The majority of the individuals diagnosed with breast cancer in Belmont CT 3572 were women. Two men were diagnosed with breast cancer in Belmont CT 3572 during this time. This gender pattern is also consistent with general prevalence patterns of breast cancer.

C. Analysis of Geographic Distribution of Cancer Incidence

In addition to determining incidence rates for each cancer type, a qualitative evaluation of the geographic pattern of cancer diagnoses was conducted, particularly as it relates to areas of environmental concern. The place of residence for individuals diagnosed with bladder cancer, brain and CNS cancer, breast cancer, and stomach cancer in Belmont CTs 3571 and 3572 was evaluated to identify any geographic concentrations of cases that might be present within the community.

Based on a review of address at the time of diagnosis for each individual diagnosed with the cancer types evaluated in this section, no atypical spatial patterns that would suggest a common factor (environmental or nonenvironmental) related to the incidence of cancer in the surrounding communities were noted. In general, any patterns observed were consistent with what would be expected based on the population distribution and areas of higher population density. Further, no apparent concentrations of cancer diagnoses (of any type) were observed in Belmont CTs 3571 or 3572 that would suggest a potential relationship to Cambridge Plating.

D. Discussion

The MDPH selected six cancer types (e.g., kidney cancer, leukemia, liver cancer, lung and bronchus cancer, NHL, and pancreatic cancer) for evaluation in the 2005 PHA report based on available environmental data for Cambridge Plating and information in the scientific literature on known or suspected associations with the contaminants of concern. However, to address community concerns about breast, brain, bladder and stomach cancers, cancer incidence rates for these four types were calculated and presented as additional analyses in this report.

The MDPH reviewed cancer incidence data for the Belmont CTs surrounding the facility for the four cancer types. The overall time period, 1982–2003, as well as four smaller time periods were analyzed to evaluate possible trends over time. Three of the four cancer types evaluated during 1982–2003 occurred approximately near expected rates in Belmont CTs 3571 and 3572. Breast cancer occurred statistically significantly more often than expected in Belmont CT 3572 during the overall time period, 1982–2003, and during two smaller time periods, 1988–1993 and 2000–2003.

To better understand the pattern of breast cancer in Belmont CT 3572, the stage of cancer at the time of diagnosis was also reviewed. The staging of breast cancer categorizes the extent of the disease and its spread at the time of diagnosis. Communities in which many women receive routine breast cancer screening are expected to have a greater number of women diagnosed at the early stages of the disease. Conversely, communities with low screening rates would be expected to have more diagnoses occurring at the later stages of

disease. Invasive breast cancer is typically classified as one of four stages of disease: localized, regional, distant, and unknown. Localized breast cancer represents a diagnosis in which the tumor is invasive but the cancer is confined to the breast. Regional refers to a tumor that has spread beyond the organ of origin (breast), including spread to adjacent tissues and organs, lymph nodes, or both. Distant stage breast cancer is a cancer that has metastasized or spread to organs other than those adjacent to the organ of origin, to distant lymph nodes, or both (MCR 1996). Some diagnoses are reported to the Massachusetts Cancer Registry (MCR) with an unknown stage meaning that, at the time of reporting by a hospital or other facility (e.g., physician's office), the tumor had not been staged.

Since the inception of the MCR in 1982, breast cancer staging has changed over time in Massachusetts. Prior to 1995, the MCR did not require the use of a standardized staging system, so hospital registrars used different staging systems when reporting diagnoses to the MCR. In 1995, the MCR adopted one staging system and required all hospital registrars to report staging information using this system. Due to the variability of staging data for diagnoses prior to 1995, this analysis examined the more reliable staging data (for both Belmont residents and the state as a whole) that was available for the 1995-2003 time period[1].

From 1995-2003, approximately 61% of breast cancer diagnoses among individuals in Belmont CT 3572 were diagnosed at the local stage of disease, approximately 34% were diagnosed at the regional stage, and just fewer than 3% were diagnosed at either the distant stage or with an unknown stage. The combined earlier stages are similar to the distribution observed statewide during this time period (65.7% local, 26.4% regional, 4.4% distant, and 3.5% unknown).

[1] Breast cancer staging information is based on the staging systems in use at the time of diagnosis and/or reporting. For pre-1995 staging data, in most cases, the American Joint Committee on Cancer's TNM (Tumor, Node, Metastasis) staging system was used. For post-1995 staging data (through 2003), the SEER (Surveillance, Epidemiology and End Results) Summary Stage system was used. Regardless of the time period, for the purposes of this report, breast cancer stages were categorized as local, regional, distant, or unknown. These categories are more general than currently used by physicians and tumor registrars, but are appropriate for descriptive epidemiological investigations.

Despite the vast number of studies on the causation of breast cancer, known risk factors are estimated to account for slightly more than half of all diagnoses in the general population (Madigan et al. 1995). Researchers are continuing to examine other potential genetic, hormonal, and environmental (e.g., pesticides or PCBs) risk factors for breast cancer. Nonetheless, it is known that a female's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon well-established risk factors for this cancer type. Females with a family history of breast cancer, those who have never had children, or have had their first child after the age of 30, are at an increased risk for developing this disease (ACS 2006c). Women who take menopausal hormone therapy (estrogen plus progestin) for 5 or more years after menopause also appear to have an increased risk of developing breast cancer (National Cancer Institute 2005). Information related to family history of breast cancer, reproductive factors, and use of hormone replacement therapy after menopause is not included in the MCR database.

Although the number of reported diagnoses of breast cancer exceeded the number of expected diagnoses during the 22-year time period evaluated, no unusual patterns were noted. From the available data, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of breast cancer in Belmont CTs 3571 or 3572. The MDPH will continue to monitor the incidence of all cancer types in the town through city/town cancer incidence reports published by the MCR.

IV. Conclusions

- With two exceptions, the cancer types evaluated in relation to Cambridge Plating occurred near or below the expected rates for Belmont CTs 3571 and 3572 and the town of Belmont as a whole during the 4-year time period, 2000–2003. Similar trends were observed in the surrounding communities of Cambridge, Arlington, and Watertown. The incidence of NHL among females in Belmont and among females in CT 3571 was statistically significantly elevated during this time period, as was the incidence of pancreatic cancer among males and females in CT 3572. A review of available risk factor information and spatial and temporal patterns for individuals

diagnosed with NHL or pancreatic cancer in Belmont showed that patterns observed in Belmont are similar to those seen in the general population. No unusual concentrations of individuals diagnosed with cancer (including those cancer types with a potential association with exposure to TCE and chromium) were observed in the vicinity of the Cambridge Plating site or in any other area of Belmont, Cambridge, Arlington, or Watertown.

- With one exception, the cancer types (bladder, brain and CNS, breast, stomach) evaluated at the request of the community occurred near or below expected rates for Belmont CTs 3571 and 3572. Breast cancer among females in Belmont CT 3572 was statistically significantly elevated, however, review of the geographic distribution of individuals diagnosed with breast cancer in CTs surrounding Cambridge Plating revealed no apparent spatial patterns at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents. Review of available risk factor information for individuals diagnosed with breast cancer (e.g., age, gender) suggests that the trends observed in Belmont CTs are similar to those seen in the general population.

V. References

American Cancer Society (ACS). 2006a. Lymphoma, Non-Hodgkin's type. Available at: http://www.cancer.org

American Cancer Society (ACS). 2006b. Pancreatic Cancer. Available at: http://www.cancer.org

American Cancer Society (ACS). 2006c. Breast Cancer. Available at: http://www.cancer.org

Anderson D, Potter J, Mack T. 1996. Pancreatic cancer. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press.

Devesa SS, Blot WJ, Stone BJ, Miller BA, Tarone RE, Fraumeni JF, Jr. 1995. Recent cancer trends in the United States. Journal Natl Canc Inst. 87:175-182.

Environmental Systems Research Institute (ESRI). 2006. ArcGIS, Arcview license, ver. 9.2, Redlands, California.

Henderson BE, Pike MC, Bernstein L, Ross RK. Breast cancer. In: Schottenfeld D, Fraumeni, JF, Jr, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 1022-1035.

Ji BT, Silverman DT, Stewart PA, et al. 2001. Occupational exposure to pesticides and pancreatic cancer. Am J Ind Med 39(1):92-9.

Kessler LG. 1992. The relationship between age and incidence of breast cancer: population and screening program data. Cancer 69:1896-1903.

Madigan MP, Ziegler RG, Benichou J, et al. 1995. Proportion of breast cancer cases in the United States explained by well-established risk factors. J Natl Cancer Inst 87(22): 1681-1685.

Massachusetts Cancer Registry (MCR). 1996. Massachusetts Cancer Registry Abstracting and Coding Manual for Hospitals. 2nd ed. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Boston, MA.

Massachusetts Department of Public Health (MDPH). 2005. Public Comment Release: Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999. Community Assessment Program. October 2005.

National Cancer Institute (NCI). 2005. Facts about Menopausal Hormone Therapy. Available at: http://www.nhlbi.nih.gov/health/women/pht_facts.htm

Ojajarvi IA, Partanen TJ, Ahlbom A, et al. 2000. Occupational exposures and pancreatic cancer: a meta-analysis. Occup Environ Med 57(5):316-24.

Porta M, Malats N, Jariod M, et al. 1999. Serum concentrations of organochlorine compounds and K-ras mutations in exocrine pancreatic cancer. Lancet 354:2125-9.

Tatham L, Tolbert P, Kjeldsberg C. 1997. Occupational risk factors for subgroups of non-Hodgkin's lymphoma. Epidemiology. 8(5):1551-8.

Wong O, Raabe GK. 2000. Non-Hodgkin's lymphoma and exposure to benzene in a multinational cohort of more than 308,000 petroleum workers, 1937–1996. J Occup Environ Med 42(5):554-68.

Zahm SH, Weisenburger DD, Babbit PA, et al. 1990. A case-control study of non-Hodgkin's lymphoma and the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) in eastern Nebraska. Epidemiology 1(5):349-56.

Zahm SH, Weisenburger DD, Saal RC, et al. 1993. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women. Arch Environ Health 48(5):353-8.

TABLES

TABLE 1
Cancer Incidence
Belmont, Massachusetts
2000-2003

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	7	16.1	44 *	17	90	3	9.6	NC	NC	NC	4	6.5	NC	NC	NC
Leukemia	15	13.7	109	61	180	4	7.2	NC	NC	NC	11	6.5	169	84	302
Liver	4	5.8	NC	NC	NC	2	4.3	NC	NC	NC	2	1.5	NC	NC	NC
Lung & Bronchus	56	87.5	64 *	48	83	22	44.7	49 *	31	75	34	42.8	79	55	111
NHL	32	23.6	136	93	191	11	11.9	92	46	165	21	11.7	180 *	111	275
Pancreatic	19	14.9	127	77	199	8	6.8	117	51	231	11	8.1	136	68	243

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
NHL = Non-Hodgkin's Lymphoma
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 2
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
2000-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	1	3.4	NC	NC -- NC	0	2.0	NC	NC -- NC	1	1.4	NC	NC -- NC
Leukemia	4	2.9	NC	NC -- NC	2	1.5	NC	NC -- NC	2	1.4	NC	NC -- NC
Liver	1	1.2	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	8	19.3	42	* 18 -- 82	3	9.7	NC	NC -- NC	5	9.6	52	17 -- 121
NHL	12	5.1	237	* 123 -- 415	4	2.5	NC	NC -- NC	8	2.6	312	* 134 -- 614
Pancreatic	2	3.4	NC	NC -- NC	1	1.5	NC	NC -- NC	1	1.9	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
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 3
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
2000-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	1	2.1	NC	NC -- NC	1	1.3	NC	NC -- NC	0	0.8	NC	NC -- NC
Leukemia	1	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
Liver	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung & Bronchus	7	10.9	64	26 -- 132	4	5.8	NC	NC -- NC	3	5.1	NC	NC -- NC
NHL	3	2.9	NC	NC -- NC	2	1.5	NC	NC -- NC	1	1.3	NC	NC -- NC
Pancreatic	6	1.8	337	* 123 -- 733	2	0.9	NC	NC -- NC	4	0.9	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
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 4
Cancer Incidence
Cambridge, Massachusetts
2000-2003

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	40	42.8	94	67 --	127	23	25.2	91	58 --	137	17	17.6	97	56 --	155
Leukemia	36	38.1	95	66 --	131	21	20.3	103	64 --	158	15	17.8	84	47 --	139
Liver	21	15.3	137	85 --	209	12	11.4	105	54 --	184	9	4.0	227 *	104 --	432
Lung & Bronchus	174	221.1	79 *	67 --	91	84	110.1	76 *	61 --	94	90	111.0	81	65 --	100
NHL	50	65.5	76	57 --	101	21	33.7	62 *	39 --	95	29	31.8	91	61 --	131
Pancreatic	39	36.8	106	75 --	145	16	16.7	96	55 --	155	23	20.0	115	73 --	172

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
NHL = Non-Hodgkin's Lymphoma
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 5
Cancer Incidence
Arlington, Massachusetts
2000-2003

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	29	27.5	105	71	-- 151	19	15.8	120	72	-- 187	10	11.7	86	41	-- 157
Leukemia	22	23.5	94	59	-- 142	13	11.9	109	58	-- 187	9	11.6	77	35	-- 147
Liver	9	9.8	92	42	-- 175	5	7.1	70	23	-- 164	4	2.7	NC	NC	-- NC
Lung & Bronchus	117	150.5	78 *	64	-- 93	49	73.2	67 *	50	-- 88	68	77.3	88	68	-- 111
NHL	45	40.8	110	80	-- 148	22	19.8	111	70	-- 168	23	21.0	110	70	-- 165
Pancreatic	20	25.6	78	48	-- 121	5	11.1	45	15	-- 106	15	14.6	103	58	-- 170

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
NHL = Non-Hodgkin's Lymphoma
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 6
Cancer Incidence
Watertown, Massachusetts
2000-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	23	20.1	114	72 -- 171	16	11.4	140	80 -- 227	7	8.7	80	32 -- 165
Leukemia	15	17.7	85	47 -- 139	3	8.9	NC	NC -- NC	12	8.8	136	70 -- 237
Liver	4	7.1	NC	NC -- NC	3	5.1	NC	NC -- NC	1	2.0	NC	NC -- NC
Lung & Bronchus	106	112.0	95	77 -- 114	57	54.0	106	80 -- 137	49	58.0	84	62 -- 112
NHL	29	30.8	94	63 -- 135	13	14.8	88	47 -- 150	16	16.0	100	57 -- 163
Pancreatic	15	19.3	78	43 -- 128	9	8.1	110	50 -- 210	6	11.2	54	20 -- 117

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
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 7a
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
1982-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	29	27.2	107	71 -- 153	15	18.9	79	44 -- 131	14	8.3	168	92 -- 282
Brain and CNS	12	10.0	120	62 -- 209	4	4.7	NC	NC -- NC	8	5.3	150	65 -- 296
Breast	121	112.7	107	89 -- 128	0	0.8	NC	NC -- NC	121	111.9	108	90 -- 129
Stomach	9	14.8	61	28 -- 115	6	8.3	72	26 -- 157	3	6.5	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 7b
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	10	8.2	121	58 -- 223	4	5.8	NC	NC -- NC	6	2.5	243	89 -- 529
Brain and CNS	3	2.8	NC	NC -- NC	1	1.3	NC	NC -- NC	2	1.6	NC	NC -- NC
Breast	34	28.5	119	82 -- 166	0	0.2	NC	NC -- NC	34	28.4	120	83 -- 167
Stomach	3	4.6	NC	NC -- NC	2	2.6	NC	NC -- NC	1	2.0	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 7c
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	7.7	117	53 -- 222	6	5.3	112	41 -- 244	3	2.3	NC	NC -- NC
Brain and CNS	4	3.0	NC	NC -- NC	2	1.3	NC	NC -- NC	2	1.7	NC	NC -- NC
Breast	37	31.8	116	82 -- 161	0	0.2	NC	NC -- NC	37	31.5	117	83 -- 162
Stomach	3	4.1	NC	NC -- NC	2	2.3	NC	NC -- NC	1	1.9	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 7d
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
1994-1999

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	7	7.2	97	39 -- 200	3	5.0	NC	NC -- NC	4	2.2	NC	NC -- NC
Brain and CNS	3	2.5	NC	NC -- NC	1	1.3	NC	NC -- NC	2	1.2	NC	NC -- NC
Breast	35	30.7	114	79 -- 159	0	0.2	NC	NC -- NC	35	30.4	115	80 -- 160
Stomach	3	3.6	NC	NC -- NC	2	2.1	NC	NC -- NC	1	1.5	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 7e
Cancer Incidence
Census Tract 3571, Belmont, Massachusetts
2000-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	3.8	NC	NC -- NC	2	2.7	NC	NC -- NC	1	1.1	NC	NC -- NC
Brain and CNS	2	1.6	NC	NC -- NC	0	0.8	NC	NC -- NC	2	0.8	NC	NC -- NC
Breast	15	18.8	80	45 -- 132	0	0.2	NC	NC -- NC	15	18.6	81	45 -- 133
Stomach	0	2.3	NC	NC -- NC	0	1.3	NC	NC -- NC	0	1.0	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 8a
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
1982-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	13	14.0	93	50 -- 159	8	10.1	79	34 -- 156	5	3.9	129	42 -- 302
Brain and CNS	9	6.3	143	65 -- 271	4	3.2	NC	NC -- NC	5	3.1	163	53 -- 381
Breast	87	60.9	143	* 114 -- 176	2	0.4	NC	NC -- NC	85	60.4	141	* 112 -- 174
Stomach	8	7.4	108	46 -- 212	6	4.5	133	48 -- 289	2	2.9	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 8b
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	4.4	NC	NC -- NC	3	3.2	NC	NC -- NC	0	1.2	NC	NC -- NC
Brain and CNS	3	1.7	NC	NC -- NC	1	0.8	NC	NC -- NC	2	0.9	NC	NC -- NC
Breast	15	15.2	99	55 -- 163	0	0.1	NC	NC -- NC	15	15.1	99	56 -- 164
Stomach	2	2.4	NC	NC -- NC	1	1.4	NC	NC -- NC	1	0.9	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 8c
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
1988-1993

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	5	3.9	127	41	295	2	2.9	NC	NC	NC	3	1.1	NC	NC	NC
Brain and CNS	1	1.9	NC	NC	NC	0	0.9	NC	NC	NC	1	1.0	NC	NC	NC
Breast	29	16.6	174 *	117	250	1	0.1	NC	NC	NC	28	16.5	169 *	113	245
Stomach	2	2.0	NC	NC	NC	2	1.2	NC	NC	NC	0	0.8	NC	NC	NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 8d
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
1994-1999

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	3.7	NC	NC -- NC	1	2.7	NC	NC -- NC	2	1.0	NC	NC -- NC
Brain and CNS	3	1.6	NC	NC -- NC	2	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
Breast	22	17.2	128	80 -- 193	0	0.1	NC	NC -- NC	22	17.1	129	81 -- 195
Stomach	1	1.8	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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 8e
Cancer Incidence
Census Tract 3572, Belmont, Massachusetts
2000-2003

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	2	2.0	NC	NC	NC	2	1.5	NC	NC	NC	0	0.5	NC	NC	NC
Brain and CNS	2	1.1	NC	NC	NC	1	0.6	NC	NC	NC	1	0.5	NC	NC	NC
Breast	21	11.3	186 *	115	285	1	0.1	NC	NC	NC	20	11.2	179 *	109	276
Stomach	3	1.2	NC	NC	NC	2	0.8	NC	NC	NC	1	0.4	NC	NC	NC

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
CNS = Central Nervous System
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.

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).

References

American Cancer Society (ACS). 2006a. Cancer Facts & Figures 2006. Atlanta: American Cancer Society, Inc.

American Cancer Society (ACS). 2006b. Detailed Guide: Bladder Cancer. Available at: http://www.cancer.org.

Castelao JE, Yuan JM, Skipper PL, et al. 2001. Gender- and smoking-related bladder cancer risk. J Natl Cancer Inst 93(7):538-45.

Johansson SL, Cohen SM. 1997. Epidemiology and etiology of bladder cancer. Semin Surg Oncol 13:291-298.

Silverman D, Morrison A, Devesa S. 1996. Bladder Cancer. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

Villanueva M, Fernandez F, Malats N, Grimalt JO, and Kogevinas M. 2003. Meta-analysis of studies on individual consumption of chlorinated drinking water and bladder cancer. J. Epidemiol. Community Health 57(3): 166 – 173.

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).

References

American Cancer Society. 2006. Cancer Facts & Figures 2006. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2006a. Detailed Guide: Brain/CNS Tumors in Adults. Available at: http://www.cancer.org. Cited March 31, 2006.

American Cancer Society. 2006b Detailed Guide:.Brain/Central Nervous System (CNS) Tumors in Children. Available at: http://www.cancer.org. Cited March 31, 2006.

Blot WJ, Henderson BE, Boice JD, Jr. 1999. Childhood cancer in relation to cured meat intake: review of the epidemiological evidence. Nutr Cancer 34(1):111-8.

Bunin G. 2000. What causes childhood brain tumors? Limited knowledge, many clues. Pediatr Neurosurg 32(6):321-6.

Cocco P, Dosemeci M, Heineman EF. 1998. Brain cancer and occupational exposure to lead. J Occup Environ Med 40(11):937-42.

Hadfield MG, Adera T, Smith B, Fortner-Burton CA, Gibb RD, Mumaw V. 1998. Human brain tumors and exposure to metal and non-metal elements: a case control study. J Environ Pathol Toxicol Oncol 17(1):1-9.

Kheifets LI. 2001. Electric and magnetic field exposure and brain cancer: a review. Bioelectromagnetics Suppl 5:S120-31.

Muscat JE, Malkin MG, Thompson S, Shore RE, Stellman SD, McRee D, Neugut AI, Wynder EL. 2000. Handheld cellular telephone use and risk of brain cancer. JAMA 284(23):3001-7.

Pogoda JM, Preston-Martin S. 2001. Maternal cured meat consumption during pregnancy and risk of paediatric brain tumour in offspring: potentially harmful levels of intake. Public Health Nutr 4(2):183-9.

Preston-Martin S, Mack W. 1996. Neoplasms of the nervous system. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

Yeni-Komshian H, Holly EA. 2000. Childhood brain tumours and exposure to animals and farm life: a review. Paediatr Perinat Epidemiol 14(3):248-56.

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's Disease 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.

References

American Cancer Society. 2006. Cancer Facts & Figures 2006. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2006a. Breast Cancer. Available at: http://www.cancer.org.

American Cancer Society. 1998. The Risk Factors for Breast Cancer from: http://cancer.org/bcn/info/brrisk.html

Aschengrau A, Paulu C, Ozonoff D. 1998. Tetrachloroethylene contaminated drinking water and risk of breast cancer. Environ Health Persp 106(4):947-953.

Boyle P, Leake R. Progress in understanding breast cancer: epidemiological and biological interactions. Breast Cancer Res 1988;11(2):91-112.

Broeders MJ, Verbeek AL. Breast cancer epidemiology and risk factors. Quarterly J Nuclear Med 1997;41(3)179-188.

Davis DL, Axelrod D, Osborne M, Telang N, Bradlow HL, Sittner E. Avoidable causes of Breast Cancer: The Known, Unknown, and the Suspected. Ann NY Acad Sci 1997;833:112-28.

Ekbom A, Hsieh CC, Lipworth L, Adami HQ, Trichopoulos D. Intrauterine Environment and Breast Cancer Risk in Women: A Population-Based Study. J Natl Cancer Inst 1997;89(1):71-76.

Ellison RC, Zhang Y, McLennan CE, Rothman KJ. Exploring the relation of alcohol consumption to the risk of breast cancer. Am J Epi 2001; 154:740-7.

Goldberg MS, Labreche F. Occupational risk factors for female breast cancer: a review. Occupat Environ Med 1996;53(3):145-156.

Hansen J. Breast Cancer Risk Among Relatively Young Women Employed in Solvent-Using Industries. Am J Industr Med 1999;36(1):43-47.

Harris JR, Lippman ME, Veronesi U, Willett W. Breast Cancer (First of Three Parts). N Engl J Med 1992;327(5):319-328.

Henderson BE, Pike MC, Bernstein L, Ross RK. 1996. Breast Cancer, Chapter 47 in Cancer Epidemiology and Prevention. 2nd ed. Schottenfeld D and Fraumeni JF Jr.,eds. Oxford University Press. pp: 1022-1035.

Holford TR, Zheng T, Mayne ST, Zahm SH, Tessari JD, Boyle P. Joint effects of nine polychlorinated biphenyl (PCB) congeners on breast cancer risk. Int J Epidemiol 2000;29(6):975-982.

Kelsey JL. Breast Cancer Epidemiology. Epidemiol Reviews 1993;15:7-16.

Laden F, Hunter DJ. Environmental Risk Factors and Female Breast Cancer. Ann Rev of Public Health 1998;19:101-123.

Lewis-Michl EL, Melius JM, Kallenbach LR, Ju CL, Talbot TO, Orr MF, and Lauridsen PE. 1996. Breast cancer risk and residence near industry or traffic in Nassau and Suffolk counties, Long Island, New York. Arch Environ Health 51(4):255-265.

Lipworth L. Epidemiology of breast cancer. Eur J Cancer Prev 1995;4:7-30.

Madigan MP, Ziegler RG, Benichou J, Byrne C, Hoover RN. Proportion of Breast Cancer Cases in the United States Explained by Well-Established Risk Factors. J Natl Cancer Inst 1995;87(22):1681-5.

Massachusetts Cancer Registry (MCR) 2005. *Cancer Incidence and Mortality in Massachusetts 1998-2002: Statewide Report*. May 2005. Massachusetts Department of Public Health, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

McTiernan A. Exercise and Breast Cancer—Time To Get Moving? The N Engl J Med 1997;336(18):1311-1312.

Petralia SA, Vena JE, Freudenheim JL, Dosemeci M, Michalek A, Goldberg MA, Brasure J, Graham S. Risk of premenopausal breast cancer in association with occupational exposure to polycyclic aromatic hydrocarbons and benzene. Scandin J Work Envir Health 1999;25(3):215-221.

Safe SH. Endocrine Disruptors and Human Health—Is There a Problem? An Update. Environ Health Perspec 2000;108(6):487-493.

Segnan N. Socioeconomic status and cancer screening. International Agency for Research on Cancer 1997;138:369-376.

Swanson CA, Coates RJ, Malone KE, Gammon MD, Schoenberg JB, Brogan DJ, McAdams M, Potischman N, Hoover RN, Brinton LA. Alcohol Consumption and Breast Cancer Risk among Women under Age 45 Years. Epidemiology 1997;8(3):231-237.

Thune I, Brenn T, Lund E, Gaard M. Physical Activity and the Risk of Breast Cancer. N Engl J Med 1997;336(18):1269-1275

Ursin G, Ross RK, Sullivan-Haley J, Hanisch R, Henderson B, and Bernstein L. Use of oral contraceptives and risk of breast cancer in young women. Breast Cancer Res 1998;50(2):175-184.

van Dijck JAAM, Broeders MJM, Verbeek ALM. Mammographic Screening in Older Women, Is It Worthwhile? Drugs and Aging 1997;10(2):69-79.

Weiss HA, Potischman NA, Brinton L, Brogan D, Coates RJ, Gammon MD, Malone KE, Schoenberg JB. Prenatal and Perinatal Factors for Breast Cancer in Young Women. Epidemiology 1997;8(2):181-187.

Wohlfahrt J, Melbye M. Age at Any Birth is Associated with Breast Cancer Risk. Epidemiology 2001;12(1):68-73.

Wynder E, Cohen LA, Muscat JE, Winters B, Dwyer JT, Blackburn G. Breast Cancer: Weighing the Evidence for a Promoting Role of Dietary Fat. J Natl Cancer Inst 1997;89(11)766-775.

According to the American Cancer Society, approximately 21,260 Americans (13,000 men and 8,260 women) will be diagnosed with stomach cancer in 2007 (ACS 2007). Approximately 90-95% of these cases will suffer from an adenocarcinoma, a cancer which develops within the epithelial cells of the stomach's innermost lining, the mucosa. Less common types of stomach cancer include lymphoma of mucosa-associated lymphoid tissue, gastrointestinal stromal tumors, and carcinoid tumors. The majority of stomach cancers tend to occur in people over the age of 50, with most diagnoses happening after the age of 70. This type of cancer is more common in men than women, and is found more frequently among Asian, Pacific Islander, Hispanic, and African populations than in non-Hispanic white Americans (Shibata and Parsonnet 1996).

Stomach, or gastric, cancer is an increasingly rare form of cancer in the United States. It was once the leading cause of cancer deaths in the United States, yet since the mid-twentieth century, its prevalence has been drastically reduced. It is currently the seventh-leading cause of cancer deaths in the U.S. (NCI 2007). This reduction can be attributed to many factors, including increased refrigeration of foods and decreased consumption of salted and smoked meats. Some physicians feel that it can also be attributed to the widespread use of antibiotics to kill infections, such as *h. pylori*, which may increase one's risk for developing stomach cancer (NCI 2007). Stomach cancer is a much larger problem globally, particularly in underdeveloped nations. It is the second-leading cause of cancer deaths worldwide, with approximately 700,000 deaths in 2002 (ACS 2007).

While the exact cause of stomach cancer is unknown, many risk factors for the disease have been identified. Risk factors for stomach cancer include *h. pylori* infection, which can lead to chronic atrophic gastritis, a possible pre-cancerous change in the lining of the stomach (ACS 2007). *H. pylori* infection can also lead to the formation of peptic ulcers. The majority of people who carry the *h. pylori* bacterium do not develop cancer, but it has been confirmed as increasing one's risk for stomach cancer. This risk may be increased when someone is taking medicines known as histamine antagonists and proton-pump inhibitors (PPIs) to inhibit acid production in the stomach, which may, in turn, allow for increased bacterial growth. Many researchers now suggest eradication of *h. pylori* before beginning these medicines (Shibata and Parsonnet 1996).

Dietary factors may also affect one's risk for developing stomach cancer. Increased risk is associated with higher levels of consumption of smoked and salted fish and meats and pickled vegetables. Diets high in whole grains, fruits, and vegetables which contain vitamins A and C have been shown to reduce the risk of stomach cancer. Recent studies have also found that certain chemicals in barbequed and grilled muscle meats may increase cancer risk. These chemicals, known as heterocyclic amines (HCAs) are formed when muscle meats (beef, pork, fowl, and fish) are cooked at high temperatures for an extended period of time (to a medium-well or well-done temperature). Frying, boiling, and grilling cause the formation of most HCAs, but this effect can be somewhat negated by microwaving meats before cooking them.

Other notable causes for increased risk for stomach cancer include tobacco use. Smoking increases risk for cancers of the upper portion of the stomach closest to the esophagus, and the rate of stomach cancer is approximately doubled for smokers over nonsmokers (ACS 2007). Obesity has also emerged as a factor contributing to cancer in this area of the stomach.

Medical and familial history may also contribute to one's risk for developing stomach cancer. People who have had previous stomach surgery, such as a gastric bypass or removal of an ulcer, have an increased risk for stomach cancer due to a higher concentration of bacteria in the stomach and potential for reflux of bile from the small intestine, as well as a change in the pH balance of the stomach. According to the American Cancer Society, people with Type A blood have a higher risk for stomach cancer, for unknown reasons (ACS 2007). Pernicious anemia is also noted as a risk factor for stomach cancer. Inherited genetic disorders, such as hereditary nonpolyposis colorectal cancer and familial adenomatous polyposis cause a slightly increased risk for stomach cancer in individuals affected by the inherited gene mutations. Also, people with several first-degree relatives with stomach cancer are more likely to develop the disease.

References

American Cancer Society (ACS). 2007. Detailed Guide: Stomach Cancer. Available at http://www.cancer.org.

National Cancer Institute (NCI). 2007. What You Need to Known About Stomach Cancer. Available at http://www.cancer.gov.

Shibata A, Parsonnet J. 1996. Stomach Cancer. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni JF. New York: Oxford University Press.