ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts 1982-2003

CAMBRIDGE PLATING COMPANY
(a/k/a PURECOAT NORTH, LLC)
EPA FACILITY ID: MAD001034016
NOVEMBER 26, 2007

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

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PUBLIC HEALTH ASSESSMENT

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Prepared by:

Community Assessment Program
Bureau of Environmental Health
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Under a Cooperative Agreement with:
Agency for Toxic Substances and Disease Registry
U.S. Department of Health and Human Services

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SUMMARY

At the requests of the Belmont Department of Health, community residents, and in response to a legislative directive, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health conducted an evaluation of possible environmental exposures and cancer in relation to the Cambridge Plating Company, an active electroplating facility which has been in operation at 39 Hittinger Street in Belmont, Massachusetts since 1968. This project was conducted under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct health assessments in Massachusetts.

The investigation reviews available environmental data for the Cambridge Plating site and considers potential ways that people may come into contact with chemicals from the facility. The evaluation also looks at the pattern of cancer in Belmont and surrounding communities, focusing on residential neighborhoods near Cambridge Plating. Six cancer types were evaluated in this investigation: cancers of the kidney, liver, lung and bronchus, and pancreas as well as leukemia and non-Hodgkin's lymphoma (NHL). Using data from the Massachusetts Cancer Registry, rates for these cancer types were calculated for the town of Belmont as a whole and for the census tracts (CT) that comprise the town, with a particular focus on CT 3572, where Cambridge Plating is located, and the adjacent CT 3571. In addition, this report provides cancer incidence analyses for the surrounding communities of Arlington, Cambridge, and Watertown. Appendix A of the report presents more detailed information about the occurrence of cancer in all four communities as well as in relation to other locations of potential environmental concern. Available information about risk factors related to the development of cancer was also considered. In general, most of the six cancer types evaluated in relation to Cambridge Plating occurred near or below the rates expected for Belmont CTs 3571, 3572, and the town of Belmont as a whole during the 18-year time period, 1982–1999. With some exceptions, cancer incidence in the surrounding communities of Arlington, Cambridge, and Watertown was also near or below expected rates.

A number of potential exposure pathways exist, in particular potential exposure to historical air emissions from Cambridge Plating. Contaminants of potential concern at the facility include past emissions of trichloroethylene (TCE) and chromium. No ambient air sampling data are available, however, to determine whether air emissions from the facility have existed in the surrounding neighborhoods and/or the extent to which they might have contributed to cancer or noncancer health effects reported by residents in this area. It is possible that some neighborhood residents have experienced irritant health effects associated with air emissions from the facility. However, it is important to note that some individuals, particularly those with pre-existing conditions such as asthma and allergies, may experience irritant reactions that would not necessarily impact the general population similarly. There were no completed exposure pathways for area residents in relation to Cambridge Plating.

Sampling conducted previously at the Cambridge Plating site identified some contamination of groundwater and subsurface soil on the site. No available information suggests that contaminants such as volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons, and metals detected at levels above health-based comparison values in groundwater and soil pose a health threat to individuals living in neighborhoods adjacent to the facility under present conditions. For example, the groundwater beneath the site is not a source of drinking water, and it is unlikely that residents would come into contact with subsurface soil on the property since the majority of the site is paved. Based on the available environmental data reviewed, concentrations of VOCs detected in groundwater samples collected down-gradient and close to the site boundary near Hittinger Street are below levels expected to result in indoor air impacts in nearby homes. Based on recent site investigations, including additional characterization of groundwater conditions and flow patterns, it is not expected that residents north of the site along Channing Road would have opportunities for exposure to levels of VOCs in indoor air that could present a health risk.

Based on criteria established by ATSDR, the Cambridge Plating site would be classified as posing an Indeterminate Public Health Hazard in the past and No Apparent Public Health Hazard in the present and future. No ambient air sampling data exist; therefore, it cannot be determined whether people living near the Cambridge Plating site were at risk of exposure to air emissions (such as TCE or chromium) from the facility in the past. However, based on a review of

available environmental data, analysis of possible exposure pathways, and an evaluation of the pattern of cancer in the area surrounding the facility, results do not suggest that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in Belmont census tracts 3571 and 3572, in the town of Belmont as a whole, or in the surrounding communities during the 18-year time period, 1982–1999.

I. INTRODUCTION AND STATEMENT OF ISSUES

At the request of the Belmont Department of Health, community residents, and in response to a legislative directive, the Community Assessment Program (CAP) in the Bureau of Environmental Health of the Massachusetts Department of Public Health (MDPH), conducted an evaluation of cancer incidence in Belmont, Massachusetts, and the surrounding communities of Cambridge, Arlington, and Watertown. This project was conducted under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct health assessments in Massachusetts. This evaluation was initiated because of community concerns about possible environmental exposure and cancer in relation to the Cambridge Plating Company, an electroplating facility located at 39 Hittinger Street in Belmont. The facility has been in operation since 1968 and has been known by a variety of names during its history; the current electroplating operation at the site is Purecoat North, LLC. For the purposes of this report, however, all electroplating operations at 39 Hittinger Street will be referred to as Cambridge Plating.

This report evaluates the potential for exposure related to chemicals used at or emitted from Cambridge Plating and also provides a review of several types of cancer in Belmont and the surrounding communities of Cambridge, Arlington, and Watertown, with a particular focus on Belmont census tracts (CT) 3572 and 3571. Cambridge Plating is located in CT 3572, and CT 3571 is an adjacent census tract that is near the facility (shown in Figure 1). Appendix A of the report provides more detailed information about the occurrence of cancer in all four communities as well as in relation to other locations of potential environmental concern. In addition, available information about risk factors, including environmental factors, related to the development of cancer was considered. To evaluate concerns about potential exposure to hazardous substances from the Cambridge Plating site, MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) and the U.S. Environmental Protection Agency (EPA) to obtain and review available environmental information for the facility.

Cancer rates were evaluated for the town of Belmont and the census tracts that comprise the town and for Cambridge, Arlington, and Watertown for the years 1982–1999, the time period for which the most recent and complete cancer incidence data were available from the

Massachusetts Cancer Registry (MCR) at the initiation of this analysis. Belmont is divided into eight smaller geographic areas or census tracts (CTs). A census tract is a smaller geographic subdivision of a city or town designated by the U.S. Census Bureau. Because age group and gender specific population information is required to calculate incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated. Belmont CT 3572, where Cambridge Plating is located, comprises an area of 0.6 square miles and has a total population of 3,204 (U.S. Census Bureau 2000). Census tract 3571, which is adjacent, has an area of 0.8 square miles and a population of 4,148. Figure 1 shows the location of Cambridge Plating and the boundaries of CTs 3571 and 3572.

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, may help determine whether a common cause or etiology is possible, and can serve to identify areas where further public health investigations or actions may be warranted. Descriptive analyses may also indicate that an excess of known risk factors associated with a disease, such as environmental exposures, exists in a certain geographic area. This descriptive analysis of cancer incidence data cannot be used to establish a causal link between a particular risk factor (either environmental or nonenvironmental) and the development of cancer. In addition, this analysis cannot determine the cause of cancer for any one individual. The purpose of this evaluation is (1) to report the findings of the patterns of cancer in Belmont (with particular focus on CTs 3571 and 3572) and in the surrounding communities of Cambridge, Arlington and Cambridge and (2) to evaluate the findings in the context of the available environmental information related to Cambridge Plating to determine whether recommendations for further public health action are needed.

II. OBJECTIVES

The specific objectives of this investigation were as follows:

- To evaluate the extent to which contamination at or emissions from the Cambridge Plating facility could result in exposure to people in the area and whether adverse health effects would be possible if exposure occurred.

- To review the incidence of six cancer types in the town of Belmont and the surrounding communities of Cambridge, Arlington, and Watertown, with a particular focus on CTs 3571 and 3572 in Belmont.

- To evaluate the geographic distribution of individuals diagnosed with cancer in the four communities and see if there are any patterns in particular areas of the communities or in areas of potential environmental concern.

- To review descriptive information available from the Massachusetts Cancer Registry for individuals diagnosed with cancer in the four communities, to see if there are any particular characteristics related to known or suspected risk factors, including environmental factors, for developing these diseases.

- To discuss possible exposure pathways related to Cambridge Plating and the results of the cancer incidence evaluation in the context of the available scientific and medical literature on cancer and contaminants of concern to determine whether further investigation or public health action is warranted.

III. BACKGROUND AND COMMUNITY ENVIRONMENTAL CONCERNS

Community environmental concerns related to Cambridge Plating Company have focused specifically on historical air emissions as well as groundwater and soil contamination identified on the property. Residents have expressed concerns about cancer and several non-cancer health outcomes including upper respiratory irritation, nausea, and headaches. In addition, complaints about odors and noise have been reported by individuals residing near the facility (Belmont Health Department 2003a; 2003c). In order to address these community concerns, the MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) Metro/Northeast Regional Office and the U.S. Environmental Protection Agency (EPA) Region 1 to obtain and review available environmental information pertaining to Cambridge Plating.

Cambridge Plating is located at 39 Hittinger Street in Belmont, Massachusetts (see Figure 2). This site is located in census tract 3572 (shown in Figure 1). Since 1968, electroplating operations have been conducted at the site (Coler and Colantonio 2002a). A number of other

commercial and industrial activities are reported to have occurred at the site before the electroplating operations began. One company used the site to produce concrete burial vaults and bricks (Coler and Colantonio 2002a). Historical operations reported at or near the Cambridge Plating site include a clay mining operation and, in the early 1900s, an open burning town dump (Coler and Colantonio 2002a).

Cambridge Plating is regulated for air emissions, sewage discharge, on-site storage of hazardous waste, and transport of hazardous waste off the site for disposal. There have been several spills or accidental releases reported and investigated at the Cambridge Plating facility during the company's years of operation; a partial drum spill of muriatic acid in 1980; a tank overfill of diesel fuel in 1982; and, in 1983, the discovery of soil contaminated with diesel fuel from a former underground storage tank (Paragon Environmental Services 1996). Some fires have also been reported at the site, including an indoor office fire on March 15, 1998, and a fire in the wastewater treatment system area on May 25, 2002 (Coler and Colantonio 1998c; EPA Region 1 2003a).

In 1989, the detection of an apparent release of unspecified hazardous materials on a portion of the property resulted in regulatory actions that required subsurface environmental sampling to establish the extent of contamination. Site investigations that included sampling of on-site soil and groundwater were completed in 1996 and 2002. More recently, soil contaminated with chromium discovered in a portion of the property adjacent to the Massachusetts Bay Transportation Authority (MBTA) railroad tracks resulted in a site investigation of this area in May/June 2003 (Coler and Colantonio 2003a). In addition, in response to an MDEP requirement, an investigation of groundwater and soil gas was conducted at the site in January 2004 (Coler and Colantonio 2004). Additional investigations of groundwater and on-site surface soil were conducted in response to an MDEP requirement in 2005 (OHI 2005). Off-site surface soil sampling was conducted by MDEP in 2007 (MDEP 2007). The available environmental data from these investigations, along with information provided by EPA, were considered in this public health assessment.

Within the immediate vicinity of the Cambridge Plating facility there are a range of land uses. To the north, Cambridge Plating is abutted by the MBTA commuter rail tracks and by residential

and commercial properties beyond the tracks. To the east are Brighton Street and a convenience store, a gas station, and a small business park. A residential neighborhood is located south of the facility and to the west there are school playing fields and a school parking lot. The Belmont High School building is approximately 500 feet away.

Cambridge Plating is located within the Mystic River Watershed and is not located within a potentially productive aquifer (Coler and Colantonio 2002a). Nearby surface water bodies include Clay Pit Pond, which is in front of Belmont High school; Blair Pond, which is behind the commercial buildings on Brighton Street to the east; and Little Pond, which is a fifth of a mile north of the site (locations are shown in Figure 2).

The public health assessment titled "*Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999*" was released on October 20, 2005, for a public comment period ending on December 15, 2005. Public comments were received by the MDPH and are addressed in Appendix E and Appendix F.

IV. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with Cambridge Plating, MDPH reviewed information from several reports on file with MDEP and EPA, as well as the Toxics Release Inventory (TRI) data available from EPA. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify substances that need to be considered for further analysis to determine whether they may be of potential health concern. The screening analysis identifies maximum concentrations of contaminants detected in various types of environmental media (i.e., air, soil, water) and compares these concentrations to health-based comparison values established by ATSDR (ATSDR 2003a, 2003b). If an ATSDR comparison value was not available for a specific chemical, the maximum detected concentration of that chemical was compared to risk-based concentrations (RBCs) developed by EPA Region 3 (Hubbard 2000) or the applicable groundwater or soil standards established by MDEP (MDEP 2003b), in that order. For compounds detected in groundwater, maximum concentrations were also compared with state or federal drinking water standards.

The ATSDR comparison values are specific concentrations of a chemical for air, soil, or water that are used by health assessors to identify environmental contaminants that require further evaluation. These comparison values are developed on the basis of health guidelines and assumed exposure situations that represent conservative estimates of human exposure. Chemical concentrations detected in environmental media at levels that are lower than their comparison values are not likely to pose a health threat. However, the fact that a chemical concentration is detected in environmental media at a level above a comparison value does not necessarily mean that a person who comes into contact with that concentration will be harmed. If the concentration of a chemical is greater than the appropriate comparison value, the potential for exposure to the chemical is further evaluated to determine whether exposure is occurring and whether health effects might be possible as a result of that exposure. The factors related to exposure that are unique to the specific situation under investigation need to be considered to determine whether adverse health effects from a chemical of concern are plausible.

ATSDR has identified levels of metals and polyaromatic hydrocarbons (PAHs) that are considered normal for soil in urban and suburban communities. The United States Geological Society (USGS) has also identified measurements for levels of metals that are considered typical for soil in the eastern United States (Shacklette and Boerngen 1984). These levels are called background levels and are used along with comparison values for both metals and PAHs in this analysis.

A. Soil

Soil sampling has been conducted at the Cambridge Plating site for a variety of reasons, ranging from environmental sampling associated with specific releases of contaminants to environmental sampling for more comprehensive site investigations. The majority (n = 18) of the samples were collected from soil between 2 to 8 feet below the ground surface. Typically, surface soil samples are taken from the top 0–3 inches of soil. This is of particular interest when evaluating possible exposure as it is more likely that individuals would have more frequent contact with surface soil than with deeper soils. However, the soil samples for Cambridge Plating, with the exception of samples collected for chromium analysis, were not collected from soil shallower than 2 feet in depth.

The maximum value for each contaminant for which environmental data were available from the file review was compared to the appropriate screening value. Analysis of the on-site soil samples found arsenic, chromium, trichloroethylene, and a range of polycyclic aromatic hydrocarbons (PAHs) at levels above relevant comparison values (see Table 1). Sampling locations are shown in Figure 3. The information reviewed indicated that no off-site soil sampling was conducted.

Arsenic was detected in five of eight soil samples collected at Cambridge Plating. The maximum concentration of 44 milligrams per kilogram (mg/kg) was detected in soil sampling conducted during excavation activities for the installation of a new loading dock in 1994. However, subsequent sampling of soil in the same area found lower levels of arsenic. Based on the information reviewed, it is unclear whether excavated soils were removed from the site. In the other four samples with elevated levels of arsenic, the levels ranged from 5.1 mg/kg to 7.6 mg/kg. Although all five detected arsenic concentrations exceed ATSDR's cancer risk evaluation guide (CREG) for arsenic in soil of 0.5 mg/kg, all of the levels detected are within the range of typical background concentrations for soil in the eastern United States (ATSDR 1993; Shacklette and Boerngen 1984). Table 1 shows the maximum concentrations of contaminants detected in the on-site soil samples.

Elevated levels of total chromium were detected in on-site soil during sampling conducted in response to a release identified in an approximate 450-square foot area of the property located north of the building abutting the MBTA railroad tracks. The maximum concentration of total chromium (32,000 mg/kg) was detected in a composite soil sample collected 3 to 5 feet below ground surface in this area. Total chromium was detected in 25 of the soil samples from this area; concentrations exceeded the reference media evaluation guide (RMEG) value for childhood exposure to chromium VI (200 mg/kg) nine times and for adult exposure to chromium VI (2,000 mg/kg) three times. There are no ATSDR comparison values for total chromium in soil so RMEGs for chromium VI, which is the most toxic form of chromium, were used as comparison values.

Because chromium VI, the hexavalent form of chromium, is a carcinogen, soil samples collected from the area north of the building were also analyzed for chromium VI. The maximum

concentration of chromium VI (210 mg/kg) was detected in subsurface soil, approximately at or below the ATSDR comparison values (see Table 1). Chromium levels identified in subsurface soils from other areas of the site were well below comparison values. Sampling of surface soil in this area (discussed in detail below) indicates that both total chromium and hexavalent chromium levels are approximately at or below the ATSDR comparison values.

In November 2005, Cambridge Plating submitted surface soil sampling results for both total chromium and chromium VI in response to a requirement by the Massachusetts Department of Environmental Protection (MDEP) (OHI 2005). Ten additional on-site locations were sampled, including three locations along Hittinger Street, three along the MBTA railroad tracks, two along the fence adjacent to the baseball field, and two along Brighton Street. These locations were required by MDEP based on air emissions modeling results indicating that areas of maximum impact would be located on the site; this modeling was done in order to select reasonably conservative sampling locations (J. Miano, MDEP, personal communication, 2006). All OHI samples were collected from surface soils at depths between 0 and 6 inches and analyzed for both total and hexavalent forms of chromium. As previously mentioned, surface soil samples are of particular interest when evaluating possible exposure as it is more likely that individuals would have more frequent contact with surface soil than with the deeper soils discussed above. Total chromium was detected in all ten surface soil samples at concentrations ranging from 23 to 220 mg/kg (OHI 2005). The maximum detected concentration of total chromium was below the RMEG value for adult exposure to chromium VI (2,000 mg/kg) and similar to the RMEG value for childhood exposure to chromium VI (200 mg/kg) (ATSDR comparison values for total chromium were not available, so comparison values for chromium VI were used). Chromium VI was detected in one of the ten surface soil samples at 1.2 mg/kg (OHI 2005), which is well below both the adult and child RMEG values for chromium VI.

In June 2007, in response to a request from the community and a MDPH recommendation in the Cambridge Plating Public Health Assessment report released for public comment in 2005, the MDEP sampled surface soil at off-site locations near Cambridge Plating to confirm the results of air emissions modeling and to determine if elevated levels of chromium were present in off-site soil. The seven off-site samples were located near the tennis courts and ball field east of the facility, near residences north of the facility, near the intersection of Hittinger and Baker Streets

south of the facility, and at three locations along Brighton Street northwest, west, and southwest of the facility (Figure 4). Chromium VI (hexavalent) was not detected in any of the seven off-site surface soil samples (MDEP 2007). Total chromium was detected in all seven samples at concentrations ranging from 12 to 46 mg/kg, but were well below ATSDR comparison values for chromium VI (MDEP 2007). These concentrations were also below concentrations of total chromium considered typical for soil in the eastern United States (1-1,000 mg/kg) (Shacklette and Boerngen 1984).

The maximum level of benzo(a)pyrene (11 mg/kg) detected exceeds the ATSDR soil CREG of 0.1 mg/kg. There are a number of other PAHs that exceed EPA's risk-based concentrations (RBCs). RBCs were used because there are no ATSDR comparison values for these compounds. However, the majority of PAH compounds detected (benzo(a)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene) were within the range of typical background concentrations for urban soil. Because PAHs are by-products of incomplete combustion processes, they have been reported to be associated with previous land uses in this area, such as brick making and an open-burn dumpsite (Coler and Colantonio 2002a).

B. Groundwater

There are 17 monitoring wells at the Cambridge Plating site that have been used to collect groundwater samples on several different occasions. Groundwater at the site is between 3 and 7 feet below the surface (Coler and Colantonio 2004). At some of these wells, arsenic, trichloroethylene (TCE), cis-1,2-dichloroethene, vinyl chloride, and PAHs have been detected at levels above drinking water comparison values (Table 2 and Figure 3). However, groundwater at the Cambridge Plating site is not used as a drinking water supply, and all of Belmont is serviced with municipal water. The information reviewed does not indicate that any groundwater sampling has been conducted off the site.

There is little historical groundwater sampling of volatile organic compounds (VOCs) for the majority of monitoring wells at the site, and no specific time trends are apparent. Elevated levels of TCE and cis-1,2-dichloroethene (cis-1,2–DCE) have been consistently detected in samples collected from wells located west of the main building and down-gradient from the locations of floor drains and sump tanks in the facility (monitoring wells CC-105 and MW-03 – see Figure

3). Under an agreement with MDEP, a quarterly monitoring program for these two wells is currently in place to help determine whether the site represents a current source of TCE and cis-1,2-DCE to groundwater (Coler and Colantonio 2002b). The maximum groundwater concentrations of TCE (49,800 micrograms per liter [µg/L]) and cis-1,2 DCE (8,150 µg/L) were detected in monitoring well MW-03 during a 2004 sampling event (Coler and Colantonio 2004). Both concentrations exceed EPA's RBCs for drinking water (ATSDR comparison values were not available). Groundwater concentrations measured during sampling in 2005 showed lower levels of both TCE (360 µg/L) and cis-1,2 DCE (4,500 µg/L) in monitoring well MW-03, but both concentrations continue to exceed the EPA's RBCs for drinking water (OHI 2005). Vinyl chloride was detected in three out of ten monitoring wells sampled and the maximum concentration of vinyl chloride (1,200 µg/L) was detected above the CREG value for drinking water (0.03 µg/L) in monitoring well MW-03 in September 2005. The source of these elevated VOC levels has not been determined, although both cis-1,2–DCE and vinyl chloride are known breakdown products of TCE (ATSDR 1997). Depths to groundwater in monitoring wells MW-03 and CC-105 wells during 2002–2004 sampling ranged from 5.95 feet–6.69 feet and 3.41 feet–4.18 feet, respectively (Coler and Colantonio 2004).

While TCE and cis-1,2, DCE were detected at much lower levels in monitoring well CC-2, which is located closer to the property boundary near Hittinger Street, the maximum concentration of TCE detected in this well (380 µg/L in 2000) exceeds available drinking water comparison values (Coler and Colantonio 2002a). Vinyl chloride was not detected in this well. Monitoring well CC-2 is located approximately 160 feet south of monitoring well MW-03 (refer to Figure 3).

Additionally, in an *Interim Deadline* letter dated May 20, 2005, MDEP required that Cambridge Plating Company obtain a groundwater sample from beneath the floor in the TCE degreasing area or boiler room in order to investigate the source of elevated levels of VOCs at the loading dock on the southern side of the building (MDPH 2005b). The groundwater sample was taken 10 feet below the floor of the degreasing area by OHI Engineering, Inc. The sample had detections of TCE (15 µg/L), cis-1,2 DCE (20 µg/L), and vinyl chloride (5 µg/L) (OHI 2005).

C. Air Emissions

Cambridge Plating first submitted an application for an air quality emissions permit to the MDEP Bureau of Waste Prevention (BWP) in 1982 and the most recent permit was submitted in 1995 (MDEP 1982, 1995). The major sources of air emissions at the facility have been two solvent degreasers and a hard chrome-plating bath (MDEP 1995). However, routine stack sampling is not required under the air-permitting program and no ambient air sampling data are available for the neighborhood areas in the immediate vicinity of the facility.

A review of data reported in EPA's Toxics Release Inventory (TRI) was conducted. The TRI is a reporting system that estimates the annual releases of toxic chemicals to the environment. The system evolved from the Emergency Planning and Community Right-to-Know Act. Businesses are required to report the locations and quantities of chemicals stored on a site to state and local agencies to help communities prepare to respond to potential chemical spills and emergency releases (EPA 2005). Although TRI annual release estimates cannot be used to specifically evaluate whether individuals living near the Cambridge Plating site are actually at risk of exposure to air emissions, the information can be helpful when evaluating the pattern of cancer in the residential areas surrounding the facility and the possibility that environmental factors may have played a role.

Review of available TRI data for Cambridge Plating for the years 1987–2005 indicates that fugitive emissions (emission from sources other than stacks or vents) of TCE were highest in the late 1980s. The maximum TCE emissions (66,960 pounds) were reported for the year 1989. Annual TCE emissions fluctuated between approximately 14,000 and 19,000 pounds in the intervening years, with another peak in emissions reported for the year 1996 (58,500 pounds). TRI emissions data indicate that TCE was last released from the facility in 2003 when fugitive TCE emissions at Cambridge Plating were 11,638 pounds (EPA 2005). Currently, air emissions of TCE have been eliminated because TCE is no longer being used at the facility (Belmont Department of Health 2004a, U.S. EPA Region 1 2005). Other compounds reported to the TRI as being emitted to air from the Cambridge Plating facility at some point during the years for which TRI data were available included hydrochloric acid, nickel compounds, nitric acid, sodium hydroxide solution, and sulfuric acid (see Table 3).

While not reported in TRI data, air permit applications and the presence of a hard chromium-electroplating process at the facility indicate that Cambridge Plating has also been a source of hexavalent chromium air emissions (MDEP 1995). Although there is no information on what time chromium-electroplating operations began at the facility, it was reported that the "hard chrome" tank was installed in 1969 (Cambridge Plating 2001). On the basis of the permit information reviewed, the hard chromium electroplating process area was located toward the back portion of the building (away from Hittinger Street) and the stack height associated with this process was reported to be 20 feet above the ground surface (MDEP 1995). According to the 1995 permit, stack emissions of chromium were reported to be 0.007 tons per year (MDEP 1995).

According to EPA Region 1, the applicable air regulations for chromium came into effect for Cambridge Plating in 1995. While some form of air pollution control was probably in place prior to that, previous emissions and/or controls of chromium from the facility are unknown (EPA Region 1 2003b). In 2002, Cambridge Plating undertook an agreement with EPA to completely close the chromium-electroplating portion of its operations (EPA Region 1 2003b, 2002b). As a result, chromium air emissions associated with the use of this process ended in December 2002. While a "chromating" process is still in operation, it is not heated and air emissions are not expected (EPA Region 1 2005).

V. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS

An evaluation of potential pathways of exposure was conducted to help evaluate whether emissions or releases at the Cambridge Plating facility could be affecting the health of residents in the surrounding neighborhood in the present, could affect the health of residents in the future, or could have affected the health of residents in the past. A person must first be exposed to a chemical before any potential adverse health effects can result. Five conditions must be present for exposure to a chemical to occur. First, there must be a source of the chemical. Second, an environmental medium must be contaminated by either the source or by chemicals transported away from the source. Third, there must be a location where a person can potentially come into contact with the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, or dermal

absorption. Fifth, there must be a population to be exposed. Even if a person is exposed to a chemical, it doesn't mean that the person will be harmed. For a person to be harmed by exposure, the chemical must actually reach the target organ susceptible to the toxic effects caused by that particular substance at a sufficient dose and for a sufficient exposure time for an adverse health effect to occur (ATSDR 1993).

A completed exposure pathways exists when all of the five conditions previously described are present. A potential exposure pathway exists when one or more of the five elements is missing or uncertain and indicates that exposure could have occurred in the past, may be occurring in the present, or could occur in the future. An exposure pathway can be eliminated if at least one of the five elements is missing and will not likely be present in the future. Refer to Table 4 for a summary of exposure pathways discussed in this section.

A. Exposure to Soil

Most of the Cambridge Plating site is covered by asphalt or concrete (Coler and Colantonio 2002a; Paragon Environmental Services 1996), so it is unlikely that residents who live nearby will be exposed to soil from the site. In addition, the facility is still in active industrial use; therefore, it is unlikely that non-employees would be accessing the site extensively. Reports in the Belmont Health Department files indicate that young adults have trespassed on the Cambridge Plating property in the past; specifically, there was an incidence of youth climbing on the roof of the building (Belmont Department of Health 2003a, 2003c). It is possible that individuals who trespassed near a portion of the property abutting the MBTA railroad tracks could have had infrequent contact with chromium in soil before the chromium contamination was discovered and access restricted by a fence. Assuming that an older child trespassed on this area of the site in the past and incidentally ingested the maximum concentration of total chromium detected in soil samples collected between 0 and 4 feet below ground surface (3,900 mg/kg), for 1 day per week for 26 weeks over 5 years, the level would be below EPA's chronic Reference Doses for both Chromium III and Chromium VI (ATSDR Minimal Risk Levels and

U.S. EPA cancer slope factors were not available for total chromium)[1]. Therefore, it is unlikely that individuals who might have trespassed in this area of the site would have had sufficient exposure to result in health effects.

In the 2005 public comment release of the public health assessment titled "*Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999,*" the MDPH recommended additional surface soil sampling and analysis for chromium to determine those areas most impacted by chromium deposition in the past. Air modeling was conducted by the MDEP during 2006. The model used meteorological conditions, stack emission rate, and other model inputs to predict the relative distribution of chromium that might be present in soils in the vicinity of Cambridge Plating due to stack emissions and deposition. Based on the modeling, which used regional data for values such as wind speed and wind directions, MDEP predicted that the areas with the highest concentrations of chromium would likely be located on the site.

As part of Phase II assessment work at the Cambridge Plating site, MDEP required that ten specific locations around the perimeter of the property be sampled to determine levels of chromium in surface soils. The November 2005 sampling locations were chosen by MDEP based on air emissions modeling results which indicated that areas of maximum impact would be located on the site. An analysis of the soil sampling data indicated that the chromium levels present in the on-site surface soil were low and are unlikely to result in adverse health effects in nearby residents (see Section IV-A).

To further confirm and validate the results of air emissions modeling in regards to off-site conditions, in June 2007, the MDEP sampled surface soil at off-site locations near Cambridge Plating. Concentrations of chromium measured at off-site areas were below both ATSDR comparison values and concentrations considered typical for soil in the eastern United States.

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[1] Noncancer Effects Exposure Factor = $\frac{(1 \text{ day/week})\ (26 \text{ weeks/year})\ (5 \text{ years})}{(5 \text{ years})\ (365 \text{ days/year})} = 0.07$

Maximum total chromium detected in samples collected at depths from 0 to 4 feet = 3,900 mg/kg

Noncancer Effects Exposure Dose = $\frac{(3900 \text{ mg/kg})\ (200 \text{ mg/day})\ (0.07)\ (1 \text{ kg}/10^6 \text{ mg})}{35 \text{ kg}} = 1.6 \times 10^{-3}$ mg/kg/day

EPA's Chronic Rfd (Chromium III) = 1.5 mg/kg/day

EPA's Chronic Rfd (Chromium VI) = 3×10^{-3} mg/kg/day

Therefore, chromium in off-site soil is unlikely to result in adverse health effects in nearby residents.

Workers at the site who are involved with electroplating activities are unlikely to be exposed to soil beneath the site. Although it is possible that workers who visit the site for construction or excavation activities (such as undertaking soil borings for the purposes of environmental analysis or removing or replacing underground storage tanks) may be exposed in the future, such activities are likely to be undertaken using proper health and safety precautions to minimize exposure potential.

A source of concern to nearby residents has been the Cambridge Plating facility's history of non-compliance with wastewater limits and discharge to the sewer (Belmont Department of Health 2003c). Although the sewer pipe that runs along Hittinger Street was cracked in the past, it is currently reported to be in good condition (Belmont Department of Community Development 2003) and revealed no evidence of damage or leakage (OHI 2005). It is possible that wastewater discharged to the sewer from this facility resulted in some contamination of surrounding soils and possibly groundwater in the vicinity of any cracks. It is unlikely, however, that residents of this area would be exposed to such contamination because the potentially impacted soils are located beneath the ground surface and, as stated previously, groundwater in this area is not a source of drinking water.

B. Exposure to Groundwater

A recent groundwater elevation study showed a generally southern groundwater flow direction on the eastern portion of the facility and a southwesterly flow on the western side of the facility. Groundwater at the site is between 3 and 7 feet below the surface (Coler and Colantonio 2004). The highest levels of VOC contamination detected in groundwater are in monitoring wells MW-03 and CC-105, which are west of the Cambridge Plating buildings (Figure 3). VOC concentrations detected in groundwater are much lower closer to the southern property boundary near Hittinger Street, but the concentrations of TCE detected in the most westerly monitoring well (CC-2) were also elevated above available drinking water comparison values (MCL = 5 µg/L). Specifically, TCE was detected in groundwater samples from well CC-2 at 380 µg/L, 200

µg/L, and 162 µg/L during the years 2000, 2003, and 2004, respectively (Coler and Colantonio 2002; 2004).

The groundwater wells sampled at Cambridge Plating are for monitoring purposes only, and no one drinks water from these wells. All residential properties in the town of Belmont are supplied with drinking water from the Massachusetts Water Resources Authority, and there are no known private drinking water wells (MWRA 2003; Belmont Department of Health 2003b). Because groundwater in this area is not being used as a source of drinking water, ingestion is not a possible route of exposure for residents.

C. Exposure to Indoor Air

Data currently available for VOCs in groundwater indicate that the highest levels of TCE and 1,2-DCE were detected west of the Cambridge Plating building and on the site (monitoring wells CC-105 and MW-03), and that levels closer to the southern site boundary near Hittinger Street were lower but still elevated above drinking water comparison values. Volatilization of VOCs to indoor air in nearby homes would be possible if groundwater is shallow and VOCs are present in groundwater beneath homes at sufficient concentrations.

To evaluate whether the levels of TCE detected in groundwater at the site could contaminate indoor air in nearby residences, MDPH asked ATSDR to run a model incorporating site-specific information on groundwater, soil, and housing for the area surrounding the facility (ATSDR 2003c). ATSDR used information on the predicted groundwater flow direction at the Cambridge Plating site, the depth to contamination, the type of soil, and the typical size and age of nearby homes and applied the Johnson-Ettinger mathematical model to determine what concentrations of TCE in groundwater would result in indoor air concentrations of TCE at levels above health-based comparison values for air exposure. Modeling results indicated that the potential for a vapor intrusion exposure pathway could exist when TCE is present in groundwater at concentrations greater that 12,800 µg/L (ATSDR 2003c). With the exception of monitoring wells MW-03 and CC-105 located west of the Cambridge Plating building, TCE was detected below the groundwater concentration at which a vapor intrusion exposure pathway would be of potential concern at the site. In particular, the concentrations of TCE detected in groundwater monitoring well CC-2 located down-gradient and closer to the site boundary near Hittinger Street

were 380 µg/L and lower; therefore, it is unlikely that persons living near this area of the site are being exposed to TCE in indoor air at levels of health concern.

Because of the historical levels of VOCs detected in groundwater wells and uncertainties associated with the direction of groundwater flow at the site, MDEP required Cambridge Plating to evaluate whether VOCs in groundwater are resulting in soil gas that could impact nearby residences along Hittinger Street (MDEP 2003a). Results of groundwater sampling from this investigation showed that levels similar to those detected in monitoring wells MW-03 and CC-105 are not widespread and do not appear to be migrating off the site (Coler and Colantonio 2004). Soil gas sampling conducted both adjacent to the building and closer to Hittinger Street indicated that VOCs in soil gas decrease significantly in sampling locations closer to the property boundary. This investigation also applied a dilution attenuation factor of 1,000 to measured VOC soil gas concentrations as a screening approach to estimate possible indoor air concentrations (Coler and Colantonio 2004). None of the estimated VOC indoor air concentrations exceeded ATSDR comparison values for air exposure.

While the environmental information reviewed indicates a south/southwestern groundwater flow direction at the site, there was little information available on the concentrations of TCE in groundwater north of the building. In the public comment release of the public health assessment titled "*Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999,*" the MDPH recommended additional characterization of VOCs in groundwater north of the building. Additional groundwater characterization, conducted by OHI Engineering, Inc. on behalf of Cambridge Plating Company, was submitted to the MDEP in November 2005. Using the new information about groundwater conditions, MDEP determined that shallow groundwater flow is generally in a south-southwest direction across the site (J. Miano, MDEP, personal communication, 2006). According to the MDEP, it is unlikely that VOCs found at high concentrations near the loading dock would flow upgradient (i.e., against the flow of groundwater) toward homes north of the site. Thus, because groundwater does not appear to flow toward these residences, it is not expected that residents north of the site have opportunity for exposure to contaminants in groundwater via vapor intrusion.

Additionally, in an *Interim Deadline* letter dated May 20, 2005, MDEP required that Cambridge Plating Company obtain a groundwater sample from beneath the floor in the TCE degreasing area or boiler room in order to investigate the source of elevated levels of VOCs at the loading dock on the southern side of the building. The groundwater sample was taken 10 feet below the floor of the degreasing area by OHI Engineering, Inc. The groundwater sample had detections of TCE (15 µg/L), cis-1,2 DCE (20 µg/L), and vinyl chloride (5 µg/L) (OHI 2005). These levels were much lower than concentrations of VOCs in other areas of the Cambridge Plating property: 49,800 µg/L TCE, 8,150 µg/L cis-1,2 DCE, and 1,200 µg/L vinyl chloride. Therefore, OHI Engineering, Inc. concluded and MDEP concurred that there did not appear to be an ongoing source of VOC contamination beneath the Cambridge Plating building that could contribute to vapor intrusion in homes north of the facility.

On the basis of the Johnson-Ettinger modeling results provided by ATSDR and soil gas and groundwater investigations at Cambridge Plating required by MDEP, high levels of VOCs detected in groundwater near the building (monitoring wells MW-03 and CC-105) do not appear to have migrated south toward the site boundary at sufficient concentrations to raise health concerns related to VOCs in indoor air in nearby homes along Hittinger Street. Also, because groundwater from beneath the Cambridge Plating facility does not appear to flow toward the northern site boundary, it is not expected that residents north of the site would have opportunities for exposure to elevated levels of VOCs in indoor air.

D. Exposure to Ambient Air

On the basis of the air permit information and TRI data reviewed, opportunities for exposure to ambient air emissions from Cambridge Plating are a possibility for nearby residents. While TCE is no longer being used at the facility, it is possible that fugitive emissions of TCE in the past may have resulted in potential exposure. Stack emissions of chromium associated with electroplating operations at the facility may also have resulted in exposure opportunities to chromium in ambient air in the past. As stated previously, chromium-electroplating operations that resulted in stack emissions of chromium were eliminated in December 2002.

There are no historical ambient air data available for Cambridge Plating or the surrounding neighborhood, making it difficult to evaluate whether facility emissions may have resulted in

chemical concentrations in ambient air greater than health-based screening values. However, because the majority of air emissions at Cambridge Plating are fugitive (that is, not from stacks or vents) and because stack heights are relatively low, the greatest potential for exposure to facility emissions in ambient air would likely be in the nearby residential neighborhoods of Belmont. The emissions would be less likely to impact neighborhoods farther away (e.g., in the towns of Arlington, Cambridge, and Watertown). Therefore, the pattern of those cancer types suggested in scientific studies of workers and/or animals to be associated with inhalation exposure to TCE and chromium (i.e., kidney, liver, lung, non-Hodgkin's lymphoma, and leukemia) were evaluated in relation to the Cambridge Plating facility as part of the cancer incidence analysis.

VI. ANALYSIS OF CANCER INCIDENCE

A. Methods for Analyzing Cancer Incidence

1. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–1999 were obtained for the town of Belmont and the surrounding communities of Arlington, Cambridge, and Watertown from the Massachusetts Cancer Registry (MCR), a division of the Bureau of Health Information, Statistics, Research and Evaluation within the MDPH. Six cancer types were evaluated in this investigation: cancers of the kidney, liver, lung and bronchus, and pancreas as well as leukemia and non-Hodgkin's lymphoma (NHL). Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. (The incidence coding definitions used in this report for these cancer types are shown in Appendix B.) These cancer types were selected for evaluation on the basis of elevations observed at the town level in a preliminary review of cancer rates in Belmont, potential associations with contaminants of concern at the Cambridge Plating site (primarily TCE and chromium), and/or residents' concerns over suspected elevations in some cancer types. Only cases reported to the MCR as a primary site cancer for one of the six cancer types and diagnosed among residents of Belmont, Arlington, Cambridge, and Watertown were included in the

analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis.

The MCR is a population-based surveillance system that began in 1982 to collect information on Massachusetts residents diagnosed with cancer in the state. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111s.111B). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment). Once these steps are finished, the data for that year are considered "complete." Due to the volume of information received by the MCR, the large number of reporting facilities, and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of 2 years prior to the current date. The 18-year period, 1982–1999, constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis.[2]

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

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital/diagnostic facility

[2] The data summarized in this report are drawn from data entered on MCR computer files before April 28, 2003. The numbers presented in this report may change slightly in future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than 2 months after the initial diagnosis (MCR 1996). Therefore, duplicate reports of an individual diagnosed with cancer were removed from the analyses whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases. In the town of Belmont, four duplicate reports were identified during the years 1982–1999 and excluded from the analyses. In addition, seven duplicate reports in Arlington, 16 duplicate reports in Cambridge, and 12 duplicate reports in Watertown were identified during the years 1982–1999 and excluded from the analyses.

2. Calculation of Standardized Incidence Ratios

To determine whether elevated numbers of cancer cases occurred in Belmont and the surrounding communities of Arlington, Cambridge, and Watertown, cancer incidence data were tabulated by gender according to six age groups to compare the observed number of cancer cases to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for the period 1982–1999 for each of the six primary cancer types for Belmont as a whole as well as for each census tract (CT) within Belmont. In addition, SIRs were calculated for the city of Cambridge as a whole and the three Cambridge CTs located on the border of Belmont. Townwide SIRs were also calculated for Arlington and Watertown. SIRs were also calculated for three smaller time periods, 1982–1987, 1988–1993, 1994–1999, to evaluate patterns or trends in cancer incidence over time.

To calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated on the basis of 1980, 1990, and 2000 U.S. Census data for each CT in Belmont and Cambridge and for the town of Belmont as a whole, the city of Cambridge as a whole, and the towns of Arlington and Watertown (U.S. Census Bureau 1980, 1990, and 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990, and 1996). To estimate the population between census years, an assumption

was made that the change in population occurred at a constant rate throughout the 10-year interval between each census.[3]

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

According to the U.S. Census, the town of Belmont is subdivided into eight census tracts (i.e., CTs 3571 through 3578). Three of the 30 census tracts in Cambridge are located on the border of Belmont (i.e., CTs 3543, 3546, and 3549) (U.S. Census Bureau 2000). The town boundaries and census tract locations for Belmont and surrounding communities are shown in Figure 1. Cases for which census tract designation was not possible were included in the city/town totals for Belmont and Cambridge.

3. Interpretation of a Standardized Incidence Ratio

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

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

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

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

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on four expected cases and six observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed for a particular cancer type.

4. Calculation of the 95% Confidence Interval

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

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105–130), there is a statistically significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer cases is statistically significantly lower than expected. If the confidence

interval range includes 100, the true SIR may be 100. In this case, it cannot be determined with certainty that the difference between the observed and expected number of cases reflects a real cancer increase or decrease or is the result of chance. It is important to note that statistical significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

5. *Evaluation of Cancer Risk Factor Information*

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, smoking history, and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to lung and bronchus, kidney, and pancreatic cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several types of cancer. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for residents of the four communities who were diagnosed with any of the six cancer types included in this report. However, information about personal risk factors such as family history, hormonal events, diet, and other factors that may

also influence the development of cancer is not collected by the MCR; therefore, it was not possible to evaluate these factors in this investigation.

6. Determination of Geographic Distribution of Cancer Cases

In addition to calculation of SIRs, address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2002). This allowed assignment of census tract location as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer cases in each of the four communities. In instances where the address information from the MCR was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using telephone books issued within 2 years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. (Note: MDPH is bound by Massachusetts General Laws and the federal Health Insurance Portability and Accountability Act (HIPAA) not to reveal the name or identifying information of an individual diagnosed with cancer whose case is reported to the MCR.)

B. Cancer Incidence in Belmont

Cambridge Plating is located in CT 3572 close to the border of CT 3571 (see Figure 1). This section presents cancer incidence rates for the town of Belmont as a whole as well as Belmont CTs 3571 and 3572, during the 18-year time period, 1982–1999. A summary of the cancer experience in the communities of Cambridge, Arlington, and Watertown is provided in Section C. To evaluate possible trends over time, cancer incidence data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–1999. In addition to the results presented in this section, Appendix A provides comprehensive analyses for the town of Belmont as a whole and all eight census tracts that comprise the town. Cancer incidence results for Belmont and the census tracts that comprise the town are presented in Tables 1a – 6d of Appendix A. SIRs were not calculated for some cancer types in smaller time periods 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.

1. *Results*

a. *Town of Belmont*

With one exception, which is discussed later in this section, the cancer types evaluated in this report generally occurred approximately near or below expected rates in the town of Belmont as a whole during the 18-year time period, 1982–1999, as well as smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–1999).

Overall, kidney cancer, lung and bronchus cancer, and NHL occurred less often than expected during 1982–1999. The incidence of kidney cancer appears to have decreased over time among both male and female residents of Belmont. For example, during 1982–1987, kidney cancer was elevated among males (15 diagnoses observed vs. 9.0 expected) and occurred slightly less than expected among females (6 diagnoses observed vs. 7.0 expected). The elevation among males was not statistically significant. During the most recent time period evaluated, 1994–1999, 13 individuals were diagnosed with kidney cancer compared to about 21 diagnoses expected. Both males and females experienced lower than expected incidence of kidney cancer during this time period.

Lung and bronchus cancer occurred statistically significantly less often than expected in the town as a whole (270 diagnoses observed vs. 375.3 expected, SIR = 72, 95% CI = 64–81) during the 18-year time period, 1982–1999. The incidence of this cancer type was also lower than expected during each of the smaller time periods evaluated and statistically significantly lower than expected during 1982–1987 and 1994–1999.

NHL occurred less often than expected in Belmont during 1982–1999, primarily due to a lower-than-expected rate among males in the town (32 diagnoses observed vs. 44.6 expected, SIR = 72), a rate that was borderline statistically significant (95% CI = 49-101). Males in Belmont experienced fewer diagnoses of NHL than expected during each of the smaller time periods evaluated. Among females, more cases occurred during 1982–1999 than expected (52 diagnoses observed vs. 46.5 expected), but this elevation was not statistically significant.

Females were diagnosed about as expected during 1982–1987 and slightly more often than expected during the most recent time periods (1988–1993 and 1994–1999). During each of these time periods, approximately three excess cases were diagnosed among females; however, neither elevation was statistically significant (19 diagnoses observed vs. 15.3 expected during 1988–1993; 20 diagnoses observed vs. 17.0 expected during 1994–1999).

Pancreatic cancer occurred about as expected during 1982–1999 based on the statewide cancer experience. Although the overall rate remained consistent over time, different trends were observed among males and females when evaluated separately by gender. Specifically, the incidence of pancreatic cancer in Belmont appears to have increased over time among males while decreasing over time among females. However, no difference in either gender was statistically significant.

Sixteen diagnoses of liver cancer were observed in Belmont during 1982–1999 versus about 14 expected. The increase was based on approximately one additional diagnosis each over the expected number among males and females and was not statistically significant. When examined by smaller time period, liver cancer occurred about as expected in all three time periods.

The incidence of leukemia among females was elevated in Belmont during 1982–1999 (32 diagnoses observed vs. 23.2 expected, SIR = 138). This elevation was not statistically significant. Among males, leukemia occurred slightly less often than expected during the 1982–1999 period (26 diagnoses observed vs. 27.1 expected). Although leukemia was diagnosed less often than expected during the earliest time period, 1982–1987, both males and females experienced elevations of this cancer type during 1988–1993 and 1994–1999. None of the observed elevations were statistically significant. Elevations among males were based on one to three excess cases, while the elevations in females were due to about four excess cases in each time period (11 diagnoses observed vs. 6.9 expected during 1988–1993; 13 diagnoses observed vs. 8.7 expected during 1994–1999).

b. Belmont CTs 3571 and 3572

Rates of kidney cancer, lung and bronchus cancer, and NHL were near or below the expected rates among males and females combined in Belmont CT 3571 during the 18-year time period, 1982–1999. Slightly more leukemia, liver cancer, and pancreatic cancer diagnoses were observed during the overall time period; however, none of the elevations was statistically significant. Lung and bronchus cancer occurred statistically significantly less often than expected among males during this time (29 diagnoses observed vs. 46.4 expected, SIR = 63, 95% CI = 42-90).

In general, when cancer rates in CT 3571 were evaluated for smaller time periods, no consistent trends over time were observed. While leukemia occurred at about the rate expected during the first time period, 1982–1987, this cancer type occurred slightly more often than expected during the two later time periods, 1988–1993 and 1994–1999. These increases were based on one to two additional diagnoses during each time period and were not statistically significant. Lung and bronchus cancer occurred statistically significantly less often than expected among males during the first time period, 1982–1987 (7 diagnoses observed vs. 16.1 expected, SIR = 43, 95% CI = 17–90). The incidence of this cancer type among males was also lower than expected during 1988–1993 and 1994–1999. The overall elevation of pancreatic cancer in CT 3571 was primarily due to an elevation during the earliest time period (1982–1987). In general, kidney cancer, liver cancer, and NHL occurred about as expected in CT 3571 during each of the smaller time periods evaluated.

Of the six cancer types evaluated in this report, five (leukemia, liver cancer, lung and bronchus cancer, NHL, and pancreatic cancer) occurred about as or less often than expected in Belmont CT 3572, where Cambridge Plating is located, during the 18-year time period (1982–1999) among males and females combined. Lung and bronchus cancer occurred statistically significantly less often than expected during this time period (31 diagnoses observed vs. 46.2 expected, SIR = 67, 95% CI = 46–95).

Eight individuals were diagnosed with kidney cancer among males and females combined while 7.2 would have been expected. About three excess cases occurred among males (7 diagnoses observed vs. 4.4 expected), while kidney cancer among females occurred less often than

expected (1 diagnosis observed vs. 2.8 expected). The elevation in kidney cancer among males was primarily attributable to an excess of approximately two cases during 1982–1987. During 1994–1999, kidney cancer occurred less often than expected among males in census tract 3572.

When examined by smaller time periods, all four diagnoses of NHL in CT 3572 during 1982–1999 occurred during the earliest time period, 1982–1987 (versus 3.0 expected), with no diagnoses during 1988–1999 compared to almost eight expected. For leukemia, liver cancer, lung and bronchus cancer, and pancreatic cancer, most occurred less frequently or about as expected during each time period for each gender. Any elevations observed were based on about one or two additional cases above the expected. Refer to Tables 1a – 6d in Appendix A for details.

2. Review of Cancer Risk Factor Information in Belmont

As previously mentioned, 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 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 each of the six cancer types evaluated in this report 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 and surrounding communities in this investigation. For detailed information regarding risk factors associated with the cancer types evaluated in this report, please refer to Appendix C.

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. 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 these cancer types was reviewed to assess the role tobacco smoking may have played in the development of these cancers 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 by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these 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.

Finally, histologic (cell type) distribution was reviewed for diagnoses of lung and bronchus cancer and leukemia. Patterns of disease were compared to known or established incidence trends to assess whether any unexpected patterns exist in these areas.

Available information on risk factors was compared to known or established trends to evaluate whether any unexpected patterns exist in Belmont. In general, the trends observed in Belmont are similar to those seen in the general population. Review of these data suggests that smoking likely played some role in the diagnosis of some cancer types (e.g., cancers of the kidney, lung and bronchus, and pancreas) among some individuals. Review of available data indicated that occupational exposures may have been important in the development of cancer among some individuals as well. However, it is difficult to fully assess the extent to which these factors influenced the overall patterns of cancer in Belmont because of the large number of individuals for whom smoking history and/or occupation was unknown. Refer to Appendix A for more

detailed information regarding the prevalence of these risk factors among individuals diagnosed with cancer in Belmont.

3. Analysis of Geographic Distribution of Cancer Incidence in Belmont

In addition to determining incidence rates for each cancer type, a qualitative evaluation of the geographic pattern of cancer diagnoses was conducted for the town of Belmont. 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 report, no apparent concentrations of cancer diagnoses (of any type) were observed in the vicinity of the Cambridge Plating site. A small concentration of leukemia diagnoses was noted in the southeastern corner of Belmont. However, leukemia is a general term that describes a group of four different major subtypes [i.e., acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML)], and a few rare types, such as hairy cell leukemia. Further review of specific case information for these individuals revealed a variety of subtypes of leukemia diagnosed among individuals in this area, indicating the occurrence of different diseases with different risk factors.

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 is greater.

Although elevations in the incidence of some cancer types were noted in Belmont during one or more time periods evaluated, in general, the geographic distribution of diagnoses for these cancer types seemed to coincide closely with the pattern of population and cases did not appear to be concentrated in any one area of the town. 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.

C. Cancer Incidence in Surrounding Communities

With some exceptions, cancer incidence in the surrounding towns of Cambridge, Arlington and Watertown during the 18-year time period, 1982–1999, and smaller time periods evaluated was approximately at or near expected rates for the majority of the six cancer types evaluated. The following sections provide a summary of cancer incidence results for the three surrounding communities. Appendix A of this report provides comprehensive analyses of cancer incidence in the communities of Cambridge, Arlington, and Watertown.

1. Results

a. City of Cambridge

Cancer incidence rates were evaluated for the city of Cambridge, with a particular focus on census tracts adjacent to Belmont (i.e., CTs 3543, 3546, and 3549). The results of these analyses are summarized for each of the six cancer types in Tables 7a – 12d of Appendix A. During the 1982–1999 time period, citywide incidence rates were lower than expected for kidney cancer, leukemia, lung and bronchus cancer, and NHL. Rates were about as expected for pancreatic cancer and slightly higher than expected for liver cancer. Overall, 40 individuals were diagnosed with liver cancer compared to 34.2 expected (SIR = 117). The elevation in liver cancer was not statistically significant.

When evaluated by smaller time periods, kidney cancer, leukemia, lung and bronchus cancer, and NHL occurred consistently less than expected over time. Citywide rates of liver cancer were higher than expected during 1982–1987, about as expected during 1988–1993, and slightly higher than expected during 1994–1999. Neither of the elevations was statistically significant. Analysis of trends over time suggests that the incidence of pancreatic cancer is decreasing in the

city as a whole over time. During the most recent time period evaluated, 1994–1999, 43 individuals were diagnosed with pancreatic cancer in Cambridge compared to 48.0 expected (SIR = 89).

Census tract-specific SIRs could not be calculated for the majority of cancer types because of the small number of observed cases; however, the number of cases observed was compared with the number expected to determine if an atypical pattern of cancer was occurring. Kidney cancer, leukemia, lung and bronchus cancer, and NHL generally occurred equal to or less often than expected in Cambridge CTs 3543, 3546, and 3549 during 1982–1999. Slight elevations in liver cancer were observed in some census tracts, however elevations were based on one to two additional diagnoses over that expected. While pancreatic cancer occurred about as expected in CTs 3543 and 3546 during 1982–1999, an elevation in pancreatic cancer was observed in CT 3549 during this time (13 diagnoses observed vs. 7.0 expected, SIR=187). This elevation was borderline statistically significant (95% CI = 99–320) and was due to a statistically significant elevation among males in this area of Cambridge (9 males diagnosed vs. 3.3 expected, SIR = 273). This rate appears to be the result of slight elevations in the incidence of this cancer type among males during each of the smaller time periods in CT 3549.

b. Town of Arlington

In general, residents of Arlington experienced cancer approximately at or below the rates expected during 1982–1999 and in the three smaller time periods evaluated. Cancer incidence rates for Arlington are presented in Tables 13a – 13d of Appendix A. Kidney cancer, leukemia, and lung and bronchus cancer all occurred less often than expected during 1982–1999. Overall, the incidence of lung and bronchus cancer in Arlington was statistically significantly decreased with respect to the state rate between 1982–1999 (544 diagnoses observed vs. 643.8 expected, SIR = 84, 95% CI = 78–92) and statistically significantly decreased during each of the three smaller time periods evaluated. Both NHL and pancreatic cancer were diagnosed at about the rates expected during 1982–1999 and the incidence of liver cancer was higher than expected during this time (28 diagnoses observed vs. 23.4 expected). Townwide incidence ratios for liver cancer among males and females combined and for pancreatic cancer among males were statistically significantly elevated during the earliest time period evaluated (1982–1987). An

elevation in the incidence of leukemia was also observed among females aged 0–19 years in Arlington.

c. Town of Watertown

With some exceptions, Watertown residents experienced cancer approximately at or near the rates expected during 1982–1999 and during the three smaller time periods evaluated. Cancer incidence rates for the town of Watertown are provided in Appendix A, Tables 14a through 14d. Specifically, the incidence of both lung and bronchus cancer and pancreatic cancer were lower than expected during the time period 1982–1999, and the overall incidence of lung and bronchus cancer may be decreasing over time in Watertown. Slight elevations in kidney cancer and leukemia were observed during the overall time period, however neither elevation was statistically significant. The incidence of liver cancer among males and females combined was higher than expected based on the state rate, and the rate of liver cancer among males alone during 1982–1999 was statistically significantly elevated (21 diagnoses observed vs. 11.2 expected, SIR = 188). Liver cancer was diagnosed more often than expected among males in Watertown during each of the smaller time periods evaluated (1982–1987, 1988–1993, and 1994–1999), however none of these elevations was statistically significant. Finally, NHL occurred more often than expected in the town of Watertown during 1982–1999 (137 diagnoses observed vs. 113.2 expected, SIR = 121), an elevation that was statistically significant (95% CI = 102–143).

2. Review of Risk Factor Information in Surrounding Communities

Available risk factor information for individuals diagnosed with cancer in Cambridge, Arlington, and Watertown was also compared to known or established trends to assess whether any unexpected patterns exist in the town. As with the town of Belmont, the trends observed in the surrounding communities are similar to those seen in the general population. Review of these data, which is provided in Appendix A, suggests that smoking likely played a role in the development of some types of cancer such as cancers of the kidney, lung and bronchus, and pancreas among some individuals in these towns. However, because of the large number of individuals for whom smoking history and/or occupation was unknown, it is difficult to fully

assess the extent to which these factors influenced overall cancer patterns in the surrounding towns.

3. *Analysis of Geographic Distribution of Cancer Incidence in Surrounding Communities*

The place of residence for individuals diagnosed with cancer in Cambridge, Arlington, and Watertown was evaluated to identify any geographic concentrations of cases that might be present within the three communities. 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 the three surrounding towns that would suggest a potential relationship to Cambridge Plating. While portions of Cambridge CTs 3546 and 3549 are located in the vicinity of Cambridge Plating, land use in the western portion of CT 3549 is primarily non-residential. Census tract 3546, however, is a residential neighborhood and is adjacent to the Belmont border. There were no unusual concentrations of cancer diagnoses in either of these census tracts.

A small concentration of leukemia diagnoses was observed near the center of Arlington. Specifically, six individuals (ages 15–77) in a high-density residential neighborhood were diagnosed with leukemia between 1994 and 1998. However, a variety of histology types were represented among these individuals—indicating the occurrence of different diseases. While an elevation in the incidence of leukemia was observed among females aged 0–19 years in Arlington (9 diagnoses observed vs. 2.6 expected), in general, the nine diagnoses did not appear to be unusually concentrated in time or space. A small concentration of individuals diagnosed with NHL was also noted in Watertown. On the basis of the available risk factor information for these individuals, including a review of residential histories, it does not appear that any single factor (environmental or nonenvironmental) would explain the observed distribution. A complete discussion of geographic distribution of cancer incidence in the surrounding communities is provided in Appendix A, Section VII.

VII. DISCUSSION

As part of this public health assessment, MDPH evaluated cancer incidence data for Belmont and the surrounding communities of Cambridge, Arlington, and Watertown, with a particular focus on Belmont CTs 3571 and 3572, and reviewed available environmental information for Cambridge Plating to determine possible pathways of exposure for nearby residents. Although some of the soil and groundwater at the site has been contaminated in the past, and is still contaminated in recent samples, the information reviewed does not suggest that under present conditions the contaminants detected in subsurface media at the Cambridge Plating facility are likely to result in health concerns to individuals visiting or residing in neighborhoods adjacent to the facility.

Most of the Cambridge Plating property is covered by asphalt or concrete and it is unlikely that residents who live nearby would come into contact with contaminated soil from the site. Past intermittent exposure to chromium-contaminated soil could have been possible for individuals who might have trespassed near a 450-square foot area located north of the facility adjacent to the MBTA railroad tracks. However, it is unlikely that anyone would have had contact with soil from this area for sufficient frequency and duration of time to result in health effects. It is also important to note that while subsurface soil concentrations of total chromium were quite high in this location, levels of hexavalent chromium (a carcinogen) were approximately at or below health-based comparison values. In addition, new information indicates that surface soil concentrations of chromium are approximately at or below health-based comparison values and, therefore, unlikely to result in adverse health effects for residents who may be exposed to on-site surface soil.

In the Cambridge Plating Public Health Assessment report that was released for public comment in 2005, the MDPH recommended additional sampling to determine chromium levels in soil surrounding Cambridge Plating. Air emissions modeling conducted by MDEP determined that areas with the highest chromium concentrations would likely be located on-site. To confirm the results of the air emissions modeling and to address the recommendation for additional sampling, MDEP sampled surface soil at off-site locations near the facility in 2007 (MDEP 2007). This sampling event confirmed the air emissions modeling and indicated that surface soil

concentrations of chromium are well below health-based comparison values and, therefore, unlikely to result in adverse health effects for residents living near Cambridge Plating.

TCE and its chlorinated breakdown products (i.e., cis-1,1-DCE and vinyl chloride, which are also VOC compounds) were detected in groundwater samples collected at the Cambridge Plating site. Sampling of groundwater monitoring wells west of the Cambridge Plating buildings showed the highest concentrations of TCE and cis-1,1-DCE. Although these chlorinated VOCs were detected at levels exceeding health-based comparison values for drinking water (see Table 2), exposure to contaminated groundwater is unlikely for individuals residing in neighborhoods abutting the Cambridge Plating site because groundwater in this area is not being used for drinking water purposes. In addition, the application of the Johnson-Ettinger model by ATSDR indicates that concentrations of VOCs detected in a groundwater well located closer to the site boundary near Hittinger Street are not at levels that would be expected to result in indoor air concentrations in nearby homes that would be of health concern. This finding is further supported by results of a soil gas and groundwater investigation conducted by Coler and Colantonio, which indicated that the high concentrations of VOCs detected in groundwater west of the Cambridge Plating building are not likely to result in elevated levels in indoor air of homes along Hittinger Street (2004). Also, because groundwater from beneath the Cambridge Plating building does not appear to flow toward the northern site boundary, it is not expected that residents north of the site would have opportunities for exposure to levels of VOCs in indoor air that could present a health risk.

A limitation on future uses of the Cambridge Plating property will reduce the possibility of any future exposures to subsurface contamination present at the site. As a result of site investigations conducted in 2000, Cambridge Plating filed a Notice of Activity and Use Limitation for the property stating it cannot be developed for residential use without subsurface investigation and, if necessary, remediation (Coler and Colantonio 2002a).

Review of air permit information and TRI data for Cambridge Plating indicate that TCE and chromium emissions to air are primary contaminants of concern in the past at Cambridge Plating. Fugitive (non-stack) air emissions of TCE have been reported in TRI data since 1988 (Table 3); however, TCE is no longer being used at the facility and air emissions of TCE have been

eliminated. Stack emissions of chromium were also emitted to the air during the operational history of Cambridge Plating, until chrome-plating operations ceased at the end of 2002 (EPA Region 1 2003b). There are no ambient air sampling data available to determine whether past air emissions of TCE and chromium from the facility resulted in air concentrations in the adjacent neighborhoods at levels sufficient to result in exposure and/or health effects to residents. In addition, because limited surface soil sampling data were available, it was not possible to evaluate potential exposures associated with deposition of chromium air emissions in the past.

Occupational studies of workers exposed to unmeasured levels of TCE in air have been unable to provide definitive evidence for an increased cancer risk and are often limited by multiple chemical exposures and small numbers of study participants. While some studies have shown no association between inhalation exposure to TCE and cancer, others have found slight increases in a number of cancer types. However, problems with study design were often reported, such as the inability to distinguish between the effects of TCE and other chemicals used in the same area as TCE, and associations were often based on small numbers of individuals and complicated by confounding factors (ATSDR 1997).

As observed in several toxicological investigations in experimental mice and rats, TCE has been shown to cause liver, lung, and kidney cancer and, to a lesser extent, lymphomas and leukemia. In terms of non-cancer health outcomes associated with TCE, studies of workers chronically exposed to TCE in air have reported sleepiness, dizziness, headaches, and nausea, and there is some suggestion that people who breathe high levels of TCE may develop damage to the nerves in the face (ATSDR 1997).

The toxicological and epidemiological literature on chromium shows that inhalation exposure to chromium may have health effects, depending on the specific form of chromium compound involved and the nature of exposure (length of time, concentration). Both human epidemiological and animal studies show that chromium, in particular hexavalent chromium, is a carcinogen. In particular, many studies show that occupational exposure to chromium VI can be associated with an increased risk of respiratory cancer (ATSDR 2000a). A range of respiratory symptoms, gastrointestinal effects and irritations were seen in workers exposed to chromium

(VI) compounds, although in many of the studies there were confounding factors (ATSDR 2000a).

Most of the types of cancer evaluated in this report were selected based on their potential association with contaminants of concern identified or historically emitted at Cambridge Plating (i.e., TCE and chromium). However, in large part, the rates of those cancer types evaluated in the community surrounding the facility (i.e., Belmont CTs 3571 and 3572) were approximately near or below the rates expected based on cancer incidence in the state of Massachusetts as a whole for the 18-year time period, 1982–1999. Similar trends were observed in the town of Belmont as a whole and in Cambridge, Arlington, and Watertown. Specifically, with a few exceptions, cancer incidence in the four towns during the 18-year time period, 1982–1999, and the three smaller time periods evaluated was approximately at or near expected rates for the six cancer types evaluated in this report. Further, available risk factor information for those diagnosed with cancer was compared to known or established trends to assess whether any unexpected patterns exist in Belmont, Cambridge, Arlington, or Watertown. In general, cancer trends observed in these communities are similar to those seen in the general population. Review of the data suggests that smoking likely played some role in the development of certain types of cancer (e.g., kidney cancer, lung and bronchus cancer, and pancreatic cancer) among some individuals. Also, occupational exposures may have been important in the development of certain cancers, such as leukemia, lung and bronchus cancer, and NHL, among some individuals. However, because of the large number of individuals for whom smoking history and/or occupation was unknown, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in Belmont or the surrounding communities.

In addition, 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. That is, no apparent concentrations of individuals diagnosed with cancer (including 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 that might suggest an association with a common environmental factor.

Residents of Belmont living near the Cambridge Plating facility have also conveyed concerns about several non-cancer health outcomes including upper respiratory irritation, nausea, and headaches (Belmont Department of Health 2003a; 2003c). In addition, complaints about odors have been reported by individuals residing near the facility. More recently, the Belmont Department of Health indicated that odors have subsided and that complaints have decreased (Belmont Department of Health 2007). Of the types of chemicals reported in the TRI data as air emissions from the Cambridge Plating site, hydrofluoric and nitric acids have strong acidic, irritating odors, while TCE has sweet, chloroform-like odors (HSDB 2002a-c). It is possible that some residents living in close proximity to the facility could have experienced some irritant effects associated with TCE (in the past) or acids in ambient air. It is also important to note that some individuals, particularly those with pre-existing conditions such as asthma and allergies, may experience irritant reactions that would not necessarily impact the general population similarly.

On the basis of the information reviewed in this evaluation, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in the census tract that contains Cambridge Plating or the adjacent census tracts, in the town of Belmont as a whole, or in the surrounding communities of Cambridge, Arlington, and Watertown during the 18-year time period, 1982–1999.

VIII. CHILD HEALTH CONSIDERATIONS

ATSDR and MDPH recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at a greater risk than adults from certain kinds of exposure to hazardous substances emitted from waste sites or other environmental sources. They are more likely to be exposed because they play outdoors and because they often bring food into contaminated areas. Because of their smaller stature, they might breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of contaminant exposure per body weight. The developing body systems of children can sustain permanent damage if certain toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

The incidence and patterns of cancer among children in Belmont, Cambridge, Arlington, and Watertown are discussed in detail in Appendix A ("Assessment of Cancer Incidence in Belmont, Massachusetts and Surrounding Communities: 1982-1999") and summarized in Section VI of this report. As previously mentioned, an elevation in the incidence of leukemia was observed among females aged 0–19 years in Arlington. However, a review of the geographic distribution of diagnoses and available environmental information indicate that it is unlikely that a common factor played a role in the occurrence of leukemia among these individuals. There were reports in the Belmont Department of Health files indicating that older children may have trespassed on the Cambridge Plating site in the past—specifically one incidence of youth climbing onto the roof of a building. However, since the contamination identified in soil and groundwater is generally located below the ground surface, children or young adults trespassing at the site would be extremely unlikely to come into contact with these contaminated media. While trespassers might have come in contact with chromium contaminated soil identified in an area adjacent to the MBTA railroad tracks in the past, it is unlikely that exposure would have occurred for sufficient frequency and duration of time to result in health effects. Based on the information reviewed in this evaluation, no evidence was found that would indicate children are more likely than adults to come in contact with contamination identified at Cambridge Plating.

IX. LIMITATIONS

Several important limitations exist in relation to the environmental data available for this report. These limitations make it impossible to determine the role potential exposures to specific contaminants or to environmental media harboring those contaminants may have played in the development of an individual's cancer or other health impact.

The environmental data available from the Cambridge Plating site had several limitations. The total number of samples collected over the entire history of the site was limited, and the sampling was conducted for a variety of specific purposes unrelated to the current evaluation. Some samples were collected as part of the response to a spill or release whereas other samples were collected as part of state and federal site investigations. For some of the environmental data reviewed, there was no information regarding specific depths or sampling locations. Finally, with the exception of samples collected for chromium analysis in soil, no surface soil samples

were collected from the top 0-3 inches of soil where possible exposure would be considered more likely.

This assessment includes an investigation that considers descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers is unusual. The purpose of this investigation was to evaluate the patterns of cancer in a geographical context in relation to available information about factors, including environmental factors, related to cancer to see whether further investigation seems warranted. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further public health actions may be warranted. Inherent limitations in this type of analysis and the available data make it impossible to determine the precise causal relationships or synergistic roles that may have played a part in the development of individual cancers in these communities. Also, this type of analysis cannot determine what may have caused cancer in any one individual. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the disease that could not be evaluated in this report. It is believed that many types of cancers are related largely to behavioral factors such as cigarette smoking, diet, and alcohol consumption. Other factors associated with cancer are socioeconomic status, heredity/genetics, race, and geography. It is beyond the scope of this report to determine the causal relationship of these factors and the development of cancer or other health outcomes in Belmont and the surrounding communities.

X. CONCLUSIONS

- Although some of the groundwater and soil at the Cambridge Plating site has been contaminated in the past and some has been found to be contaminated in more recent samples, based on the data reviewed, there is no information to suggest that the contaminants detected in subsurface media at the Cambridge Plating facility are resulting in health impacts for individuals visiting or residing in neighborhoods adjacent to the facility (i.e., the majority of the property is paved and contaminated groundwater is not a source of drinking water or expected to be at a level of concern for indoor air close to the site boundary near Hittinger Street or Channing Road).

- It has been reported that air emissions of TCE and chromium from Cambridge Plating have occurred in the past. However, there are no ambient air sampling data available to determine whether historical air emissions resulted in air concentrations of these chemicals in the vicinity of the facility at levels sufficient to result in exposure and/or health impacts to residents.

- Air modeling conducted by MDEP predicted that the areas with the highest concentrations of chromium in soil would likely be located on-site. Additional analysis of surface soils, conducted by Cambridge Plating at the request of MDEP, indicated that exposure to surface soil concentrations of chromium located on-site would not be expected to result in adverse health effects for nearby residents. Sampling for chromium at locations around the Cambridge Plating facility also indicated that chromium in off-site surface soil is unlikely to result in adverse health effects in nearby residents.

- In general, the six 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 18-year time period, 1982–1999. Similar trends were observed in the surrounding communities of Cambridge, Arlington, and Watertown. 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.

- It is possible that residents living in proximity to Cambridge Plating may have experienced some irritant effects associated with air emissions from the facility. It is important to note that some individuals, particularly those with pre-existing conditions such as asthma and allergies, may experience irritant reactions that would not necessarily impact the general population similarly.

- On the basis of the information reviewed in this evaluation, including available environmental data for Cambridge Plating and risk factor information for individuals diagnosed with cancer, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in the census tract that

contains the Cambridge Plating site or the adjacent census tract, in the town of Belmont as a whole, or in Cambridge, Arlington, or Watertown during the 18-year time period, 1982–1999.

ATSDR requires that one of five conclusion categories be used to summarize findings of a public health assessment. These categories are as follows: (1) Urgent Public Health Hazard; (2) Public Health Hazard; (3) Indeterminate Public Health Hazard; (4) No Apparent Public Health Hazard; (5) No Public Health Hazard. A category is selected from site-specific conditions such as the degree of public health hazard based on the presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns.

Although no indication was found that people are being exposed to chemicals from Cambridge Plating, past anecdotal reports about odors and nuisance conditions as well as documented evidence of fugitive TCE air emissions and stack chromium emissions suggest that past exposures may have been possible in areas surrounding the facility. However, information regarding historical ambient air concentrations of emissions is unavailable making it difficult to evaluate whether the local population has been exposed to contaminants from the site. Therefore, using ATSDR's criteria, under past conditions Cambridge Plating would be classified as posing an Indeterminate Public Health Hazard. Available information does not indicate that possible past exposures have resulted in elevated cancer rates in Belmont CTs 3571 or 3572 or in the neighborhoods surrounding the facility.

In the Cambridge Plating Public Health Assessment released for public comment in 2005, current and future opportunities for exposures were classified as an Indeterminate Public Health Hazard due to data gaps related to groundwater and surface soil. Since then, additional surface soil sampling was conducted to evaluate whether air emissions in the past resulted in deposition of chromium in surface soil at a level of health concern. The results indicated that surface soil concentrations of chromium located both on-site and off-site are unlikely to result in adverse health effects for residents who may be exposed. New groundwater monitoring data and additional groundwater characterization indicated that groundwater from beneath the Cambridge Plating facility does not appear to flow toward the northern site boundary; therefore, it is not

expected that residents north of the site would have opportunities for exposure to levels of VOCs in indoor air that could present a health risk. Based on the previously available data and the additional data gathered since the public comment release in 2005, ATSDR would classify current and future opportunities for exposures as presenting No Apparent Public Health Hazard. MDPH continues to support the efforts of Belmont town officials, state and federal environmental agencies, and Cambridge Plating to reduce any potential nuisance impacts to the surrounding neighborhoods.

XI. RECOMMENDATIONS

- Future characterization of groundwater conditions (e.g., chemical concentrations) and flow patterns should be conducted in accordance with MDEP regulations, the MCP 310 CMR 40.000, in the event of any planned construction/demolition or alternative development of the Cambridge Plating property to ensure adequate protection of public health. On the basis of the subsurface contamination identified at Cambridge Plating (e.g., elevated levels of TCE detected in monitoring wells located close to the Cambridge Plating building) and potential exposures related to alternative future land uses on the property, the MDPH supports the Notice of Activity and Use Limitation registered with the Middlesex County Land Court by the company in April 2003.

- If additional environmental data on historical air emissions or ambient air quality related to Cambridge Plating become available, MDPH should further characterize the potential for past exposure upon request of the Belmont Health Department.

- Due to the unpleasant odors and nuisance conditions reported in the past by residents living near the facility, MDPH supports continued collaboration between state and federal regulatory agencies, the Belmont Department of Health, and representatives of Cambridge Plating to determine any additional actions that would help ensure that odor and nuisance conditions do not return in the future.

XII. PUBLIC HEALTH ACTION PLAN

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

- Should new environmental data be generated for the Cambridge Plating site, particularly if site use will change, or if additional data on VOCs in groundwater or historical ambient air quality data become available, the MDPH will further characterize opportunities for exposure upon request of the Belmont Department of Health.

- The MDPH will continue to monitor the incidence of all cancer types in the towns of Belmont, Cambridge, Arlington, and Watertown through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

XIII. REFERENCES

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PREPARER

This document was prepared by the Bureau of Environmental Health of the Massachusetts Department of Public Health (MDPH). If you have any questions about this document, please contact Suzanne K. Condon, Director of the MDPH Bureau of Environmental Health, at 250 Washington Street, 7th Floor, Boston, Massachusetts 02108.

CERTIFICATION

The Public Health Assessment *Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982-1999,* was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Public Health Assessment was initiated.

Technical Project Officer, CAT, SPAB, DHAC, ATSDR

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

Team Lead, CAT SPAB, DHAC, ATSDR

FIGURES

Figure 1
Area of Analysis
Arlington, Belmont, Cambridge, and Watertown, MA

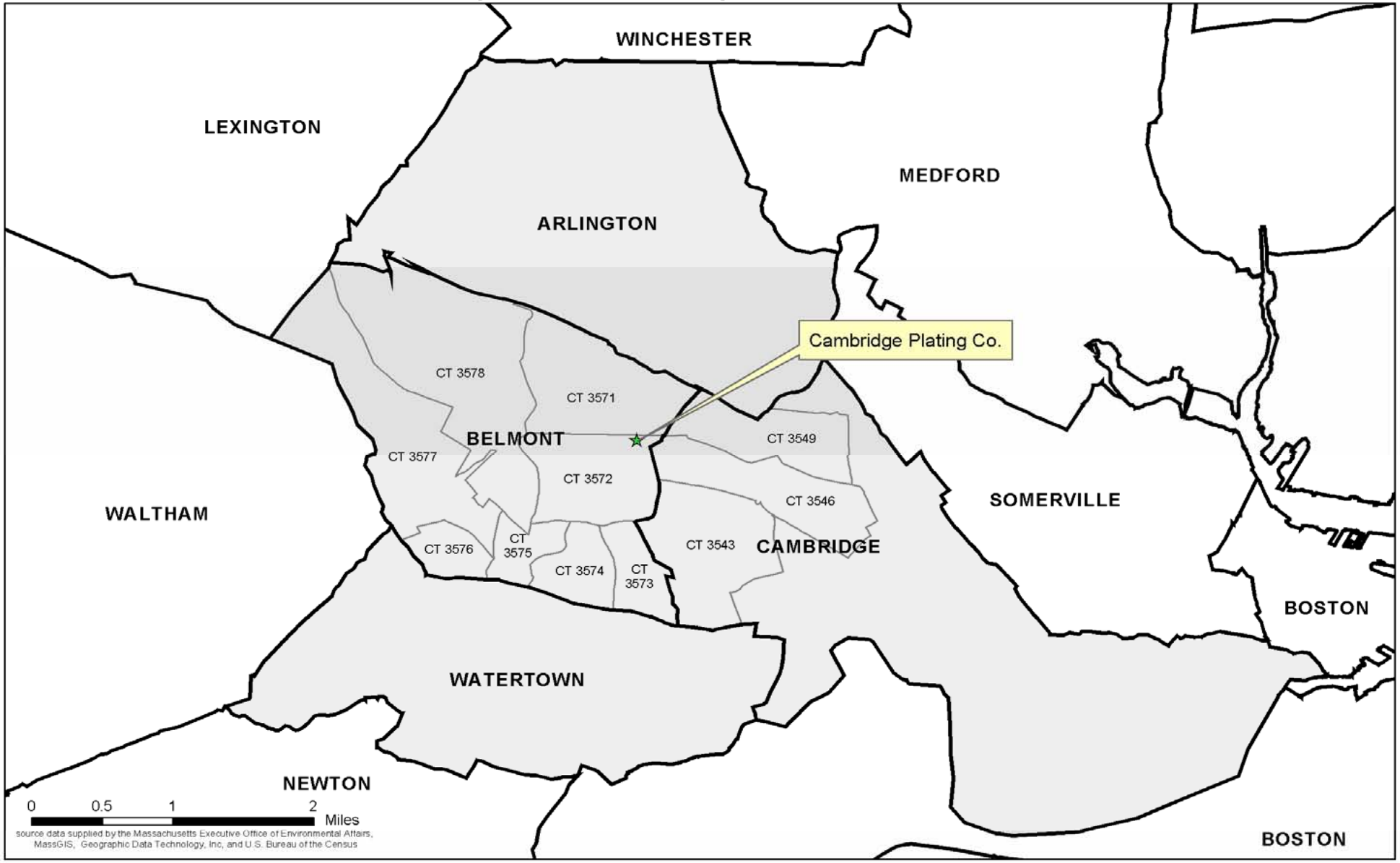


Figure 2
Location of Cambridge Plating
Belmont, Massachusetts

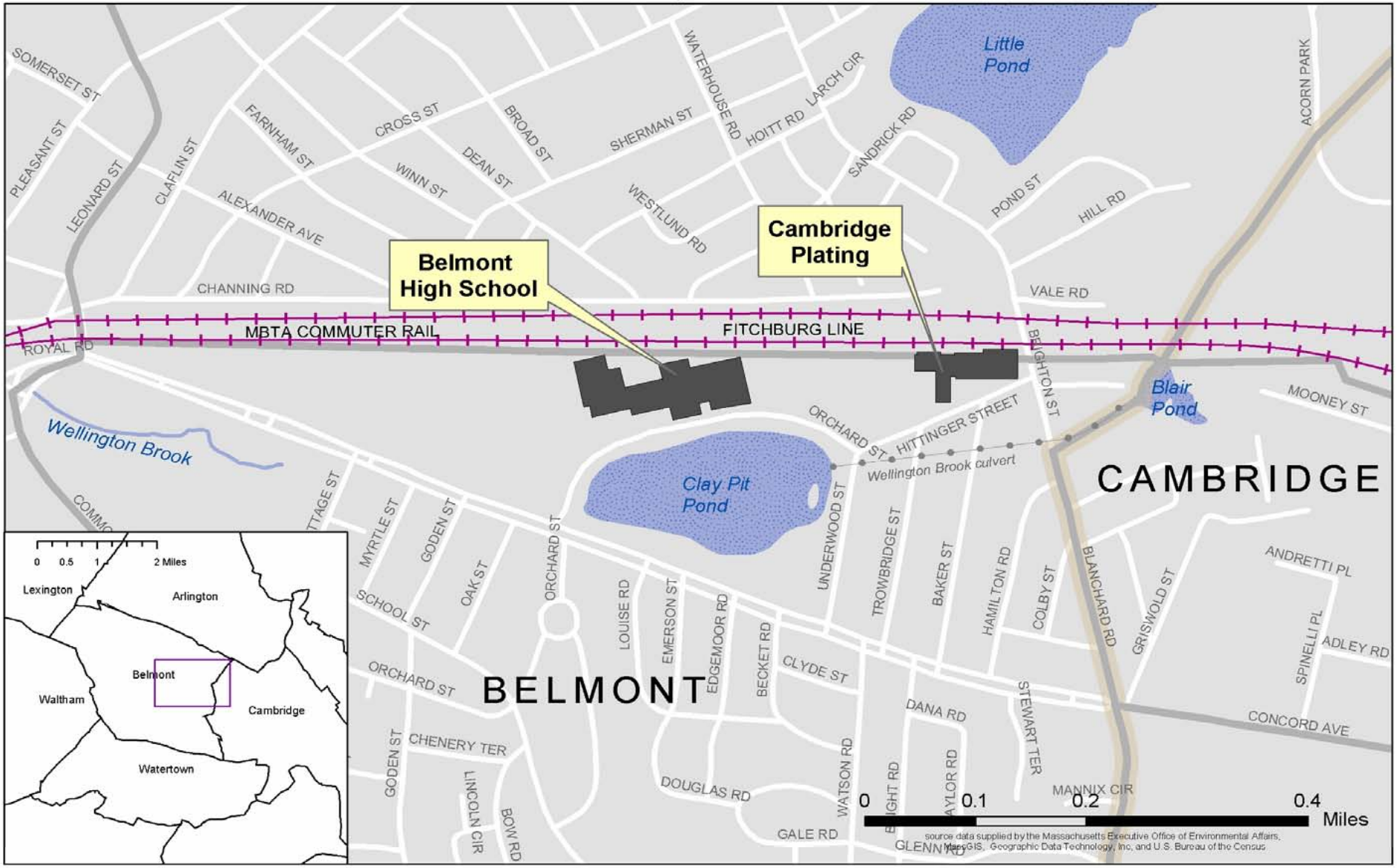
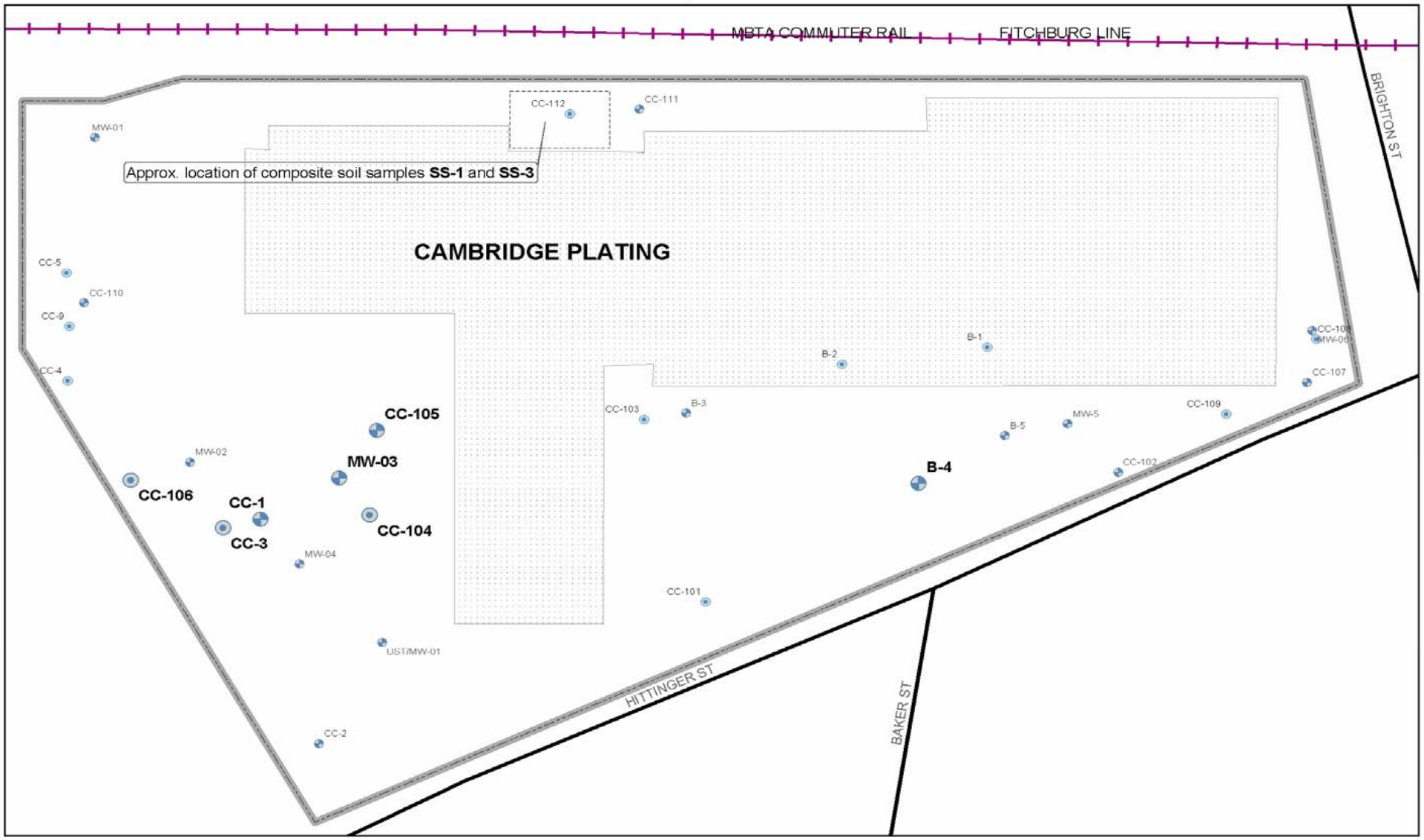


Figure 3
Approximate Locations of Monitoring Wells and Soil Borings
Cambridge Plating
Belmont, Massachusetts



0.01 0.005 0 0.01 0.02 Miles

source data supplied by the Massachusetts Executive Office of Environmental Affairs, MassGIS, Geographic Data Technology, Inc., and U.S. Bureau of the Census

Bureau of Environmental BEHA Health Assessment

Legend

- Monitoring Well with Maximum Level
- Soil Boring with Maximum Level
- Monitoring Well
- Soil Boring
- Commuter Rail
- Street
- Property Line
- Restricted Area

Figure 4
Approximate Locations of Off-Site Soil Samples
Belmont, Massachusetts

Bureau of BEH Environmental Health

Geographic data supplied by: Massachusetts Executive Office of Environmental Affairs, MassGIS; Geographic Data Technology, Inc.

N
W
E
S

0 0.02 0.04 0.08 Miles

Coordinate System: Massachusetts Mainland State Plane Meters (NAD83)

TABLES

Table 1
Maximum concentrations of contaminants that exceeded comparison values in soil samples from the Cambridge Plating Site (samples taken from 1994–2003)

Contaminant	Sample depth (feet)	Date of sample	Descriptive location of sample*	Maximum concentration (ppm)	Comparison value (ppm)	Background soil levels (ppm)
Arsenic	not specified	6/1/1994	Soil pile resulting from excavation activities associated with contruction of loading dock	44‡	CREG = 0.5 Chronic EMEG (child), RMEG (child) = 20 Chronic EMEG (adult), RMEG (adult) = 200	7.4† (range: <0.1 - 73)
Benzo(a)anthracene	5-7	12/15/1997	CC-1 - west of the Cambridge Plating building	10	EPA RBC = 0.87	0.005 - 0.02 (rural soil) 0.169 - 59 (urban soil)
Benzo(a)pyrene	5-7	12/15/1997	CC-1	11	CREG = 0.1	0.002 - 1.3 (rural soil) 0.165 - 0.22 (urban soil)
Benzo(b)fluoranthene	5-7	12/15/1997	CC-1	11	EPA RBC = 0.87	0.02 - 0.03 (rural soil) 15 - 62 (urban soil)
c11-c22 aromatics	1-3	7/7/1998	B-4 - east of the Cambridge Plating building	340	S-1 & GW-1 MDEP standard = 200	-
Chromium (total)	3-5	5/13/2003	SS-3 (composite) - north of Cambridge Plating building	32,000	RMEG (child) for Cr VI = 200 RMEG (adult) for Cr VI = 2,000	52†
Chromium (VI)	3-5	5/13/2003	SS-1 (composite) - north of Cambridge Plating building	210	RMEG (child) = 200 RMEG (adult) = 2,000	-
Chrysene	5-7	12/15/1997	CC-1	10	EPA RBC = 87	0.0383 (rural soil) 0.251 - 0.64 (urban soil)
Dibenzo(a,h)anthracene	5-7	12/15/1997	CC-1	1	EPA RBC = 0.087	-
Indeno(1,2,3-cd)Pyrene	5-7	12/15/1997	CC-1	5.8	EPA RBC = 0.87	0.01 - 0.015 (rural soil) 8.0 - 61 (urban soil)
Trichloroethylene	4-8	10/6/2000	CC-106 -west of the Cambridge Plating building	8.5	EPA RBC = 58	-

Table 1 (continued)

*For sample locations, see Figure 3

‡ This value was from soil taken from the excavation of a loading dock. The next highest value for arsenic, 7.6 ppm, still exceeds the CREG

†Estimated arithmetic mean for the Eastern United States (east of 96th meridian). Cited in ATSDR 1993. ATSDR Public Health Assessment Guidance Manual. Atlanta: U.S. Department of Health and Human Services.

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Unless otherwise noted, soil background concentrations are from ATSDR Toxicological Profiles 2000 (on CD-ROM), ATSDR 2000.

Comparison values (source organization, reference)

CREG = Cancer risk evaluation guide for 1 x 10-6 excess cancer risk (ATSDR, ATSDR 2003b)

Chronic EMEG (adult/child) = Environmental media evaluation guide (i.e., for adult or childhood exposures mirroring greater than 1 year) (ATSDR, ATSDR 2003b)

RMEG (adult/child) = Reference dose media evaluation guide (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure). (ATSDR, ATSDR 2003b)

EPA RBC = EPA Region 3 risk-based concentration for soil (September 2001)

Intermediate EMEG (pica child) = Environmental media evaluation guide for children who consume very high quantities of soil (i.e., for exposures between 14 days and 1 year, and considers vulnerabilities of children who consume high quantities of soil when it comes to environmental exposures) (ATSDR, ATSDR 2003b)

Intermediate EMEG (child) = Environmental media evaluation guide for children (i.e., for exposures between 14 days and 1 year and considers vulnerabilities of children when it comes to environmental exposures). (ATSDR, ATSDR 2003b)

Intermediate EMEG (adult) = Environmental media evaluation guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, ATSDR 2003b).

S-1 & GW-1 = MCP Method 1 soil category S-1 standards [310 CMR 40.0975(6)(a)] (MDEP, MDEP 2003b)

Table 2
Maximum concentrations of contaminants that exceeded comparison values in samples of groundwater from the Cambridge Plating site (samples taken from 1996–2005)

Contaminant	Date of maximum sample	Descriptive location of sample*	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Arsenic	7/12/1996	MW-3 - west of the Cambridge Plating building (Fig. 3)	37	CREG = 0.02 Chronic EMEG (child) RMEG (child) = 3 Chronic EMEG (adult), RMEG (adult), EPA MCL = 10
Benzo(a)anthracene	1/8/1998	CC-1 - west of the Cambridge Plating building	1	EPA RBC = 0.092
Benzo(a)pyrene	1/8/1998	CC-1	1.3	CREG = 0.005 EPA RBC = 0.0092 MCLG = 0 MDEP MMCL, MCL = 0.2
Benzo(b)fluoranthene	1/8/1998	CC-1	1.5	EPA RBC = 0.092
c11-c22 aromatics	7/14/1998	CC-3 - west of Cambridge Plating Building near to property line	1,800	MDEP GW-1 = 200
1,2-Dichloroethene (total)	7/12/1996	MW-3	5,100	EPA RBC = 55 MCL; MCLG; LTHA for cis isomer = 70 MCL; MCLG; LTHA for trans isomer = 100
1,2-Dichloroethene, cis-	1/27/2004	MW-3	8,150	Intermediate EMEG (child) = 3,000 Intermediate EMEG (adult) = 10,000 EPA RBC = 61 MCL; MCLG; LTHA = 70
Indeno(1,2,3-cd)Pyrene	1/8/1998	CC-1	1	EPA RBC = 0.092
Trichloroethylene	1/27/2004	MW-3	49,800	EPA RBC = 1.6 MCLG = 0 MDEP MMCL, MCL = 5

Table 2 (continued)

Vinyl Chloride	9/8/2005	MW-3	1,200	CREG = 0.03 Chronic EMEG (child) = 0.2 Chronic EMEG (adult) = 0.7 RMEG (child) = 30 RMEG (adult) = 100 MCLG = 0 MDEP MMCL, MCL = 2

*For sample locations see Figure 3

Data sources

Coler and Colantonio, Inc. 2004. Letter from Coler and Colantonio responding to November 12, 2003, MADEP Interim Deadline. RTN 3-2151. February 23, 2004.

Coler and Colantonio, Inc. 2003a. Letter from Coler and Colantonio to MDEP NERO - attaching results of groundwater sampling for November and December 2002 and Feb 2003. February 20, 2003.

Coler and Colantonio, Inc. 2002a. Phase II - Comprehensive Site Assessment and Class A-3 Response Action Outcome Statement Pursuant to the Massachusetts Contingency Plan 310 CMR 40.0830 and 40.0850 for 39 Hittinger Street, Belmont, Massachusetts.

Coler and Colantonio, Inc. 1998. RAO documents for RTN #3-16600. April, June, July, 1998.

Comparison values (source organization, reference)

CREG = Cancer risk evaluation guide for 1 x 10-6 excess cancer risk (ATSDR, ATSDR 2003a)

Chronic EMEG (adult/child) = Environmental media evaluation guide (i.e., for adult or childhood exposures of more than 1 year) (ATSDR 2003a)

RMEGs (adult/child) = Reference dose media evaluation guides (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure) (ATSDR 2003a)

EPA RBC = EPA Region 3 risk-based concentration for tap water (September 2001)

MMCL = Massachusetts maxiumum contaminant level (MDEP, MDEP 2001)

MCL = Maximum contaminant level for drinking water (EPA, ATSDR 2003a)

MDEP GW-1 = MCP Method 1 groundwater standards applicable in areas where the groundwater is considered to be category GW-2 per 310 CMR 40.0932 [310 CMR 40.0974 (2)] (MDEP, MDEP 2003)

LTHA = Lifetime health advisory for drinking water (EPA, ATSDR 2003a)

Intermediate EMEG (adult) = Environmental media evaluation guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, ATSDR 2003a)

Intermediate EMEG (child) = Environmental media evaluation guide for children (i.e., for exposures between 14 days and 1 year; considers vulnerabilities of children to environmental exposures) (ATSDR, ATSDR 2003a)

Table 3
Toxics Release Inventory data for Cambridge Plating (1987–2005)

Chemical name	Media	Unit Of Measurement	Year																		
			2005	2004	2003	2002	2001	2000	1999	1998	1997	1996	1995	1994	1993	1992	1991	1990	1989	1988	1987
Cyanide compounds	AIR FUG*	Pound	NR[†]	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	250
Cyanide compounds	SURF IMP[‡]	Pound	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	72
Hydrochloric acid[§]	AIR FUG	Pound	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	250	458	NR	750	7,100	7,300	6,000	6,400	6,400
Nickel compounds	AIR FUG	Pound	NR	NR	NR	NR	NR	NR	NR	NR	5	NR	NR	316	NR	255	NR	5	NR	NR	NR
Nitric acid	AIR FUG	Pound	250	250	250	250	250	250	250	250	250	45	250	694	NR	982	NR	650	1,000	1,000	1,000
Sodium hydroxide (solution)	AIR FUG	Pound	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	3,500	3,500
Sulfuric acid[§]	AIR FUG	Pound	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	754	NR	779	NR	NR	NR	NR	NR
Trichloroethylene	AIR FUG	Pound	NR	NR	11,638	13,585	9,536	15,260	15,207	15,285	23,900	58,500	14,854	18,580	NR	18,640	18,480	52,200	66,960	47,360	NR
Trichloroethylene	DISP NON METALS	Pound	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	45,400	66,960	NR	NR

*AIR FUG = fugitive or nonpoint air emissions

[†]NR = not reported

[‡]SURF IMP = on-site surface impoundment disposal

[§]1995 and after "acid aerosols" only

Data source: Toxics Release Inventory (TRI) US EPA, 2005.

Table 4
Summary of Possible Exposure Pathways for
Cambridge Plating
Belmont, Massachusetts

Environmental Medium	Exposure Pathway	Contaminant(s)	Point of Exposure	Route of Exposure	Receptor Populations	Time Frame	Type of Pathway	Notes
Soil	Soil/dust (near MBTA Tracks)	Chromium	On-site	Incidental Ingestion/ Dermal contact/ Inhalation	Trespassers	Past	Potential	Contaminated area currently enclosed by fence. Estimated past exposures to surface soil unlikely to result in health effects.
	Subsurface contamination	Metals, VOCs, PAHs	On-site	Dermal contact	Trespassers/ Workers	Past, Present, Future	Eliminated	Contamination identified below ground surface and majority of site is paved.
Groundwater	Groundwater contamination	Metals, VOCs, PAHs	Off-site	Ingestion	Residents	Past, Present, Future	Eliminated	Groundwater is not a source of drinking water.
	Volatilization of shallow groundwater to indoor Air	VOCs (e.g. TCE)	Nearby residences	Inhalation	Residents	Past, Present, Future	Potential	Concentrations detected in down-gradient groundwater samples close to property boundary are below levels of health concern for indoor air.
Ambient Air	Stack and non-stack facility emissions	TCE, Chromium	Nearby residences	Inhalation	Residents	Past	Potential	Historical ambient air concentrations are unknown.

Appendix A

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

1982–1999

Massachusetts
Department
Of
Public Health

Assessment of Cancer Incidence in Belmont, Massachusetts, and Surrounding Communities: 1982–1999

May 2005

Bureau of
Environmental Health,
Community Assessment
Program

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I. BACKGROUND AND STATEMENT OF ISSUES

In response to a legislative directive and to requests from community residents and the Belmont Department of Health, the Community Assessment Program (CAP) in the Bureau of Environmental Health (BEH) of the Massachusetts Department of Public Health (MDPH), conducted an evaluation of cancer incidence in Belmont, Massachusetts, and the surrounding communities of Arlington, Watertown, and parts of Cambridge, Massachusetts. This evaluation was initiated because of community concerns about cancer and possible environmental exposures in relation to an electroplating facility in Belmont, the Cambridge Plating Company.

II. OBJECTIVES

This investigation reviews the pattern of the incidence of six cancer types in Belmont and adjacent communities and compares that incidence with the incidence of these types of cancer in the state of Massachusetts as a whole. In addition, available information about risk factors, including environmental factors, related to the development of cancer was evaluated.

This report provides a descriptive evaluation of the occurrence of cancer for the years 1982–1999 in the town of Belmont as a whole and its individual census tracts, in the city of Cambridge as a whole and selected census tracts in Cambridge, and in the towns of Arlington and Watertown. This time period was chosen because it is the time period for which the most recent and complete cancer incidence data were available from the Massachusetts Cancer Registry (MCR) when this analysis was initiated. The locations of these towns are shown in Figure 1.

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, may help determine whether a common cause or etiology is possible, and can serve to identify areas where further public health investigations or actions may be warranted. Descriptive analyses may also indicate that an excess of known risk factors associated with a disease, such as environmental exposures, exists in a certain geographic area. This descriptive analysis of cancer incidence data cannot be used to establish a causal link between a particular risk factor (either environmental or nonenvironmental) and the development of cancer. In addition, this analysis cannot determine the cause of cancer for any one individual.

The purpose of this evaluation is to report the findings of the patterns of cancer in Belmont and surrounding communities and discuss them in the context of the available environmental information to determine whether recommendations for further public health action are needed. The specific objectives of this investigation were as follows:

- To evaluate the incidence of six cancer types in Belmont at the census tract level and the whole town level, in adjacent census tracts in Cambridge, and in the towns of Watertown and Arlington. The census tract-specific analyses help in understanding whether the incidence of cancer observed townwide might be explained by an increase or decrease in the number of diagnoses of cancer in a particular geographic area of town.

- To evaluate the geographic distribution of individual cancer cases in Belmont and surrounding communities and see if there are any patterns in particular areas of the towns or in relation to known sources of environmental contamination.

- To review descriptive information available from the Massachusetts Cancer Registry (MCR) for individuals diagnosed with cancer in Belmont, Cambridge, Arlington, and Watertown, to see if there are any particular characteristics related to risk factors for developing these diseases.

- To review available information regarding oil and hazardous material releases in Belmont, Arlington, Cambridge, and Watertown as reported to the Massachusetts Department of Environmental Protection (MDEP).

- To consider the results of this evaluation in the context of the available scientific and medical literature on cancer to determine whether further investigation or public health action is warranted.

III. METHODS

A. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–1999 were obtained for the town of Belmont and the surrounding communities of Arlington, Cambridge,

and Watertown from the Massachusetts Cancer Registry (MCR), a division of the Bureau of Health Information, Statistics, Research and Evaluation within the MDPH. Six cancer types were evaluated in this investigation, including cancers of the kidney, liver, lung and bronchus, and pancreas as well as leukemia and non-Hodgkin's lymphoma (NHL). Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. (See Appendix B for the incidence coding definitions used in this report for these cancer types.) These cancer types were selected for evaluation on the basis of elevations observed at the town level in a preliminary review of cancer rates in Belmont, potential associations with contaminants of concern at the Cambridge Plating site (primarily TCE and chromium), and/or residents' concerns about suspected elevations in some cancer types. Only cases reported to the MCR as a primary cancer for one of the six cancer types and diagnosed among residents of Belmont, Arlington, Cambridge, and Watertown were included in the analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis.

The MCR is a population-based surveillance system that began collecting information on Massachusetts residents diagnosed with cancer in the state in 1982. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within six months of the date of diagnosis (M.G.L. c.111s.111B). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment). Once these steps are finished, the data for that year are considered "complete." Due to the volume of information received by the MCR, the large number of reporting facilities, and the six-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of two years prior to the current date. The 18-year period 1982–1999 constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis.[1]

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Epidemiologic studies have revealed that different types of cancer are individual

diseases with separate causes, risk factors, characteristics, and patterns of survival (Berg 1996). Cancer types are classified by the location in the body where the disease originated (the primary site) and the tissue or cell type of the cancer (histology). Each cancer type reviewed in this report was evaluated separately. Cancers that occur as the result of the metastasis or the spread of a primary site cancer to another location in the body are not considered as separate cancers and therefore were not included in this analysis.

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital or diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than two months after the initial diagnosis (MCR 1996). Therefore, duplicate reports of an individual diagnosed with cancer were removed from the analyses whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases. In the town of Belmont, four duplicate reports were identified during the years 1982–1999 and excluded from the analyses. In addition, seven duplicate reports in Arlington, 16 duplicate reports in Cambridge, and 12 duplicate reports in Watertown were identified during the years 1982–1999 and excluded from the analyses.

B. Calculation of Standardized Incidence Ratios

To determine whether elevated numbers of cancer cases occurred in Belmont and the surrounding communities of Arlington, Cambridge, and Watertown, cancer incidence data were tabulated by gender according to six age groups to compare the observed number of cancer cases

[1] The data summarized in this report are drawn from data entered on MCR computer files before April 28, 2003. The numbers presented in this report may change slightly in future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for the period 1982–1999 for each of the six primary cancer types for Belmont as a whole as well as for each census tract (CT) within Belmont. In addition, SIRs were calculated for the city of Cambridge as a whole and the three Cambridge CTs located on the border of Belmont. Townwide SIRs were also calculated for Arlington and Watertown. SIRs were also calculated for three smaller time periods, 1982–1987, 1988–1993, 1994–1999, in order to evaluate patterns or trends in cancer incidence over time.

To calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated on the basis of 1980, 1990, and 2000 U.S. Census data for each CT in Belmont and Cambridge and for the town of Belmont as a whole, the city of Cambridge as a whole, and the towns of Arlington and Watertown (U.S. Census Bureau 1980, 1990, and 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990 and 1996). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the 10-year interval between each census.[2]

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

According to the U.S. Census, the town of Belmont is subdivided into eight census tracts (i.e., CTs 3571–3578). Of the 30 census tracts in Cambridge, three are located on the border of Belmont (i.e., CTs 3543, 3546, and 3549) (U.S. Census Bureau 2000). The town boundaries and census tract locations for Belmont and surrounding communities are illustrated in Figure 1. Cases for which census tract designation was not possible were included in the city/town totals for Belmont and Cambridge.

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

C. Interpretation of a Standardized Incidence Ratio

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

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

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

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on four expected cases and six observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed for a particular cancer type.

D. Calculation of the 95% Confidence Interval

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

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

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

E. Evaluation of Cancer Risk Factor Information

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types

evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes the individual's age at time of diagnosis, the stage of disease, and the individual's smoking history and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to lung and bronchus, kidney, and pancreatic cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for residents of the four communities who were diagnosed with any of the six cancer types included in this report. However, information about personal risk factors such as family history, hormonal events, diet, and other factors that may also influence the development of cancer is not collected by the MCR, and therefore, it was not possible to evaluate these factors in this investigation.

F. Determination of Geographic Distribution of Cancer Cases

In addition to calculation of SIRs, address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2002). This allowed assignment of census tract location as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer cases in each of the four communities. In instances where the address information from the MCR was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using telephone books issued within two years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. (Note: MDPH is bound by Massachusetts General Laws and the new federal Health Insurance Portability and Accountability Act (HIPAA) not to reveal the name or identifying information of an individual diagnosed with cancer whose case is reported to the MCR.)

IV. RESULTS OF CANCER INCIDENCE ANALYSIS

The following sections present cancer incidence rates for Belmont and its individual census tracts, Cambridge and selected census tracts, Arlington, and Watertown during the 18-year time period 1982–1999. To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–1999. SIRs were not calculated for some cancer types in smaller time periods 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 excess numbers of cancer cases were occurring.

A. Cancer Incidence in Belmont

With a few exceptions, the six cancer types evaluated in this report generally occurred approximately near or below expected rates in the town of Belmont as a whole during the 18-year time period 1982–1999 as well as smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–1999) (see Tables 1a through 6d). The increases observed were mostly based on approximately three or fewer additional cases above the expected number and incidence rates were not statistically significant. Overall, kidney cancer, lung and bronchus cancer, and NHL occurred less often than expected during 1982–1999. In general, with some exceptions which are noted in the following paragraphs, residents of most Belmont census tracts experienced cancer approximately at or near the rates expected during 1982–1999 and during the smaller time periods evaluated in this report.

1. Kidney Cancer

As detailed in Tables 1a – 1d, the incidence of kidney cancer appears to have decreased over time among both male and female residents of Belmont. During the most recent time period evaluated, 1994–1999, 13 individuals were diagnosed with kidney cancer compared to approximately 21 diagnoses expected.

During the 18-year time period 1982–1999, kidney cancer occurred slightly below expected rates in the majority of census tracts in Belmont. Residents of CT 3576 experienced a slight increase in the incidence of this cancer type (7 diagnoses observed vs. 5.4 expected, SIR = 129). In CT

3572, eight individuals were diagnosed with kidney cancer compared to 7.2 expected. However, these elevations were not statistically significant. No specific trends were observed when cancer incidence data were evaluated by smaller time periods. That is, kidney cancer generally occurred about as expected (i.e., within one or two cases of the expected number) in individual census tracts during 1982–1987, 1988–1993, and 1994–1999 (see Tables 1a – 1d).

2. Leukemia

The incidence of leukemia was slightly elevated in the town of Belmont as a whole during 1982–1999 (58 diagnoses observed vs. 50.3 expected, SIR = 115). Although leukemia was diagnosed less often than expected during the earliest time period, 1982–1987, both males and females experienced slight increases in the rate of this cancer type during 1988–1993 and during 1994–1999. None of the observed elevations were statistically significant (see Tables 2a – 2d).

The incidence of leukemia was slightly elevated with respect to the state rate in several Belmont census tracts during the overall time period 1982–1999. Specifically, residents of CTs 3571, 3573, 3574, 3577, and 3578 were diagnosed with leukemia slightly more often than expected during this time period. These increases were not statistically significant. When these data were analyzed for smaller time periods, no consistent trends in the incidence of leukemia were noted. In CT 3751 during 1982–1987, leukemia occurred at about the rate expected, but occurred slightly more often than expected in 1988–1993 and 1994–1999. Although one individual in CT 3573 was diagnosed with leukemia during 1982–1993 compared to 3.2 expected, residents of this census tract experienced a statistically significant elevation in the incidence of leukemia during the most recent time period 1994–1999 (7 diagnoses observed vs. 1.8 expected, SIR = 383, 95% CI = 153–789). However, the wide confidence interval indicates that the increased SIR is somewhat unstable. Therefore, it is uncertain based on this data whether the incidence of leukemia is increasing in this area of Belmont. Additional information for the individuals diagnosed with leukemia in Belmont will be evaluated later in this report.

In CT 3574, leukemia was diagnosed slightly more often than expected during 1982–1987 and about as often as expected during 1988–1993 and 1994–1999. In CT 3577, leukemia occurred about as often as expected during 1982–1987, more often than expected during 1988–1993, and less often than expected during 1994–1999. Finally, residents of CT 3578 experienced lower-

than-expected rates of leukemia during 1982–1987, but a statistically significant elevation in the incidence of this cancer type during 1988–1993 (7 diagnoses observed vs. 2.4 expected, SIR = 290, 95% CI = 116-598). Again, this SIR should be interpreted with caution because of the wide confidence interval. The incidence of leukemia was lower than expected in more recent years (i.e., 1994–1999) in this census tract. Refer to Tables 2a – 2d for a summary of these data.

3. Liver Cancer

A slight elevation in the incidence of liver cancer was observed in the town of Belmont as a whole during 1982–1999. However, the increase was primarily based on approximately one additional diagnosis over the expected number each among males and females during the 1988–1993 time period and was not statistically significant.

No individuals were diagnosed with liver cancer during 1982–1999 in CT 3574 where about one case was expected. There was one diagnosis each in CTs 3572, 3575, 3576, and 3578. Residents of CTs 3571, 3573, and 3577 experienced slight elevations in the incidence of liver cancer during 1982 – 1999. However, these increases were generally based on fewer than three cases above the expected numbers (in most instances, the numbers of observed cases was less than five and therefore statistical significance was not evaluated). Analysis over time revealed no specific trends in the incidence of liver cancer in Belmont census tracts (see Tables 3a – 3d).

4. Lung and Bronchus Cancer

The incidence rate of lung and bronchus cancer was statistically significantly lower than expected in the town as a whole (270 diagnoses observed vs. 375.3 expected, SIR = 72, 95% CI = 64–81) during the 18-year time period 1982–1999. Analysis of trends over time revealed that incidence rates for this cancer type were lower than expected during each of the smaller time periods evaluated and statistically significantly lower than expected during 1982–1987 and 1994–1999 (see Tables 4a – 74d).

The incidence of lung and bronchus cancer was lower than expected in all Belmont census tracts during the 18-year time period 1982–1999. Moreover, the rates were statistically significantly lower than expected among males and females combined in CTs 3572, 3573, 3574, 3576, and 3578 and among males when evaluated separately by gender in CTs 3571, 3573, and 3578.

Similar trends were observed when these data were evaluated for smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–1999) (see Tables 4a – 4d).

5. Non-Hodgkin's Lymphoma

NHL occurred less often than expected in the town of Belmont as a whole during 1982–1999, primarily due to a lower-than-expected rate among males in the town (32 diagnoses observed vs. 44.6 expected, SIR = 72), a rate that was borderline statistically significant (95% CI = 49–101). Males in Belmont experienced fewer diagnoses of NHL than expected during each of the smaller time periods evaluated while females were diagnosed about as expected during 1982–1987 and slightly more often than expected during the most recent time periods (i.e., 1988–1993 and 1994–1999) (see Tables 5a – 5d). Each of the elevations in the recent time periods represented an excess of about three cases and neither was statistically significant.

While NHL occurred less often than expected in some census tracts in Belmont during 1982–1999 (i.e., CTs 3572, 3573, 3574, and 3576), this cancer type was diagnosed more often than expected among males and females combined in CTs 3575, 3577, and 3578. Although these elevations were not statistically significant, females in CT 3577 experienced a statistically significant elevation in the incidence of NHL during the 18-year time period evaluated (14 diagnoses observed vs. 7.4 expected, SIR = 189, 95% CI = 103–316). This elevation was primarily due to increases in NHL diagnoses observed among females during 1988–1993 and 1994–1999. Moreover, the elevation observed during the most recent time period (i.e., 1994–1999) was also statistically significant. Slight elevations were also noted in other Belmont census tracts during one or more of the smaller time periods evaluated; the observed elevations were generally based on one to two additional diagnoses over the expected number (see Tables 5a – 5d).

6. Pancreatic Cancer

Pancreatic cancer in the town of Belmont during 1982–1999 occurred about as expected when compared with the statewide cancer experience. Although the overall rate remained consistent over time, different trends were observed among males and females when evaluated separately

by gender. Specifically, the incidence of pancreatic cancer in Belmont appears to have increased over time among males while decreasing over time among females (see Tables 6a – 6d).

Pancreatic cancer occurred less often than expected in Belmont CTs 3572, 3573, 3574, 3575, and 3576 and more often than expected in CTs 3571, 3577, and 3578 during the 18-year time period 1982–1999. However, none of the observed elevations were statistically significant. The overall rate of pancreatic cancer in CT 3571 was due to slight elevations during the earliest time period (i.e., 1982–1987) and the most recent time period (i.e., 1994–1999). In CT 3577, slight elevations in the incidence of pancreatic cancer were observed during 1988–1993 and 1994–1999. Finally, in CT 3578, pancreatic cancer was diagnosed more often than expected during 1982–1987 and 1988–1993. The CT 3578 elevations were the result of increased diagnoses among females, who experienced a statistically significant elevation during 1982–1987 (5 diagnoses observed vs. 1.5 expected, SIR = 343, 95% CI = 110–800). The increased SIR should be interpreted with caution due to the wide confidence interval. Pancreatic cancer among females in CT 3878 was also elevated during 1988–1993 (4 diagnoses observed vs. 1.4 expected). During the more recent time period (i.e., 1994–1999), no females were diagnosed with pancreatic cancer in CT 3578 (see Tables 6a – 6d).

B. **Cancer Incidence in Cambridge**

As described previously, cancer incidence rates were also evaluated for the city of Cambridge, with a particular focus on census tracts adjacent to Belmont (i.e., CTs 3543, 3546, and 3549). During the 1982–1999 time period, citywide incidence rates were lower than expected for kidney cancer, leukemia, lung and bronchus cancer, and NHL; about as expected for pancreatic cancer; and slightly higher than expected for liver cancer. The elevation in liver cancer incidence was not statistically significant. Census tract-specific SIRs could not be calculated for the majority of cancer types because of the small numbers of observed cases; however, the number of cases observed was compared to the number of cases expected to determine if an atypical pattern of cancer was occurring. The results of these analyses are summarized in the following paragraphs and presented in Tables 7a – 12d.

1. *Kidney Cancer*

A statistically significant decrease in the incidence of kidney cancer was observed in the city of Cambridge as a whole during 1982–1999 (109 diagnoses observed vs. 146.3 expected, SIR = 75, 95% CI = 61–90). Both males and females experienced lower-than-expected rates of this cancer type with the rate among males statistically significantly less than expected. Residents of CT 3543 experienced kidney cancer at about the rate expected among males and females combined while residents of CTs 3546 and 3549 were diagnosed with kidney cancer less often than expected (see Table 7a).

Both males and females in Cambridge as a whole experienced lower-than-expected rates of kidney cancer during each of the smaller time periods evaluated (i.e., 1982–1987, 1988–1993, and 1994–1999). Moreover, the rates were statistically significantly decreased among males and females combined and among males separately during 1988–1993 (see Tables 7b – 7d).

2. *Leukemia*

Statistically significant decreases in leukemia incidence were observed among males and among males and females combined in Cambridge as a whole during 1982–1999. In addition, females experienced lower-than-expected incidence that was borderline statistically significant. Specifically, 102 individuals were diagnosed with leukemia compared to 134.1 expected (SIR = 76, 95% CI = 62–92). Overall rates were also lower than expected in each of the three Cambridge census tracts evaluated (i.e., CTs 3543, 3546, and 3549) (see Table 8a). Similar trends were noted when these data were reviewed by smaller time periods. Specifically, citywide rates were lower than expected during 1982–1987, 1988–1993, and 1994–1999 and statistically significantly lower than expected during 1988–1993 and during 1994–1999. In CTs 3543, 3546, and 3549, leukemia generally occurred at or near expected rates (i.e., within one or two cases of the expected number) during each smaller time period (see Tables 8b – 8d).

3. *Liver Cancer*

Both males and females in Cambridge experienced a slight elevation in the incidence of liver cancer during 1982–1999. Overall, 40 individuals were diagnosed with this disease compared to 34.2 expected (SIR = 117). This elevation was not statistically significant. In CT 3543, three

diagnoses were observed vs. 1.8 expected; in CT 3546, two diagnoses were observed vs. 1.7 expected; and in CT 3549, four diagnoses were observed vs. 1.8 expected (see Table 12a). Citywide rates of liver cancer were higher than expected during 1982–1987, about as expected during 1988–1993, and slightly higher than expected during 1994–1999. Neither of the elevations was statistically significant. Liver cancer generally occurred about as expected during each of the smaller time periods in CTs 3543, 3546, and 3549 (see Tables 9b – 9d).

4. Lung and Bronchus Cancer

Lung and bronchus cancer occurred statistically significantly less often than expected in the city of Cambridge during the 18-year time period 1982–1999 (799 diagnoses observed vs. 897.4 expected, SIR = 89, 95% CI = 83-95). Similar trends were observed when these data were evaluated separately by gender, with both males and females experiencing statistically significant decreases in the rate of this cancer. Residents of CTs 3543 and 3546 also experienced decreased rates of lung and bronchus cancer during this time period. In CT 3549, the incidence of lung and bronchus was about as expected (see Table 10a). Review of these data by smaller time period revealed that the rate of lung and bronchus cancer was about as expected in Cambridge during 1982–1987, statistically significantly lower than expected during 1988–1993, and statistically significantly decreased during 1994–1999. In CT 3543, the incidence of lung and bronchus cancer appears to have decreased over time. Specifically, the incidence of lung and bronchus cancer was elevated during 1982–1987 (primarily due to an elevation among males), lower than expected during 1988–1993, and statistically significantly lower than expected during 1994–1999. In CT 3546, rates were about as expected during 1982–1987 and lower than expected during 1988–1993 and 1994–1999. Finally, in CT 3549, rates of lung and bronchus cancer were about as expected during 1982–1987 and 1988–1993 and slightly elevated during 1994–1999. However, the elevation observed during the latest time period was not statistically significant (20 diagnoses observed vs. about 17 expected) (see Tables 10b – 10d).

5. Non-Hodgkin's Lymphoma

The incidence of NHL was statistically significantly lower than expected in the city of Cambridge during 1982–1999 (186 diagnoses observed vs. 242.5 expected, SIR = 77, 95% CI = 66–89). Rates were also statistically significantly below those expected among males

and borderline statistically significantly lower among females when evaluated separately by gender. NHL was also diagnosed less often than expected in CTs 3543, 3546, and 3549 during this time period (see Table 11a). The incidence of NHL in Cambridge has been consistently below the state rate over time, with statistically significant decreases during 1982–1987 and 1994–1999. Separate analyses of these data by gender revealed lower-than-expected incidence among both males and females, however, rates among males were borderline statistically significantly lower than expected during 1982–1987 and statistically significantly lower than expected during 1994–1999. NHL also occurred approximately at expected rates in Cambridge CTs 3543, 3546, and 3549 during each of the smaller time periods evaluated, with the observed numbers of cases generally within one to two cases of the expected numbers (see Tables 11b – 11d).

6. Pancreatic Cancer

Overall, pancreatic cancer was diagnosed slightly more often than expected in the city of Cambridge during 1982–1999 (139 diagnoses observed vs. 136.4 expected, SIR = 102), primarily due to an increase in diagnoses among males in the city. The elevation among males was not statistically significant (67 diagnoses observed vs. approximately 61 expected). Residents of CTs 3543 and 3546 were diagnosed with pancreatic cancer at about the rates expected. However, an elevation in the incidence of this cancer type was observed in CT 3549 during this time period (13 diagnoses observed vs. 7.0 expected, SIR = 187). This elevation was borderline statistically significant (95% CI = 99–320) and was due to a statistically significant elevation among males in this area of Cambridge (9 males diagnosed vs. 3.3 expected, SIR = 273, 95% CI = 125–519) (see Table 12a).

Analysis of trends over time suggests that the incidence of pancreatic cancer in the city as a whole is decreasing over time. During the most recent time period evaluated, 1994–1999, 43 individuals were diagnosed with pancreatic cancer in Cambridge compared to 48.0 expected (SIR = 89). Although SIRs could not be calculated due to the small number of observed cases (i.e., less than five), the incidence of pancreatic cancer also appears to be declining in CTs 3543 and 3546. The overall rate of pancreatic cancer observed in CT 3549 for the 18-year time period

1982–1999 appears to be the result of slight elevations in the incidence of this cancer type among males during each of the smaller time periods evaluated (see Tables 12b – 12d).

C. Cancer Incidence in Arlington

In general, with the exceptions noted in the following paragraphs, residents of Arlington experienced cancer approximately at or below the rates expected during 1982–1999 and the smaller time periods evaluated in this report. The results of the cancer incidence analyses for Arlington are summarized in the following paragraphs and shown in Tables 13a – 13d.

1. Kidney Cancer

During the 18-year time period 1982–1999, the overall incidence of kidney cancer was lower than expected in Arlington. While males were diagnosed with kidney cancer at about the rate expected, females in the town were diagnosed less often than expected. No consistent trends were noted when these data were evaluated by smaller time period. During 1982–1987, males were diagnosed with kidney cancer slightly more often than expected while females were diagnosed less often than expected. During 1988–1993, both males and females experienced a decreased incidence of kidney cancer with respect to the state rate. During the most recent time period evaluated, 1994–1999, females were diagnosed with kidney cancer at about the rate expected while males in Arlington experienced a slight elevation in the incidence of this cancer type. However, none of the elevations observed during smaller time periods were statistically significant.

2. Leukemia

Leukemia was diagnosed about as expected in Arlington during 1982–1999 (85 diagnoses observed vs. 86.3 expected, SIR = 98). Similar trends were observed among males and females when evaluated separately by gender. During 1982–1987, the overall rate of leukemia was slightly higher than expected in Arlington (30 diagnoses observed vs. 27.5 expected, SIR = 109). During 1988–1993, both males and females were diagnosed with leukemia less often than expected. Finally, during 1994–1999, males were diagnosed with leukemia slightly less often than expected while females experienced an elevation in the incidence of this cancer type. This elevation was not statistically significant (20 diagnoses observed vs. about 15 expected).

3. *Liver Cancer*

Overall, the incidence of liver cancer was higher than expected in Arlington during 1982–1999 (28 diagnoses observed vs. 23.4 expected, SIR = 120). This elevation, which was not statistically significant, was primarily due to an increase in the number of diagnoses among females in the town (11 diagnoses observed vs. 7.5 expected, SIR = 147). Liver cancer occurred about twice as often as expected during the earliest time period evaluated 1982–1987 (12 diagnoses observed vs. 6.0 expected, SIR = 201). This elevation was statistically significant (95% CI = 104–351). Similar trends were observed among males and females when evaluated separately by gender; however, the elevation among males was not statistically significant (8 diagnoses observed vs. about 4 expected), while an excess of about two cases was observed among females (4 diagnoses observed vs. about 2 expected). During 1988–1993, females were diagnosed with liver cancer about as expected, while males were diagnosed less often than expected. Finally, during the most recent time period, 1994–1999, males were diagnosed with liver cancer at about the rate expected while females were diagnosed slightly more often than expected (5 diagnoses observed vs. 3.0 expected).

4. *Lung and Bronchus Cancer*

Overall, during 1982–1999, the incidence of lung and bronchus cancer in Arlington was statistically significantly decreased with respect to the state rate (544 diagnoses observed vs. 643.8 expected, SIR = 84, 95% CI = 78–92). Both males and females were diagnosed with lung and bronchus cancer less often than expected during this time period. The rate among females was statistically significant. The overall incidence of lung and bronchus cancer was also statistically significantly lower than expected during each of the three smaller time periods evaluated (i.e., 1982–1987, 1988–1993, and 1994–1999). Statistically significantly lower-than-expected rates were also noted among females when evaluated separately by gender during 1982–1987 and 1994–1999).

5. *Non-Hodgkin's Lymphoma*

NHL was diagnosed at about the rate expected in Arlington during 1982–1999 (161 diagnoses observed vs. 158.7 expected, SIR = 101). Analysis of these data by smaller time period revealed

that among males, NHL was diagnosed less often than expected during 1982–1987, about as often as expected during 1988–1993, and slightly more often than expected during 1994–1999. Among females, NHL occurred more often than expected during 1982–1987, less often than expected during 1988–1993, and near the expected rate (i.e., within approximately one case of the expected number) during 1994–1999.

6. Pancreatic Cancer

The overall incidence of pancreatic cancer in Arlington was about as expected during 1982–1999 (102 diagnoses observed vs. 100.0 expected, SIR = 102). Males were diagnosed slightly more often than expected during this time period while females were diagnosed slightly less often than expected and neither difference was statistically significant. Among males and females combined, rates of pancreatic cancer have decreased over time in Arlington and were lower than expected during the most recent time period 1994–1999 (25 diagnoses observed vs. 34.0 expected, SIR = 74). Although males experienced a statistically significant elevation in the incidence of pancreatic cancer during 1982–1987 (25 diagnoses observed vs. 14.3 expected, SIR = 174, 95% CI = 113–257), rates among males were lower than expected during 1988–1993 and 1994–1999. Females were diagnosed with pancreatic cancer about as expected during 1982–1987, more often than expected during 1988–1993, and less often than expected during 1994–1999.

D. Cancer Incidence in Watertown

In general, with some exceptions noted below, residents of Watertown experienced cancer approximately at or near the rates expected during 1982–1999 and smaller time periods evaluated in this report. The results of the cancer incidence analyses for Watertown are summarized below and in Tables 14a – 14d.

1. Kidney Cancer

During the 18-year time period 1982–1999, kidney cancer occurred slightly more often than expected in Watertown as a whole (76 diagnoses observed vs. 70.4 expected, SIR = 108). This elevation was not statistically significant and was due primarily to an increased rate of this cancer type among males that was not statistically significant (48 diagnoses observed vs. about

40 expected). When these data were analyzed by smaller time periods, females were diagnosed with kidney cancer less often than expected during the two earlier time periods while males were diagnosed with this cancer type more often than expected. The elevation observed among males during 1988–1993 was statistically significant (24 diagnoses observed vs. 14.3 expected, SIR = 168, 95% CI = 108–251). However, during more recent years (i.e., 1994–1999), the incidence among males was lower than expected (11 diagnoses observed vs. 15 expected), while the incidence among females was about as expected.

2. Leukemia

A slight elevation in the incidence of leukemia in Watertown was noted during 1982–1999 (68 diagnoses observed vs. 61.1 expected, SIR = 111). However, this elevation was based on an elevation in males (38 diagnoses observed vs. 31.5 expected), and neither elevation was statistically significant. Review of smaller time periods revealed that during the earliest time period evaluated (1982–1987), 18 individuals were diagnosed with leukemia compared to 19.1 expected (SIR = 94). During 1988–1993, 21 diagnoses were reported vs. 18.5 expected (SIR = 113). Finally, during 1994–1999, 29 individuals were diagnosed where 23.5 diagnoses were expected (SIR = 124), an elevation that was not statistically significant. The elevation during 1994–1999 was due to an excess of about three cases each among males and females.

3. Liver Cancer

In Watertown during 1982–1999, the incidence of liver cancer was higher than expected based on the state rate (21 diagnoses observed vs. 16.5 expected, SIR = 127). No females were diagnosed with liver cancer during the 18 years evaluated where approximately five diagnoses were expected. The rate of liver cancer among males in Watertown was statistically significantly elevated during this time period (21 diagnoses observed vs. 11.2 expected, SIR = 188, 95% CI = 116–287). Liver cancer was diagnosed more often than expected among males in Watertown during each of the smaller time periods evaluated (i.e., 1982–1987, 1988–1993, and 1994–1999). None of the elevations in smaller time periods was statistically significant, although the incidence in the most recent time period nearly achieved statistical significance (10 diagnoses observed vs. 4.9 expected, SIR = 204, 95% CI = 98–376).

4. Lung and Bronchus Cancer

Overall, during 1982–1999, lung and bronchus cancer occurred less often than expected in Watertown (423 diagnoses observed vs. 452.4 expected, SIR = 93). Similar trends were observed among males and females when evaluated separately by gender. Results of analyses by smaller time period suggest that the overall incidence of lung and bronchus cancer may be decreasing over time in Watertown. During the most recent time period, 1994–1999, the decrease in lung and bronchus cancer diagnoses was statistically significant among males (60 diagnoses observed vs. 82.5 expected, SIR = 73, 95% CI = 56–94) and among males and females combined (134 diagnoses observed vs. 160.5, SIR = 83, 95% CI = 70–99).

5. Non-Hodgkin's Lymphoma

NHL occurred more often than expected in the town of Watertown as a whole during 1982–1999 (137 diagnoses observed vs. 113.2 expected, SIR = 121). The observed elevation was statistically significant (95% CI = 102–143). Both males and females experienced increases in the incidence of NHL during this time period; however, neither elevation was statistically significant. NHL occurred more often than expected based on the state rate among both males and females during 1982–1987 and 1988–1993, although females experienced a relatively higher incidence of this cancer type during these years. During the most recent time period evaluated, 1994–1999, the incidence of NHL among females occurred about as expected, while an excess of about five cases occurred among males (26 diagnoses observed vs. approximately 21 expected).

6. Pancreatic Cancer

The overall incidence of pancreatic cancer was lower than expected in Watertown during 1982–1999 (66 diagnoses observed vs. 70.3 expected, SIR = 94). This rate reflected a slightly higher-than-expected rate among males and a lower-than-expected rate among females when evaluated separately by gender, but no difference was statistically significant. Analysis of these data by smaller time periods revealed that among males, the incidence of pancreatic cancer occurred less often than expected during 1982–1987 and more often than expected during the latter two time periods. Among females, the incidence of pancreatic cancer was about as

expected during 1982–1987 and lower than expected during 1988–1993 and 1994–1999. No difference was statistically significant.

V. REVIEW OF CANCER RISK FACTOR INFORMATION

As previously mentioned, 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 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, Cambridge, Arlington, and Watertown. Information for each of the six cancer types evaluated in this report 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. In this investigation, therefore, it was not possible to evaluate the role these types of risk factors may have played in the incidence of cancer in Belmont and surrounding communities. For detailed information regarding risk factors associated with the cancer types evaluated in this report, please refer to Appendix C.

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. 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 these cancer types in Belmont and surrounding towns was reviewed to assess the role tobacco smoking may have played in the development of these types of 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 with specific occupational exposure 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 by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types to determine the role that occupational factors may have played in the development of these types of cancer in Belmont and surrounding towns. 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 individuals. In addition, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

Finally, histologic (cell type) distribution was reviewed for diagnoses of lung and bronchus cancer and leukemia. Patterns of disease were compared to known or established incidence trends to assess whether any unexpected patterns exist in these areas.

A. Kidney Cancer

Kidney cancer is twice as common in males as it is in females, and the incidence most often occurs in the fifth and sixth decades of life (50–70 year age group) (ACS 2001a). The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 30% to 100% (ACS 2001a). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of the renal cell cancers in women may be caused by cigarette smoking (ACS 2001a).

Although kidney cancer is not generally considered an occupationally associated cancer, some studies have suggested that environmental and occupational factors may be associated with its development. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. In addition, exposure to cadmium is associated with an increased incidence of kidney cancer, particularly among men who smoke. In addition, workplace exposure to organic solvents, such as trichloroethylene

(TCE), may increase the risk of this cancer (ACS 2001a). More recently, renal cell carcinoma (RCC), the most common type of kidney cancer, has been suggested to be associated with occupational exposure to petroleum, tar, and pitch products. However, studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between exposure to gasoline or other petroleum products and kidney cancer (Linehan et al. 1997; McLaughlin et al. 1996).

1. Age and Gender

The average age of individuals diagnosed with kidney cancer in Belmont during 1982–1999 was 63 years. Eighty-three percent (n = 43) were age 50 or older at the time of diagnosis. This pattern is consistent with what would be expected in the general population. Overall, males experienced kidney cancer at about the rate expected, while females were diagnosed less often than expected based on the state rate. Review of age-group-specific SIRs revealed a slightly increased rate of kidney cancer among males aged 20–44 years while males in older age groups (e.g., 75–84 years and 85+ years) were diagnosed less often than expected. Females in all age groups were diagnosed with kidney cancer approximately at or below rates expected.

In Cambridge, the average age of individuals diagnosed with kidney cancer during 1982–1999 was 61 years. This is comparable to that observed in the general population. The majority of those diagnosed (82%, n = 89) were age 50 or older at the time of diagnosis. Incidence rates were lower than expected among males in all age groups, resulting in an overall rate that was statistically significantly lower than expected. Similar trends were observed when age-group-specific rates were reviewed for females in Cambridge. Females in most age groups were diagnosed with kidney cancer about as or less often than expected.

In Arlington, the average age of individuals diagnosed with kidney cancer during 1982–1999 was 65 years. Eighty-four percent (n = 79) were age 50 or older at the time of diagnosis. Males in most age groups experienced kidney cancer approximately at or below expected rates. However, males between the ages of 65 and 74 were diagnosed with kidney cancer slightly more often than expected. Females in all age groups were diagnosed with kidney cancer either approximately at or below expected rates.

The average age of individuals diagnosed with kidney cancer in Watertown during 1982–1999 was 68 years. Ninety-seven percent (n = 74) were over the age of 50 at the time of diagnosis. The slight overall elevation in kidney cancer incidence among males in Watertown was the result of increased diagnoses among males aged 45–84 years. Females in most age groups experienced rates of kidney cancer that were lower than expected. However, a slight elevation in incidence was noted among females aged 65–74 years.

2. Smoking History

Of the 52 individuals diagnosed with kidney cancer in Belmont during 1982–1999, 31% (n = 16) reported being current or former smokers at the time of diagnosis. Fifty percent (n = 26) were nonsmokers and smoking history was unknown for the remaining 19% (n = 10). This distribution is different than that observed for the state as a whole during this time period. In Massachusetts, 45% of individuals diagnosed with kidney cancer were current or former smokers at the time of diagnosis, 33% were nonsmokers, and smoking history was unknown for approximately 22%.

In Cambridge, 39% (n = 42) of those diagnosed with kidney cancer in Cambridge during 1982–1999 reported being current or former smokers at the time of diagnosis. Forty-six percent (n = 50) were nonsmokers and smoking history was unknown for 16% (n = 17).

Of the 94 individuals diagnosed with kidney cancer in Arlington during 1982–1999, 41% (n = 39) reported being current or former smokers at the time of diagnosis. Forty-nine percent (n = 46) were nonsmokers and smoking history was unknown for the remaining 10% (n = 9).

In Watertown, 50% of those diagnosed with kidney cancer during 1982–1999 (n = 38) reported being current or former smokers at the time of diagnosis. Thirty-seven percent (n = 28) were nonsmokers and smoking history was unknown for the remaining 13% (n = 10). Of the four communities evaluated, Watertown was the only community with an overall elevation (not statistically significant) in kidney cancer during 1982–1999. The smoking information suggests that smoking may have played a role in the overall elevation of kidney cancer among residents of Watertown.

3. *Occupation*

Review of occupational information as reported to the MCR for individuals diagnosed with kidney cancer in Belmont did not reveal any jobs likely to be related to an increased risk of developing kidney cancer. However, occupation was reported as retired, unknown, or "at home" for almost half of these individuals (44%, n = 23).

In Cambridge, one individual reported an occupation that may be associated with exposures related to kidney cancer. No other jobs likely associated with an increased risk of kidney cancer were indicated. However, occupation was unknown or reported as retired or at home for 39% of individuals (n = 42).

In Arlington, five individuals reported jobs in which occupational exposures possibly associated with kidney cancer could have been possible. However, specific job duty information that could further define exposure potential for these individuals was unavailable. Occupation was unknown or reported as retired or at home for 38 individuals (39%).

Most individuals diagnosed with kidney cancer in Watertown during 1982–1999 did not indicate working in jobs in which occupational exposures possibly associated with kidney cancer would have been likely. However, one individual reported an occupation in which an increased risk is possible. Occupation was unknown or reported as retired or at home for the majority of those diagnosed (53%, n = 40).

B. Leukemia

In 2003, leukemia is expected to affect approximately 30,600 individuals (17,900 males and 12,700 females) in the United States, resulting in 21,900 deaths. In Massachusetts, approximately 700 individuals will be diagnosed with the disease in 2003, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also several rare types of leukemia (e.g., hairy cell leukemia, myelomonocytic leukemia). In adults, the most common types are AML and CLL. Leukemia is the most common type of childhood cancer, accounting for more than 30% of all cancers diagnosed in children. The majority of these cases are of the ALL type (ACS 2003).

The various subtypes of leukemia occur with different frequencies in the population. For the purpose of classification in this evaluation, if the histology (i.e., cell type) of the leukemia diagnosis was not otherwise specified or not classified as one of the four main subtypes, then the individual case was categorized as "other." Available information regarding the expected distribution of leukemia by histology types can vary considerably depending on coding methods, making comparisons of type-specific incidence rates from different cancer registries difficult (Linet and Cartwright 1996). In the state of Massachusetts during the time period 1982–1999, 33.7% of all leukemia cases were AML, 26.3% were CLL, 13.1% were ALL, 10.9% were CML, and 16.0% were other histology types.

Several occupational exposures have been identified as playing a role in the development of leukemia. For example, exposures to particular chemicals are thought to increase the risk of developing certain kinds of leukemia. Exposure to ionizing radiation and chronic, high-dose exposure to pesticides and other chemicals such as benzene, have also been suggested as possible risk factors for leukemia (Linet and Cartwright 1996). Chronic occupational exposure to benzene has been established as a cause of AML. High doses of radiation among survivors of atomic bomb blasts or nuclear reactor accidents are associated with an increased incidence of AML, CML, and ALL, but no association has been established for lower doses such as those used in medical diagnostics.

1. Age and Gender

The average age of individuals diagnosed with leukemia in Belmont was 67 years. Eighty-four percent (n = 49) were age 50 or older at the time of diagnosis. Three diagnoses occurred among children (all under the age of 10 at diagnosis), which is about the number expected. The townwide elevation in the incidence of leukemia was due primarily to increased incidence among females in Belmont during 1982–1999. Review of age-group-specific SIRs suggests that this elevation was not the result of increased diagnoses among females in any one age group. Males generally experienced leukemia approximately at or below the expected rates, with the exception of males aged 65–74 years who were diagnosed with leukemia slightly more often than expected.

In Cambridge, the average age at diagnosis for individuals diagnosed with leukemia during 1982–1999 was 56 years. Fourteen individuals (14%) were between the ages of 0 and 19 years at diagnosis. This was about the number expected in this age group (8 diagnoses observed among males vs. 7.7 expected; 6 diagnoses observed among females vs. 5.9 expected). As noted previously, overall rates of leukemia were statistically significantly lower than expected among both males and females. Review of age-group-specific incidence rates revealed that both males and females experienced leukemia approximately at or below the rates expected for each age group.

Among the 85 individuals diagnosed with leukemia in Arlington during 1982–1999, the average age of diagnosis was 57 years. Sixty-eight percent of the diagnoses (n = 58) occurred among adults over the age of 50 years. Twelve of the diagnoses (14%) occurred among children under the age of 19 years. Among males aged 0–19 years, three individuals were diagnosed, about the number expected. However, nine females aged 0–19 years were diagnosed with leukemia in Arlington compared to 2.6 diagnoses expected. For other age groups among males and females, no unusual patterns were observed.

In Watertown, the average age of individuals diagnosed with leukemia was 64 years. Eighty-one percent (n = 55) were over the age of 50 at the time of diagnosis. Four diagnoses occurred among children (i.e., individuals aged 0–19 years), about the number expected. Overall, there was a slight elevation in the incidence of this cancer type among males in the town while females experienced leukemia at about the rate expected. Among males, increased rates were noted among individuals aged 65–84 years. Females in most age groups were diagnosed with leukemia less often than expected. However, slight elevations were noted among females aged 45–64 and 75–84.

2. Histology

Of the 58 individuals diagnosed with leukemia in Belmont during 1982–1999, 41.4% were diagnosed with the AML subtype, 27.6% were diagnosed with CLL, 8.6% were diagnosed with ALL, 6.9% were diagnosed with CML, and 15.5% were diagnosed with other types of leukemia. This distribution is similar to that seen statewide, with the exception that the AML subtype comprised a relatively larger proportion of leukemia diagnoses while the ALL and CML

subtypes comprised a relatively smaller proportion of diagnoses. However, considering the relatively small number of total diagnoses in the town of Belmont compared to the state as a whole during the 1982–1999 time period, the overall pattern does not appear to be atypical. All three children diagnosed with leukemia in Belmont were diagnosed with the ALL subtype, the most common subtype among children.

Among the 102 leukemia diagnoses in Cambridge during 1982–1999, 38.2% of the diagnoses were of the AML subtype, 20.6% were CLL, 8.8% were ALL, 8.8% were CML, and 19.6% were other types. This pattern is generally consistent with the statewide pattern. Among the 14 individuals between the ages of 0 and 19 years at diagnosis, four were diagnosed with ALL, eight were diagnosed with AML, and two were diagnosed with other subtypes.

In Arlington, the distribution of leukemia diagnoses by histology type was generally consistent with that seen in the state as a whole. Specifically, 34.1% of the individuals were diagnosed with AML, 20.0% were diagnosed with CLL, 21.2% were diagnosed with ALL, 10.6% were diagnosed with CML, and 14.1% were diagnosed with other types of leukemia. The fact that ALL diagnoses represented a relatively larger proportion of total diagnoses in Arlington compared to the state can likely be attributed to the increased incidence of leukemia among children aged 0–19 years in Arlington, among whom 11 out of 12 were diagnosed with the ALL subtype, the most commonly diagnosed leukemia in children.

Of the 69 individuals diagnosed with leukemia in Watertown during 1982–1999, 29.4% were diagnosed with the AML subtype, 22.1% were diagnosed with CLL, 10.3% were diagnosed with ALL, 11.8% were diagnosed with CML, and 25.0% were diagnosed with other types of leukemia. This is similar to the distribution seen in Massachusetts as a whole. All four individuals under the age of 19 were diagnosed with the ALL subtype.

3. Occupation

Review of occupations for individuals diagnosed with leukemia in Belmont revealed that two individuals may have worked in jobs in which occupational exposures potentially related to the development of leukemia may have been possible. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not available.

Occupations reported for the remaining individuals are not likely to be related to an increased risk of this cancer type. However, for more than half of the individuals diagnosed with leukemia in Belmont (53%, n = 31), occupation was reported as retired, unknown, or "at home."

Occupational exposures possibly related to an increased risk of leukemia may have been possible for three individuals diagnosed in Cambridge during 1982–1999. Occupation was unknown or reported as retired or at home for almost half of those diagnosed (45%, n = 46).

None of the 85 individuals diagnosed with leukemia in Arlington indicated working in occupations in which occupational exposures associated with leukemia are likely. Occupation was unknown or reported as retired or at home for 25% of those diagnosed (n = 21).

In Watertown, one individual reported working in a job in which occupational exposures associated with an increased risk of leukemia may have been possible. Occupation was unknown or reported as retired or at home for half of those diagnosed (n = 34).

C. Liver Cancer

An estimated 17,300 people in the United States (11,700 men and 5,600 women) will be diagnosed with liver cancer in 2003, accounting for approximately 1% of all new diagnoses of cancer (ACS 2003). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Men are at least two to three times more likely to develop liver cancer than women (Yu et al. 2000). Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn 1996). Although chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS 2001b), epidemiologic and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, vinyl chloride (a known human carcinogen used in the manufacturing of some plastics) and thorium dioxide (used in the past for certain x-ray tests) are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2001b; London and McGlynn 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. In addition, arsenic has been associated with an increased risk of liver cancer (ATSDR 2000b).

1. *Age and Gender*

For the 16 individuals diagnosed with liver cancer in Belmont during 1982–1999, the average age at diagnosis was 66 years, which is consistent with trends for this cancer type in the general population. The majority of individuals (88%, n = 14) were age 50 or older at the time of diagnosis. As discussed previously, both males and females experienced slightly increased rates of liver cancer during 1982–1999 (i.e., approximately one additional diagnosis each). While males aged 75–84 were diagnosed with liver cancer more often than expected, males in each of the other age groups evaluated were diagnosed approximately at or below the rates expected. Females in each age group were generally diagnosed at approximately the rates expected.

The average age at diagnosis for individuals diagnosed with liver cancer in Cambridge during 1982–1999 was 64 years. Eight-three percent (n = 33) of those diagnosed were age 50 or older at the time of diagnosis. The slight elevation in incidence observed among males in Cambridge was primarily the result of an elevation among males aged 65–74 years. Among females, incidence was about as expected for most age groups.

In Arlington, the average age of individuals diagnosed with liver cancer during 1982–1999 was 72 years. Among the 28 individuals diagnosed, only one was under the age of 50 years at the time of diagnosis. The slight elevation in incidence observed among males during this time period was primarily due to an increased number of diagnoses among males aged 75–84 while males in other age groups were diagnosed approximately at or below expected rates. Among females, the observed elevation was due to increases in diagnoses among individuals aged 65–84 years.

The average age of individuals diagnosed with liver cancer in Watertown was 65 years. Ninety percent (n = 19) of those diagnosed were over the age of 50 at the time of diagnosis. The statistically significant elevation observed in the rate of liver cancer among males in Watertown during 1982–1999 was primarily the result of increased diagnoses among individuals aged 45–84 years. There were no liver cancer diagnoses among females in Watertown compared to about 5 expected.

2. Occupation

None of the 16 individuals diagnosed with liver cancer in Belmont reported working in an occupation likely to be associated with an increased risk of developing liver cancer. However, occupation was unknown or reported as "at home" for three individuals.

Review of occupational information for the 40 individuals diagnosed with liver cancer in Cambridge did not indicate any jobs likely to be associated with an increased risk of this cancer type. However, occupation was unknown or reported as retired or at home for 40% (n = 16) of those diagnosed.

None of the jobs reported by the 28 individuals diagnosed with liver cancer in Arlington were likely associated with an increased risk of this cancer type. However, occupation was unknown or reported as retired or at home for half of the individuals (n = 14).

None of the 21 individuals diagnosed with liver cancer in Watertown indicated possible occupational exposures that could be associated with liver cancer. However, occupation was unknown or reported as retired for almost a third of the individuals (29%, n = 6).

D. Lung and Bronchus Cancer

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

Lung cancer is divided into two main types: small cell lung cancer and nonsmall cell lung cancer. Nonsmall cell lung cancer is further subdivided into three types: adenocarcinoma, squamous cell carcinoma, and large cell undifferentiated carcinoma. The different types of lung cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung cancers are adenocarcinomas, 25%–30% are squamous cell carcinomas, 20% are small cell cancers, and 10%–15% of cases are large cell carcinomas (ACS 2002). Rates in Massachusetts are very similar to those seen nationally.

About 87% of all lung cancers are thought to be caused directly by smoking cigarettes or by exposure to secondhand smoke, or environmental tobacco smoke (ACS 2002). An increase in cigarette smoking among women has produced lung cancer incidence rates that more closely resemble those experienced by males. The risk of developing lung cancer depends on the intensity of an individual's smoking habits (e.g., duration of habit, amount smoked, tar yield of cigarette, and filter type). Smoking cessation decreases the elevated risk by about 50%; however, former smokers still carry a greater risk of developing lung cancer than those who have never smoked.

Several occupational exposures have been identified as playing a role in the development of lung cancer. For example, workplace exposure to asbestos is an established risk factor for this disease (ACS 2002). Underground miners exposed to radon and uranium are also at an increased risk for developing lung cancer (ACS 2002; Samet and Eradze 2000). Other occupations potentially associated with this cancer include chemical workers, talc miners and millers, paper and pulp workers, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers. In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer. Occupational exposure to these compounds in conjunction with cigarette smoking can dramatically increase the risk of developing lung cancer (Blot and Fraumeni 1996).

1. Age and Gender

Of the 270 individuals diagnosed with lung and bronchus cancer in Belmont during 1982–1999, the average age at diagnosis was 70 years. Less than one percent of these individuals (n = 2) were under the age of 40 at the time of diagnosis. Overall, during the 18-year time period 1982–1999, both males and females experienced decreased rates of lung and bronchus cancer compared to the state rates, with the rate among males statistically significant. Males in all age groups were diagnosed with lung and bronchus cancer less often than expected in Belmont during 1982–1999. Similar trends were observed among females in most age groups.

In Cambridge, the average age at diagnosis for the 799 individuals diagnosed with lung and bronchus cancer during 1982–1999 was 67 years. Less than one percent of these individuals (n = 6) were under the age of 40 at the time of diagnosis. As described previously, both males and females experienced statistically significantly lower-than-expected rates of lung and bronchus cancer in Cambridge during this time period. Incidence rates of lung and bronchus cancer were about as expected or lower than expected for males and females in all age groups evaluated.

The average age at diagnosis for individuals with lung and bronchus cancer in Arlington was 69 years. Less than one percent of these individuals (n = 5) were under the age of 40 at the time of diagnosis. Review of age-group-specific SIRs revealed that males and females in all age groups were diagnosed with lung and bronchus cancer either about at or below expected rates.

In Watertown, the average age of individuals diagnosed with lung and bronchus cancer during 1982–1999 was 70 years. Less than one percent of those diagnosed were under the age of 40 at the time of diagnosis. Males in most age groups were diagnosed with lung and bronchus cancer about as often as expected or less often than expected; however, individuals aged 75–84 experienced a slight elevation in the incidence of this cancer type. The overall incidence rate among males was lower than expected. Among females, individuals in all age groups were diagnosed approximately at or below the rates expected.

2. Histology

Of the 228 lung and bronchus cancer diagnoses in Belmont with a specific histology classification, 48% were diagnosed as adenocarcinomas, 27% were squamous cell carcinomas, 20% were small cell cancers, and 5% were large cell carcinomas. This pattern is generally consistent with the distribution of histology types seen in the general population with the exception that adenocarcinomas comprised a somewhat larger proportion of all diagnoses while large cell carcinomas comprised a somewhat smaller proportion of all diagnoses.

In Cambridge, 667 lung and bronchus cancer individuals were diagnosed with one of the four major subtypes of this disease. Of these, about 43% were adenocarcinomas, 30% were squamous cell carcinomas, 19% were small cell carcinomas, and 8% were large cell carcinomas.

The observed distribution is consistent with established prevalence patterns of disease for lung and bronchus cancer in the general population.

Among the 445 lung and bronchus cancer diagnoses in Arlington that could be classified as one of the four major subtypes, 36% were adenocarcinomas, 29% were squamous cell carcinomas, 21% were small cell carcinomas, and 14% were large cell carcinomas. This pattern is comparable to that seen in the general population.

Of the 354 lung and bronchus cancer diagnoses in Watertown with a specific histology classification, 40% were diagnosed as adenocarcinomas, 32% were squamous cell carcinomas, 18% were small cell carcinomas, and 9% were large cell carcinomas. This distribution is consistent with that observed in the general population.

3. Smoking History

Of the 270 individuals diagnosed with lung and bronchus cancer in Belmont during 1982–1999, 85% (n = 230) reported being current or formers smokers at the time of diagnosis. Eight percent (n = 22) were nonsmokers and smoking history was unknown for the remaining 7% (n = 18). In comparison, 79% of individuals diagnosed with lung and bronchus cancer in Massachusetts as a whole during this time period reported being current or former smokers, 7% were nonsmokers, and smoking history was unknown for 14%. Review of this information suggests that smoking likely played a role in the incidence of lung and bronchus cancer in Belmont.

The majority of individuals diagnosed with lung and bronchus cancer in Cambridge reported being current or former smokers at the time of diagnosis (83%, n = 663). Five percent (n = 42) were nonsmokers, and smoking history was unknown for 12% of the diagnoses (n = 94). Again, it is likely that smoking played a role in the pattern of lung and bronchus cancer in Cambridge.

Of the 544 individuals diagnosed with lung and bronchus cancer in Arlington during 1982–1999, the majority (84%, n = 459) were current or former smokers at the time of diagnosis. Seven percent (n = 39) were nonsmokers and smoking history was unknown for the remaining 8% (n = 46).

Of the 423 individuals diagnosed with lung and bronchus cancer in Watertown during 1982–1999, 79% (n = 335) reported being current or former smokers at the time of diagnosis. Ten percent (n = 44) were nonsmokers, and smoking history was unknown for the remainder (n = 44).

4. Occupation

The majority of individuals diagnosed with lung and bronchus cancer in Belmont did not indicate working in jobs likely to have been a factor in their development of cancer. However, 14 individuals (5%) reported either a history of asbestos exposure or occupations where exposures to asbestos or other chemical compounds possibly associated with lung and bronchus cancer may have been possible. Occupation was reported as retired, unknown, or at home for almost half (46%, n = 125) of the individuals.

Seven percent (n = 56) of the individuals diagnosed with lung and bronchus cancer in Cambridge reported a history of asbestos exposure or working in jobs where occupational exposures possibly related to an increased risk of this disease could have been possible. Occupation was unknown or reported as retired or at home for 43% (n = 342) of those diagnosed.

Review of job information for individuals diagnosed with lung and bronchus cancer in Arlington during 1982–1999 revealed that occupational or other exposures possibly associated with lung and bronchus cancer could have been possible for at least 35 individuals (7%). However, occupation was unknown or reported as retired or at home for 37% of the individuals (n = 199).

In Watertown, 21 individuals diagnosed with lung and bronchus cancer during 1982–1999 (5%) reported working in jobs possibly associated with an increased risk of this cancer type or a history of exposure to asbestos, radiation, or other substances that may have possibly played a role in their developing cancer. However, occupation was unknown or reported as retired or at home for almost half (48%, n = 204) of those diagnosed. Therefore, it is difficult to determine the role that occupation may have played in the incidence of lung and bronchus cancer in Watertown.

E. Non-Hodgkin's Lymphoma

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. This disease is more common in men than in women and affects whites more often than African Americans or Asian Americans (ACS 2000a). The American Cancer Society estimates that approximately 53,400 Americans will be diagnosed with NHL in 2003, making it the sixth most common cancer in the United States among both men and women, excluding nonmelanoma skin cancers (ACS 2003). 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 2000a); 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).

1. Age and Gender

The average age at diagnosis for individuals diagnosed with NHL in Belmont during 1982–1999 was 66 years, which is generally consistent with that seen in the general population. Review of age-group-specific SIRs revealed slightly different trends among males and females. While males in all age groups were diagnosed with NHL at or below expected rates, resulting in an overall incidence rate that was borderline statistically significantly lower than expected, a slight elevation in the incidence of NHL was noted among females aged 20–44. Females in other age groups experienced NHL about as often as expected or less often than expected.

In Cambridge, the average age at diagnosis for individuals diagnosed with NHL during 1982–1999 was 59 years. As described previously, incidence rates for NHL were statistically significantly lower than expected for males and females combined and for males when evaluated separately by gender. In addition, incidence rates were borderline statistically significantly lower than expected for females. Review of age-group-specific SIRs revealed that both males and

females in all age groups evaluated experienced NHL about as often as expected or less often than expected.

Among the 161 individuals diagnosed with NHL in Arlington during 1982–1999, the average age at the time of diagnosis was 67 years. The incidence of NHL among males in Arlington was approximately at or below expected rates for most age groups. The slight elevation in NHL incidence observed among females in Arlington was primarily the result of additional diagnoses among females aged 45–64 years. Females in other age groups were diagnosed with NHL about as often as expected or less often than expected in Arlington during 1982–1999.

In Watertown, a statistically significant elevation in the incidence of NHL was noted for the 18-year time period 1982–1999. Among the 137 individuals diagnosed, the average age at diagnosis was 64 years. Among males, the elevated rate was primarily due to an increase in the number of NHL diagnoses among individuals aged 20–44 and 45–64. Among females, the elevated rate was primarily the result of additional diagnoses among individuals aged 65–74 and 75–84.

2. Occupation

Review of occupational information for individuals diagnosed with NHL in Belmont revealed that three 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 almost 75% of individuals (n = 62). Therefore, it is difficult to assess the role that occupation may have played in the incidence of NHL among residents of Belmont.

On the basis of a review of job title information as reported to the MCR for individuals in Cambridge diagnosed with NHL during 1982–1999, occupational exposure do not appear likely for the majority of those diagnosed. However, one individual reported working in an occupation in which exposure to pesticides may have been possible. Occupation was unknown or reported as retired or at home for 37% (n = 68) of the individuals.

Review of occupational information for individuals diagnosed with NHL in Arlington revealed that occupational exposures possibly associated with an increased risk of NHL may have been possible for five individuals (3%). Occupation was unknown or reported as retired or at home for 43% of those diagnosed (n = 70).

In Watertown, three individuals reported occupations in which exposures possibly related to the development of NHL may have been possible. Again, specific job duty information was not available for these individuals. Occupation was reported as retired, unknown, or at home for half of all individuals (n = 69).

F. Pancreatic Cancer

The risk of developing pancreatic cancer increases with age, and the majority of cases occur between ages 60 and 80. Men are approximately 30% more likely to develop pancreatic cancer than are women (ACS 2000b). Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2000b). 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 2000b; 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 (ACS 2000b; 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).

1. *Age and Gender*

The average age at diagnosis for individuals with pancreatic cancer in Belmont during 1982–1999 was 72 years, consistent with established prevalence patterns of this disease. Eighty-two percent of those diagnosed were age 60 years or older at the time of diagnosis. Overall, females were diagnosed slightly more often than expected while males were diagnosed less often than expected. Further review of this data by age indicated that females and males in each age group experienced pancreatic cancer approximately at or below the expected rates.

In Cambridge, the average age of diagnosis for individuals diagnosed with pancreatic cancer during 1982–1999 was 62 years. Eighty-one percent (n = 113) were age 60 years or older at the time of diagnosis. The overall elevation in pancreatic cancer incidence observed among males in Cambridge was primarily the result of an increased number of diagnoses among males aged 45–64 years. Females in each age group were diagnosed with pancreatic cancer about as often as expected or less often than expected.

Among the 102 individuals diagnosed with pancreatic cancer in Arlington during 1982–1999, the average age of diagnosis was 73 years. The majority of those diagnosed were age 60 years or older at the time of diagnosis (91%, n = 93). Review of age group-specific SIRs revealed that males aged 45–64 years were diagnosed with this cancer type about half as often as expected while males aged 65–74 years were diagnosed more often than expected. Females in most age groups were diagnosed with pancreatic cancer at lower-than-expected rates, with the exception of females aged 75–84 years, who experienced an elevation in the incidence of this cancer type.

In Watertown, the average age of diagnosis for individuals diagnosed with pancreatic cancer during 1982–1999 was 71 years. Eighty-five percent of these individuals were age 60 years or older at the time of diagnosis. Overall, males were diagnosed slightly more often than expected. Males in most age groups were diagnosed about as often as expected or less often than expected; however, males aged 85 years and older were diagnosed more often than expected. Incidence rates were elevated among females aged 45–64 years. However, females aged 45–64 and 65–74 were diagnosed with pancreatic cancer less often than expected resulting in an overall incidence rate that was lower than expected among females.

2. Smoking History

Of the 57 individuals diagnosed with pancreatic cancer in Belmont during 1982–1999, 28% (n = 16) reported being current or former smokers at the time of diagnosis. Another 28% (n = 19) were nonsmokers, and smoking history was unknown for 44% (n =25). In the state as a whole, 32% of those diagnosed with pancreatic cancer during 1982–1999 were current or former smokers at the time of diagnosis, 43% were nonsmokers, and smoking history was unknown for 26%.

In Cambridge, 46% (n = 64) of those diagnosed with pancreatic cancer during 1982–1999 reported being current or former smokers at the time of diagnosis. Thirty-one percent (n = 43) were nonsmokers, and smoking history was unknown for 23% (n = 32).

Forty-six percent of the individuals (n = 44) diagnosed with pancreatic cancer in Arlington during 1982–1999 reported being current or former smokers at the time of diagnosis. Forty-three percent (n = 47) were nonsmokers, and smoking history was unknown for the remaining 11% (n = 11). Review of this information suggests that smoking likely played a role in the overall incidence of pancreatic cancer in Arlington.

In Watertown, 45% of those diagnosed with pancreatic cancer during 1982–1999 (n = 30) reported being current or former smokers at the time of diagnosis. Fifteen percent (n = 23) were nonsmokers, and smoking history was unknown for the remaining 20% (n = 13).

3. Occupation

None of the individuals diagnosed with pancreatic cancer in Belmont during 1982–1999 indicated working in a job likely to be associated with occupational exposures related to an increased risk of pancreatic cancer. However, occupation was reported as unknown, retired, or at home for almost half (47%, n = 27) of these individuals. Therefore, it is difficult to assess the role that occupation may have played in the pattern of pancreatic cancer in Belmont.

Two individuals diagnosed with pancreatic cancer in Cambridge during 1982–1999 may have had occupational exposures possibly associated with pancreatic cancer. No other jobs likely to

be associated with this cancer type were reported. However, occupation was unknown or reported as retired or at home for almost half (49%, n = 68) of these individuals.

On the basis of a review of occupational information as reported to the MCR, none of the individuals diagnosed with pancreatic cancer in Arlington during 1982–1999 worked jobs likely to be associated with an increased risk of this cancer type. However, occupation was unknown or reported as retired or at home for more than half of those diagnosed (54%, n = 55).

No jobs likely to be associated with occupational exposures related to the development of pancreatic cancer were reported among those diagnosed with this cancer type in Watertown. Occupation was reported as unknown, retired, or at home for almost half of those diagnosed (48%, n = 32).

VI. MDEP OIL AND HAZARDOUS MATERIAL RELEASES

In 1983, the Massachusetts legislature established a statewide hazardous waste site cleanup program (the state Superfund program) under Chapter 21E of Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000). Under this legislation, the Massachusetts Department of Environmental Protection (MDEP) administers investigation and cleanup of hazardous material and oil release sites, known as "21E sites," in the Commonwealth. MDPH reviewed available information regarding these releases to determine the possibility that known sources of potential environmental exposures could have played a role in the overall incidence of cancer in Belmont, Cambridge, Arlington, and Watertown.

The 21E sites are characterized by one or more releases of oil or other hazardous material. Releases can result from a variety of sources, including oil trucks, underground storage tanks, and aboveground storage drums. Releases vary widely with respect to materials involved, the relative amount of materials released, and the geographic extent of contamination. Depending on the relative severity of the release, the deadline for reporting a release to MDEP is either two hours, 72 hours, or 120 days.

The MDEP Bureau of Waste Site Cleanup has information on hazardous material and oil releases, including assessment and remedial response measures, for 1977 to the present (MDEP

2003). MDPH obtained the most recent information regarding all hazardous material and/or oil releases (approximately 1,800 records) located in Belmont, Arlington, Cambridge, and Watertown. The high number of releases in the study area precluded individual examination of each release in relation to patterns of cancer incidence. Therefore, MDPH focused the analysis on only those releases categorized by 2-hour or 72-hour reporting categories and excluded releases categorized by 120-day reporting notification and releases where reporting category information was unavailable.

Conditions requiring notification to MDEP within two hours of obtaining knowledge of the spill may include, but are not limited to the following: the release results in oil or hazardous materials found in a private drinking water well in concentrations greater than a reportable quantity for groundwater used as drinking water; the release poses an imminent hazard, including explosion, fire, public safety and serious and immediate public health and environmental hazards; and/or the release is indirectly discharged to the sanitary sewage system (310 CMR, 40.0311).

Conditions requiring notification to MDEP within 72 hours of obtaining knowledge of the spill may include, but are not limited to the following: the release is within a 400-foot radius of a public supply well; the release is within 500-feet of a private water supply well; and/or the release results in contaminated groundwater with the level of total volatile organic compounds (VOC) exceeding 5 milligrams per liter (mg/L) and the contamination exists within 30 feet of a school or residential structure at a depth less than 15 feet below the surface of the ground (310 CMR, 40.0313).

MDEP reporting categories are based upon broad categories of negative environmental impact. In general, acute risks to human health are considered in assigning categories to MDEP 21E releases, but the categories are nonspecific with respect to particular health outcomes, especially chronic health outcomes such as cancer. For example, a release may be categorized as a 2-hour release because of its potential severe impact on the local environment, but the release may pose little or no threat of human exposure to contaminants. Conversely, a relatively small amount of a contaminant might have potential impacts to human health if the opportunity exists for long-term chronic exposure, but in the short term may be considered environmentally benign.

Hazardous material and oil releases represent potential sources of exposure to contamination. It is not possible to determine whether individuals residing in the study area were actually exposed to contaminants without detailed information about contaminant movement through the environment, the population at risk of exposure, a location of actual human contact with the contaminant, and evidence that the contaminant actually entered the body of persons at risk of exposure through ingestion, dermal absorption, or inhalation.

Using a geographic information system, MDPH mapped the approximate location of 2-hour and 72-hour releases for which sufficient address information was available (ESRI 2002). According to the most current information, from 1991 to 2003, 28 releases were reported in the town of Belmont; 179 releases were reported in Cambridge; 60 releases were reported in Arlington; and 74 releases were reported in Watertown. The majority of these releases could be mapped to an address in one of the four towns (see Figure 2); however, approximately 3% of the releases (n = 12) could not be mapped because sufficient address information was not available.

The majority of the 341 releases reported (72%) involved petroleum-based oil (e.g., gasoline, fuel oil, waste oil). In addition, 13% involved a combination of oil and some other material (either known or unknown). Type of material was unknown for 22 (6%) of the releases. Information specific to each release is provided in Table 15.

As discussed in the next section, the pattern of cancer in Belmont, Cambridge, Arlington, and Watertown was reviewed in relation to these potential sources of environmental exposures.

VII. ANALYSIS OF GEOGRAPHIC DISTRIBUTION OF CANCER INCIDENCE

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

atypical pattern of any one type was occurring. 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). In addition, cancers that may be associated with specific environmental exposures of concern were also evaluated geographically to determine whether any atypical patterns of cases exist that might suggest an association with an environmental factor.

Review of the geographic distribution of cancer for the years 1982–1999 in Belmont revealed a small concentration of leukemia diagnoses in the southeastern corner of the town (i.e., CT 3573). This was the census tract with a statistically significant elevation in the incidence of leukemia during 1994–1999, and most of the diagnoses observed in this concentration occurred during this time period. However, further review of specific case information for these individuals revealed a variety of subtypes of leukemia diagnosed among individuals in this area, indicating the occurrence of different diseases. In addition, the available information indicated that the diagnosis of leukemia was not the first cancer diagnosis for some of these individuals. If these individuals received ionizing radiation therapy to treat another cancer, then that could have played a role in their subsequent development of leukemia because ionizing radiation is a known risk factor for leukemia. There were no reported hazardous material or oil releases in the immediate vicinity. On the basis of this information, it appears unlikely that a common environmental factor contributed to the concentration of leukemia diagnoses observed in Belmont CT 3573.

No other unusual spatial patterns or concentrations of diagnoses at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents were observed. 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. Moreover, although slight elevations in the incidence of some cancer types were noted in Belmont during one or more time periods evaluated, in general, the geographic distribution of diagnoses for these cancer types seemed to coincide closely with the pattern of population and cases did not appear to be concentrated in any one area of the town. In addition, no apparent concentrations of cancer

diagnoses (of any type) were observed in the vicinity of the Cambridge Plating site or in relation to any other potential sources of environmental contamination (i.e., 21E sites).

Although there was a statistically significant elevation in the incidence of leukemia in Belmont CT 3578 during 1988–1993, the cases were fairly evenly distributed throughout the census tract and were not located in any one neighborhood. A similar pattern was observed for females diagnosed with pancreatic cancer, which was statistically significantly elevated in this census tract during 1982–1987. Females in Belmont CT 3577 experienced a statistically significant elevation in the incidence of NHL during 1982–1999 and 1994–1999. Most of these individuals resided in the southern part of this census tract consistent with residential patterns in this area of Belmont.

In general, review of the geographic distribution of cancer in Cambridge, Arlington, and Watertown revealed no apparent spatial patterns at the neighborhood level that could not be attributed to such factors as areas of higher population density (e.g., the presence of multiunit housing complexes). However, a small concentration of leukemia diagnoses was observed near the center of Arlington. Specifically, six individuals (ages 15–77) in a high-density residential neighborhood were diagnosed with leukemia between 1994 and 1998. Although a fuel oil/gasoline spill resulting from an underground storage tank in the vicinity of these residences was reported to MDEP in 1994, this release is unlikely to be related to these diagnoses because of the low likelihood of exposure to residents in this area. Moreover, a variety of histology types were represented among these individuals, indicating the occurrence of different diseases.

A small concentration of individuals diagnosed with NHL was also noted in Watertown. On the basis of a review of the available risk factor information for these individuals, including residential histories, MDPH previously concluded that the occurrence of NHL among these individuals did not suggest any single factor (environmental or nonenvironmental) that might explain the observed distribution (ATSDR 1996, 2000a, 2002).

A statistically significant elevation in the incidence of pancreatic cancer among males was noted in Cambridge CT 3549, located in the western part of Cambridge adjacent to Belmont, during 1982–1999. Review of the geographic distribution of cases revealed that these individuals resided in the eastern part of the census tract, where population density is greatest. In Arlington,

statistically significant elevations were observed for liver cancer among males and females combined and for pancreatic cancer among males during 1982–1987. The majority of those diagnosed with liver cancer resided in the eastern part of Arlington, but they were not concentrated in any one census tract or in any one neighborhood. A small concentration of males with pancreatic cancer was noted in the western part of Arlington near the Lexington border. However, these individuals lived in a high-density residential neighborhood and review of case-specific information did not suggest a common factor (environmental or nonenvironmental) related to these diagnoses. A statistically significant elevation in the incidence of NHL was observed in Watertown during 1982–1999. In addition, males in Watertown experienced statistically significant elevations in the incidence of liver cancer during 1982–1999 and in the incidence of kidney cancer during 1988–1993. With the exception of a small concentration of individuals diagnosed with NHL, discussed previously, review of the geographic distribution of these cases revealed no apparent spatial concentrations of diagnoses; in general, the geographic distribution was consistent with the population density of Watertown.

As noted previously, an elevation in the incidence of leukemia was observed among females aged 0–19 years in Arlington (9 diagnoses observed vs. 2.6 expected). With one exception, the nine diagnoses did not appear to be unusually concentrated in time or space. However, two females (ages 3 and 4) who were diagnosed with leukemia in 1998 and 1999 resided in relatively close proximity to each other. The neighborhood is a high-density residential area with no known sources of environmental contamination. Therefore, the possible role of an environmental factor is unlikely. Review of more recent data available from the MCR indicates three additional childhood leukemia diagnoses in Arlington since 1999.[3] However, these individuals did not reside near each other or near the two children discussed above.

VIII. DISCUSSION

The rates of cancer types evaluated in the community surrounding Cambridge Plating (i.e., CTs 3571 and 3572) were approximately near or below the rates expected based on cancer incidence

[3] Although all newly diagnosed cases of cancer are required to be reported to the MCR within six months of diagnosis (M.G.L. C.111s.111B), due to intensive efforts to ensure data quality, there is a significant lag time between diagnosis and reporting. Therefore, data for more recent years (i.e., 2000-present) cannot be considered complete.

in the state of Massachusetts as a whole for the 18-year time period 1982–1999. Similar trends were observed in the town of Belmont as a whole and in the surrounding communities of Cambridge, Arlington, and Watertown. That is, with some exceptions, cancer incidence in these towns during the 18-year time period 1982–1999 and smaller time periods evaluated was approximately at or near expected rates for the six cancer types evaluated in this report.

Some statistically significant elevations were observed. In Belmont, the incidence of leukemia was statistically significantly elevated in CT 3573 during 1994–1999 and CT 3578 during 1988–1993. Also, females in Belmont CT 3577 experienced a statistically significant elevation in the incidence of NHL during 1994–1999 and the overall time period 1982–1999. Pancreatic cancer was also statistically significantly elevated among females in Belmont CT 3578 during 1982–1987. In Cambridge CT 3549, males were diagnosed with pancreatic cancer statistically significantly more often than expected. Townwide incidence ratios for liver cancer among males and females combined and for pancreatic cancer among males were statistically significantly elevated in Arlington during 1982–1987. Finally, there was a statistically significant elevation in the incidence of NHL in Watertown during 1982–1999. Males in Watertown also experienced statistically significant elevations in the incidence of liver cancer during 1982–1999 and kidney cancer during 1988–1993. An elevation in the incidence of leukemia was also observed among females aged 0–19 years in Arlington. Based on a review of the geographic distribution of diagnoses and available environmental information, it is unlikely that environmental factors played a role in the occurrence of leukemia among these individuals.

Available risk factor information for individuals diagnosed with cancer in Belmont, Cambridge, Arlington, and Watertown was compared to known or established trends to assess whether any unexpected patterns exist in the towns. In general, trends observed in Belmont and surrounding communities are similar to those seen in the general population. Review of this data suggests that smoking likely played some role in the diagnosis of some cancer types (e.g., cancers of the kidney, lung and bronchus, and pancreas) among some individuals in Belmont, Cambridge, Arlington, and Watertown. Also, occupational exposures may have been important in the development of cancer among some individuals. However, because of the large number of individuals for whom smoking history and/or occupation was unknown, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in these towns.

Finally, 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) related to the incidence of cancer in Belmont or surrounding communities.

Based on the information reviewed in this evaluation, including available environmental data regarding hazardous material and oil releases reported to MDEP, it does not appear that environmental exposures played a major role in the incidence of most cancer types in the town of Belmont or in adjacent communities (i.e., Cambridge, Arlington, and Watertown) during the 18-year time period 1982–1999.

IX. LIMITATIONS

This assessment is an investigation that analyzes descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected types of cancer is unusual. The purpose of this investigation is to evaluate the patterns of cancer in a geographical context in relation to available information about factors (including environmental factors) related to cancer to see whether further investigation seems warranted. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further public health actions may be warranted. Inherent limitations in this type of analysis and the available data make it impossible to determine the precise causal relationships or the synergistic roles that may have played a part in the development of individual cancers in these communities. Also, this type of analysis cannot determine what may have caused cancer in any one individual. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the disease that could not be evaluated in this report. It is believed that many types of cancer are related largely to behavioral factors such as cigarette smoking, alcohol consumption, and diet. Other factors associated with cancer are socioeconomic status, heredity/genetics, race, and geography. It is beyond the scope of this report to determine the causal relationship of these factors and the development of cancer in Belmont and surrounding communities.

X. CONCLUSIONS

- In general, the six cancer types evaluated in this report occurred approximately at, near, or below the expected rates for Belmont and its individual census tracts, and for the surrounding communities of Cambridge, Arlington, and Watertown during the 18-year time period 1982–1999.

- Review of the geographic distribution of individuals diagnosed with cancer in Belmont, Cambridge, Arlington, and Watertown 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 cancer (e.g., age, gender, smoking history, and occupation) suggest that the trends observed in Belmont and surrounding communities are similar to those seen in the general population. Moreover, this information suggests that smoking and, to a lesser extent, occupation, likely played some role in the incidence of some cancer types in Belmont, Cambridge, Arlington, and Watertown.

- Based on the information reviewed in this evaluation, including available environmental data regarding hazardous material and oil releases reported to MDEP, it does not appear that environmental exposures played a major role in the incidence of most cancers in the town of Belmont or in adjacent communities (i.e., Cambridge, Arlington, and Watertown) during the 18-year time period 1982–1999.

XI. RECOMMENDATIONS

- If requested, BEH's Environmental Health Education Program should work with the Belmont Health Department and the community to provide educational information and conduct outreach activities to Belmont residents about ways to reduce their risk of cancer.

- The MDPH/BEH will continue to monitor the incidence of cancer in Belmont, Cambridge, Arlington, and Watertown through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

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FIGURES

Figure 1
Area of Analysis
Arlington, Belmont, Cambridge, and Watertown, MA

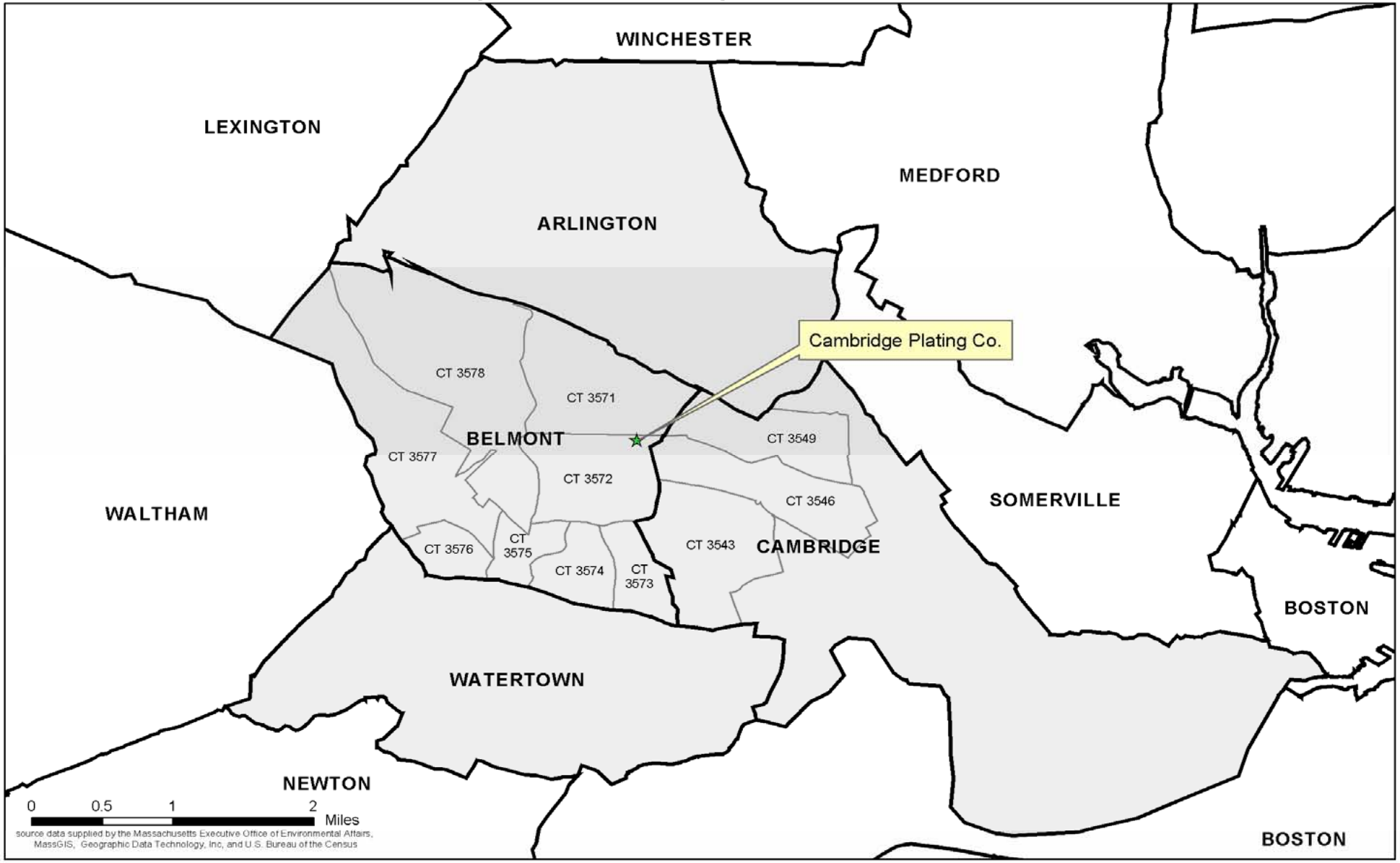
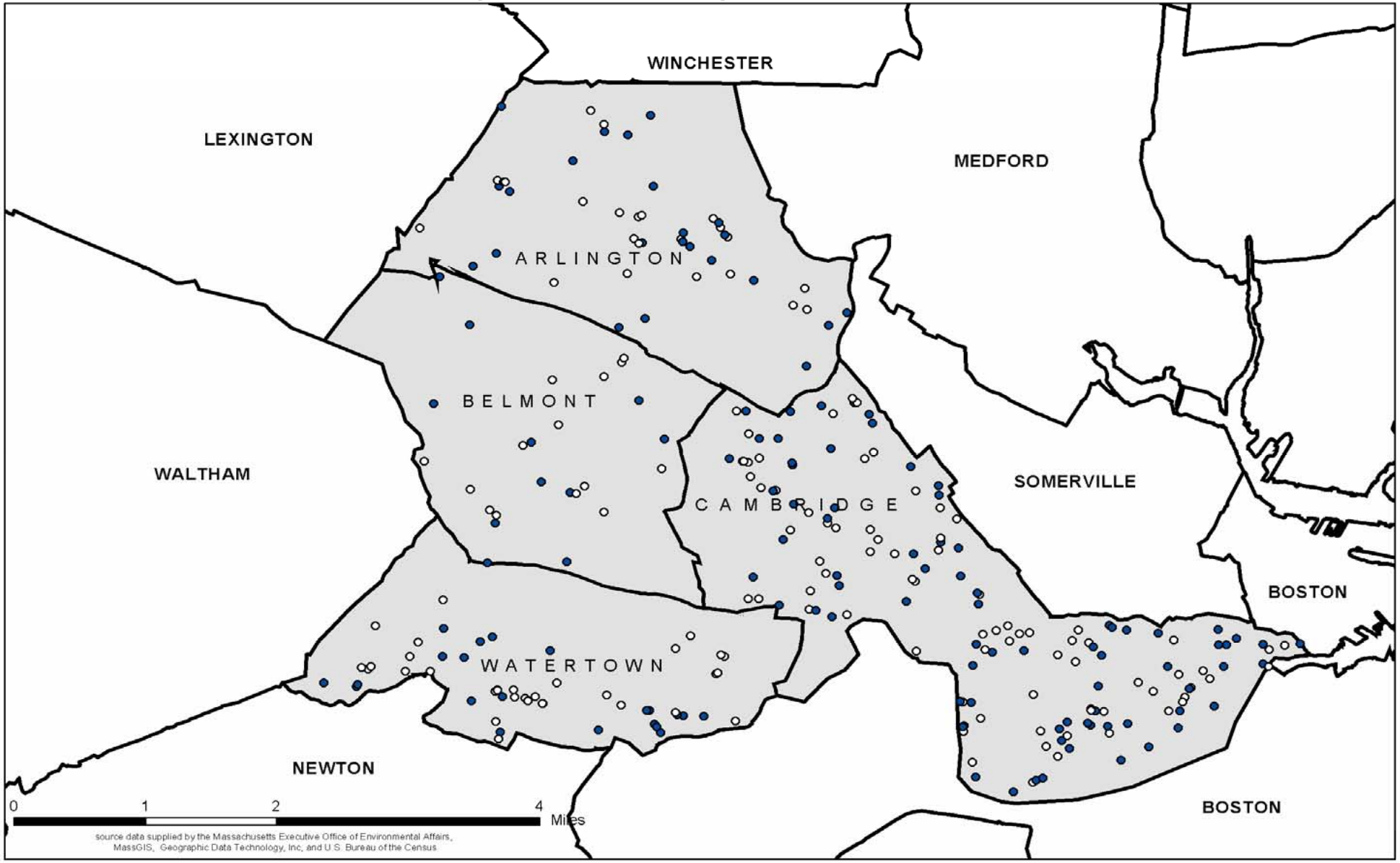


Figure 2
Locations of MDEP 21E Oil and Hazardous Material Releases
Arlington, Belmont, Cambridge, and Watertown, MA



TABLES

TABLE 1a
Kidney Cancer Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	11	12.6	88	44 -- 157	6	7.1	85	31 -- 184	5	5.5	91	29 -- 213
3572	8	7.2	111	48 -- 218	7	4.4	158	63 -- 325	1	2.8	NC	NC -- NC
3573	5	5.7	88	28 -- 204	5	3.2	155	50 -- 361	0	2.5	NC	NC -- NC
3574	3	5.0	NC	NC -- NC	3	3.0	NC	NC -- NC	0	2.0	NC	NC -- NC
3575	2	4.3	NC	NC -- NC	1	2.6	NC	NC -- NC	1	1.8	NC	NC -- NC
3576	7	5.4	129	52 -- 266	3	3.2	NC	NC -- NC	4	2.2	NC	NC -- NC
3577	6	8.2	73	27 -- 159	3	4.6	NC	NC -- NC	3	3.6	NC	NC -- NC
3578	9	9.7	93	42 -- 177	5	6.1	82	26 -- 191	4	3.6	NC	NC -- NC
City Total[†]	52	58.1	89	67 -- 117	34	34.2	99	69 -- 139	18	23.9	75	45 -- 119

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

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

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

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

TABLE 1b
Kidney Cancer Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	3	3.4	NC	NC -- NC	2	1.9	NC	NC -- NC	1	1.5	NC	NC -- NC
3572	4	1.9	NC	NC -- NC	3	1.1	NC	NC -- NC	1	0.8	NC	NC -- NC
3573	1	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
3574	3	1.4	NC	NC -- NC	3	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
3575	0	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
3576	3	1.5	NC	NC -- NC	0	0.8	NC	NC -- NC	3	0.6	NC	NC -- NC
3577	1	2.3	NC	NC -- NC	1	1.2	NC	NC -- NC	0	1.1	NC	NC -- NC
3578	5	2.6	NC	NC -- NC	4	1.6	NC	NC -- NC	1	1.0	NC	NC -- NC
City Total[†]	21	16.0	131	81 -- 201	15	9.0	167	93 -- 275	6	7.0	85	31 -- 186

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

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

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

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

TABLE 1c
Kidney Cancer Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	3	4.4	NC	NC -- NC	1	2.5	NC	NC -- NC	2	1.9	NC	NC -- NC
3572	3	2.5	NC	NC -- NC	3	1.6	NC	NC -- NC	0	1.0	NC	NC -- NC
3573	3	2.0	NC	NC -- NC	3	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC
3574	0	1.8	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.7	NC	NC -- NC
3575	2	1.5	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.6	NC	NC -- NC
3576	2	1.9	NC	NC -- NC	1	1.1	NC	NC -- NC	1	0.8	NC	NC -- NC
3577	1	2.8	NC	NC -- NC	0	1.6	NC	NC -- NC	1	1.2	NC	NC -- NC
3578	4	3.4	NC	NC -- NC	1	2.2	NC	NC -- NC	3	1.2	NC	NC -- NC
City Total[†]	18	20.3	89	52 -- 140	10	12.2	82	39 -- 151	8	8.1	98	42 -- 194

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

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

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

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

TABLE 1d
Kidney Cancer Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	5	4.5	112	36 -- 261	3	2.6	NC	NC -- NC	2	1.9	NC	NC -- NC
3572	1	2.6	NC	NC -- NC	1	1.6	NC	NC -- NC	0	1.0	NC	NC -- NC
3573	1	2.0	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC
3574	0	1.9	NC	NC -- NC	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC
3575	0	1.6	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.6	NC	NC -- NC
3576	2	2.0	NC	NC -- NC	2	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC
3577	4	2.9	NC	NC -- NC	2	1.6	NC	NC -- NC	2	1.3	NC	NC -- NC
3578	0	3.6	NC	NC -- NC	0	2.2	NC	NC -- NC	0	1.3	NC	NC -- NC
City Total[†]	13	21.1	62	33 -- 105	9	12.5	72	33 -- 137	4	8.6	NC	NC -- NC

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

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

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

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

TABLE 2a
Leukemia Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	13	10.7	121	64 -- 207	6	5.6	108	39 -- 234	7	5.2	136	54 -- 279
3572	6	6.1	98	36 -- 213	4	3.5	NC	NC -- NC	2	2.7	NC	NC -- NC
3573	8	5.1	158	68 -- 311	4	2.6	NC	NC -- NC	4	2.4	NC	NC -- NC
3574	6	4.3	139	51 -- 303	2	2.4	NC	NC -- NC	4	1.9	NC	NC -- NC
3575	3	3.8	NC	NC -- NC	2	2.1	NC	NC -- NC	1	1.8	NC	NC -- NC
3576	2	4.7	NC	NC -- NC	1	2.6	NC	NC -- NC	1	2.2	NC	NC -- NC
3577	9	7.5	120	55 -- 228	2	3.7	NC	NC -- NC	7	3.8	177	71 -- 365
3578	10	8.0	125	60 -- 231	5	4.7	107	34 -- 249	5	3.3	152	49 -- 354
City Total[†]	58	50.3	115	88 -- 149	26	27.1	96	63 -- 141	32	23.2	138	94 -- 195

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

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

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

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

TABLE 2b
Leukemia Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	3	3.3	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.6	NC	NC -- NC
3572	1	1.9	NC	NC -- NC	0	1.1	NC	NC -- NC	1	0.8	NC	NC -- NC
3573	0	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
3574	3	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	2	0.7	NC	NC -- NC
3575	0	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
3576	1	1.5	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
3577	3	2.4	NC	NC -- NC	0	1.2	NC	NC -- NC	3	1.2	NC	NC -- NC
3578	1	2.4	NC	NC -- NC	1	1.4	NC	NC -- NC	0	1.0	NC	NC -- NC
City Total[†]	12	16.0	75	39 -- 131	4	8.5	NC	NC -- NC	8	7.5	107	46 -- 210

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

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

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

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

TABLE 2c
Leukemia Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	5	3.2	156	50 -- 363	2	1.7	NC	NC -- NC	3	1.5	NC	NC -- NC
3572	1	1.9	NC	NC -- NC	1	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC
3573	1	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.7	NC	NC -- NC
3574	1	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
3575	2	1.2	NC	NC -- NC	1	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC
3576	1	1.4	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
3577	4	2.3	NC	NC -- NC	1	1.1	NC	NC -- NC	3	1.1	NC	NC -- NC
3578	7	2.4	290	* 116 -- 598	4	1.4	NC	NC -- NC	3	1.0	NC	NC -- NC
City Total[†]	22	15.2	145	91 -- 220	11	8.3	133	66 -- 237	11	6.9	160	80 -- 287

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

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

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

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

TABLE 2d
Leukemia Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	5	3.9	127	41 -- 297	2	2.1	NC	NC -- NC	3	1.9	NC	NC -- NC
3572	4	2.3	NC	NC -- NC	3	1.3	NC	NC -- NC	1	1.0	NC	NC -- NC
3573	7	1.8	383	* 153 -- 789	3	0.9	NC	NC -- NC	4	0.9	NC	NC -- NC
3574	2	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
3575	1	1.4	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.7	NC	NC -- NC
3576	0	1.8	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
3577	2	2.7	NC	NC -- NC	1	1.3	NC	NC -- NC	1	1.4	NC	NC -- NC
3578	2	3.0	NC	NC -- NC	0	1.7	NC	NC -- NC	2	1.3	NC	NC -- NC
City Total[†]	24	18.6	129	83 -- 192	11	10.0	110	55 -- 197	13	8.7	150	80 -- 257

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

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

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

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

TABLE 3a
Liver Cancer Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	5	3.0	168	54 -- 393	3	2.0	NC	NC -- NC	2	1.0	NC	NC -- NC
3572	1	1.7	NC	NC -- NC	0	1.2	NC	NC -- NC	1	0.5	NC	NC -- NC
3573	4	1.3	NC	NC -- NC	3	0.9	NC	NC -- NC	1	0.4	NC	NC -- NC
3574	0	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
3575	1	1.0	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
3576	1	1.3	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.4	NC	NC -- NC
3577	3	2.0	NC	NC -- NC	2	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC
3578	1	2.3	NC	NC -- NC	1	1.7	NC	NC -- NC	0	0.6	NC	NC -- NC
City Total[†]	16	13.8	116	66 -- 189	11	9.6	115	57 -- 205	5	4.2	120	39 -- 280

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

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

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

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

TABLE 3b
Liver Cancer Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC
3572	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3573	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC	1	0.1	NC	NC -- NC
3574	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
3575	1	0.3	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
3576	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
3577	1	0.5	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
3578	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total[†]	4	3.5	NC	NC -- NC	3	2.3	NC	NC -- NC	1	1.2	NC	NC -- NC

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

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

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

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

TABLE 3c
Liver Cancer Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	1	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.3	NC	NC -- NC
3572	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
3573	2	0.4	NC	NC -- NC	2	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3574	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3575	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
3576	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3577	2	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC	1	0.2	NC	NC -- NC
3578	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total[†]	6	4.3	141	51 -- 306	4	3.0	NC	NC -- NC	2	1.2	NC	NC -- NC

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

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

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

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

TABLE 3d
Liver Cancer Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	3	1.2	NC	NC -- NC	2	0.8	NC	NC -- NC	1	0.4	NC	NC -- NC
3572	1	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	1	0.2	NC	NC -- NC
3573	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
3574	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
3575	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3576	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
3577	0	0.8	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC
3578	0	1.0	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total[†]	6	5.7	105	38 -- 228	4	4.1	NC	NC -- NC	2	1.7	NC	NC -- NC

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

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

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

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

TABLE 4a
Lung and Bronchus Cancer Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total						Males						Females					
	Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI	
3571	70	82.9	84		66	107	29	46.4	63	*	42	90	41	36.6	112		80	152
3572	31	46.2	67	*	46	95	18	27.8	65		38	103	13	18.5	70		37	120
3573	22	36.3	61	*	38	92	11	20.2	55	*	27	98	11	16.2	68		34	122
3574	20	31.7	63	*	38	97	13	18.7	70		37	119	7	13.1	54		21	110
3575	27	27.4	98		65	143	15	15.9	94		53	155	12	11.5	104		54	183
3576	23	35.0	66	*	42	99	14	20.3	69		38	116	9	14.7	61		28	117
3577	43	52.7	82		59	110	19	29.3	65		39	101	24	23.3	103		66	153
3578	33	63.0	52	*	36	74	13	39.0	33	*	18	57	20	24.0	83		51	129
City Total[†]	270	375.3	72	*	64	81	133	217.6	61	*	51	72	137	157.7	87		73	103

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

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

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

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

TABLE 4b
Lung and Bronchus Cancer Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total						Males						Females					
	Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI		
3571	19	26.0	73	44	--	114	7	16.1	43 *	17	--	90	12	9.9	122	63	--	212
3572	10	14.3	70	33	--	128	7	9.2	76	31	--	157	3	5.1	NC	NC	--	NC
3573	4	12.5	NC	NC	--	NC	3	7.5	NC	NC	--	NC	1	5.0	NC	NC	--	NC
3574	4	10.7	NC	NC	--	NC	1	6.7	NC	NC	--	NC	3	3.9	NC	NC	--	NC
3575	8	9.3	86	37	--	169	4	5.8	NC	NC	--	NC	4	3.5	NC	NC	--	NC
3576	10	10.9	92	44	--	169	8	6.8	117	50	--	230	2	4.1	NC	NC	--	NC
3577	8	16.7	48 *	21	--	94	5	10.0	50	16	--	116	3	6.7	NC	NC	--	NC
3578	13	19.6	66	35	--	113	5	12.9	39 *	12	--	90	8	6.7	119	51	--	235
City Total†	76	119.9	63 *	50	--	79	40	75.0	53 *	38	--	73	36	45.0	80	56	--	111

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

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

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

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

TABLE 4c
Lung and Bronchus Cancer Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
3571	30	27.5	109	74	-- 156	13	15.4	84	45	-- 144	17	12.1	141	82	-- 226
3572	12	15.4	78	40	-- 136	6	9.3	64	23	-- 140	6	6.1	98	36	-- 213
3573	10	12.1	83	40	-- 152	4	6.8	NC	NC	-- NC	6	5.4	112	41	-- 244
3574	9	10.6	85	39	-- 161	6	6.3	96	35	-- 208	3	4.3	NC	NC	-- NC
3575	12	9.1	131	68	-- 229	8	5.3	150	64	-- 295	4	3.8	NC	NC	-- NC
3576	8	11.6	69	30	-- 135	6	6.8	88	32	-- 192	2	4.9	NC	NC	-- NC
3577	22	17.4	126	79	-- 191	9	9.8	92	42	-- 175	13	7.6	170	91	-- 291
3578	7	21.1	33 *	13	-- 68	4	13.1	NC	NC	-- NC	3	8.0	NC	NC	-- NC
City Total[†]	110	124.9	88	72	-- 106	56	72.8	77	58	-- 100	54	52.1	104	78	-- 135

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

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

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

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

TABLE 4d
Lung and Bronchus Cancer Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total						Males						Females					
	Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI	
3571	21	28.2	75		46 --	114	9	14.8	61		28 --	116	12	13.4	90		46 --	157
3572	9	15.8	57		26 --	108	5	8.9	56		18 --	132	4	6.9	NC		NC --	NC
3573	8	12.0	66		29 --	131	4	6.2	NC		NC --	NC	4	5.8	NC		NC --	NC
3574	7	11.1	63		25 --	130	6	6.2	96		35 --	209	1	4.9	NC		NC --	NC
3575	7	9.5	74		30 --	152	3	5.1	NC		NC --	NC	4	4.3	NC		NC --	NC
3576	5	11.9	42	*	14 --	98	0	6.4	NC		NC --	NC	5	5.5	91		29 --	213
3577	13	18.1	72		38 --	123	5	9.1	55		18 --	129	8	9.0	89		38 --	175
3578	13	21.7	60		32 --	102	4	12.5	NC		NC --	NC	9	9.2	98		45 --	186
City Total[†]	84	128.3	65	*	52 --	81	37	69.2	53	*	38 --	74	47	59.1	80		58 --	106

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

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

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

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

TABLE 5a
Non-Hodgkin's Lymphoma Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	20	19.7	102	62 -- 157	7	9.0	77	31 -- 160	13	10.6	122	65 -- 209
3572	4	11.0	NC	NC -- NC	3	5.7	NC	NC -- NC	1	5.3	NC	NC -- NC
3573	5	9.3	54	17 -- 125	1	4.4	NC	NC -- NC	4	4.9	NC	NC -- NC
3574	6	7.7	78	28 -- 169	1	3.9	NC	NC -- NC	5	3.8	132	42 -- 308
3575	8	6.9	116	50 -- 229	5	3.4	146	47 -- 340	3	3.4	NC	NC -- NC
3576	3	8.6	NC	NC -- NC	1	4.2	NC	NC -- NC	2	4.4	NC	NC -- NC
3577	21	13.6	155	96 -- 236	7	6.2	114	46 -- 234	14	7.4	189	* 103 -- 316
3578	17	14.3	118	69 -- 190	7	7.7	91	36 -- 187	10	6.6	151	72 -- 277
City Total[†]	84	91.1	92	74 -- 114	32	44.6	72	49 -- 101	52	46.5	112	84 -- 147

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

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

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

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

TABLE 5b
Non-Hodgkin's Lymphoma Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	6	5.4	110	40 -- 241	2	2.5	NC	NC -- NC	4	2.9	NC	NC -- NC
3572	4	3.0	NC	NC -- NC	3	1.5	NC	NC -- NC	1	1.5	NC	NC -- NC
3573	1	2.8	NC	NC -- NC	0	1.3	NC	NC -- NC	1	1.5	NC	NC -- NC
3574	2	2.3	NC	NC -- NC	0	1.1	NC	NC -- NC	2	1.2	NC	NC -- NC
3575	3	2.1	NC	NC -- NC	2	1.0	NC	NC -- NC	1	1.1	NC	NC -- NC
3576	1	2.4	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.2	NC	NC -- NC
3577	4	3.9	NC	NC -- NC	2	1.7	NC	NC -- NC	2	2.2	NC	NC -- NC
3578	3	3.9	NC	NC -- NC	2	2.1	NC	NC -- NC	1	1.9	NC	NC -- NC
City Total[†]	24	25.8	93	60 -- 139	11	12.2	90	45 -- 161	13	13.6	96	51 -- 164

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

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

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

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

TABLE 5c
Non-Hodgkin's Lymphoma Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	5	6.4	78	25 -- 181	0	2.9	NC	NC -- NC	5	3.5	143	46 -- 333
3572	0	3.6	NC	NC -- NC	0	1.9	NC	NC -- NC	0	1.7	NC	NC -- NC
3573	3	3.1	NC	NC -- NC	0	1.5	NC	NC -- NC	3	1.6	NC	NC -- NC
3574	1	2.6	NC	NC -- NC	0	1.3	NC	NC -- NC	1	1.3	NC	NC -- NC
3575	4	2.3	NC	NC -- NC	3	1.1	NC	NC -- NC	1	1.1	NC	NC -- NC
3576	0	2.8	NC	NC -- NC	0	1.4	NC	NC -- NC	0	1.4	NC	NC -- NC
3577	7	4.5	156	62 -- 321	2	2.0	NC	NC -- NC	5	2.5	203	65 -- 475
3578	7	4.7	148	59 -- 306	3	2.5	NC	NC -- NC	4	2.2	NC	NC -- NC
City Total[†]	27	30.0	90	59 -- 131	8	14.7	54	23 -- 107	19	15.3	124	75 -- 193

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

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

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

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

TABLE 5d
Non-Hodgkin's Lymphoma Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	9	7.2	125	57 -- 236	5	3.5	145	47 -- 338	4	3.8	NC	NC -- NC
3572	0	4.1	NC	NC -- NC	0	2.2	NC	NC -- NC	0	1.9	NC	NC -- NC
3573	1	3.4	NC	NC -- NC	1	1.7	NC	NC -- NC	0	1.7	NC	NC -- NC
3574	3	2.9	NC	NC -- NC	1	1.5	NC	NC -- NC	2	1.4	NC	NC -- NC
3575	1	2.6	NC	NC -- NC	0	1.3	NC	NC -- NC	1	1.3	NC	NC -- NC
3576	2	3.2	NC	NC -- NC	1	1.6	NC	NC -- NC	1	1.6	NC	NC -- NC
3577	10	5.0	199	95 -- 366	3	2.3	NC	NC -- NC	7	2.8	254	* 102 -- 524
3578	7	5.5	128	51 -- 264	2	2.9	NC	NC -- NC	5	2.5	197	63 -- 459
City Total[†]	33	34.0	97	67 -- 136	13	17.0	76	41 -- 131	20	17.0	117	72 -- 181

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

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

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

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

TABLE 6a
Pancreatic Cancer Incidence
Belmont, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	16	13.2	121	69 -- 196	8	5.6	143	62 -- 282	8	7.6	105	45 -- 206
3572	5	6.8	73	24 -- 171	3	3.3	NC	NC -- NC	2	3.5	NC	NC -- NC
3573	4	5.7	NC	NC -- NC	3	2.4	NC	NC -- NC	1	3.2	NC	NC -- NC
3574	3	4.7	NC	NC -- NC	0	2.2	NC	NC -- NC	3	2.5	NC	NC -- NC
3575	1	4.2	NC	NC -- NC	0	1.9	NC	NC -- NC	1	2.3	NC	NC -- NC
3576	5	5.4	93	30 -- 217	1	2.4	NC	NC -- NC	4	2.9	NC	NC -- NC
3577	10	8.8	113	54 -- 208	4	3.6	NC	NC -- NC	6	5.2	NC	NC -- NC
3578	13	9.1	143	76 -- 245	4	4.6	NC	NC -- NC	9	4.4	203	93 -- 385
City Total[†]	57	57.9	98	75 -- 127	23	26.1	88	56 -- 132	34	31.8	107	74 -- 149

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

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

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

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

TABLE 6b
Pancreatic Cancer Incidence
Belmont, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	7	4.3	164	66 -- 337	2	1.9	NC	NC -- NC	5	2.4	206	66 -- 481
3572	1	2.2	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.2	NC	NC -- NC
3573	2	2.1	NC	NC -- NC	1	0.9	NC	NC -- NC	1	1.2	NC	NC -- NC
3574	1	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	1	1.0	NC	NC -- NC
3575	0	1.6	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.9	NC	NC -- NC
3576	2	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	2	1.0	NC	NC -- NC
3577	2	3.0	NC	NC -- NC	0	1.2	NC	NC -- NC	2	1.8	NC	NC -- NC
3578	5	2.9	171	55 -- 399	0	1.5	NC	NC -- NC	5	1.5	343	* 110 -- 800
City Total[†]	20	19.5	102	63 -- 158	3	8.6	NC	NC -- NC	17	10.9	156	91 -- 250

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

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

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

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

TABLE 6c
Pancreatic Cancer Incidence
Belmont, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	3	4.2	NC	NC -- NC	2	1.7	NC	NC -- NC	1	2.4	NC	NC -- NC
3572	1	2.2	NC	NC -- NC	0	1.0	NC	NC -- NC	1	1.1	NC	NC -- NC
3573	2	1.8	NC	NC -- NC	2	0.8	NC	NC -- NC	0	1.0	NC	NC -- NC
3574	1	1.5	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.8	NC	NC -- NC
3575	1	1.3	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.7	NC	NC -- NC
3576	1	1.7	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.9	NC	NC -- NC
3577	4	2.8	NC	NC -- NC	2	1.1	NC	NC -- NC	2	1.6	NC	NC -- NC
3578	5	2.9	174	56 -- 406	1	1.5	NC	NC -- NC	4	1.4	NC	NC -- NC
City Total[†]	18	18.2	99	58 -- 156	7	8.2	85	34 -- 176	11	10.0	109	55 -- 196

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

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

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

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

TABLE 6d
Pancreatic Cancer Incidence
Belmont, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3571	6	4.5	134	49 -- 292	4	2.0	NC	NC -- NC	2	2.5	NC	NC -- NC
3572	3	2.3	NC	NC -- NC	3	1.1	NC	NC -- NC	0	1.2	NC	NC -- NC
3573	0	1.8	NC	NC -- NC	0	0.8	NC	NC -- NC	0	1.0	NC	NC -- NC
3574	1	1.6	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.8	NC	NC -- NC
3575	0	1.4	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.8	NC	NC -- NC
3576	2	1.8	NC	NC -- NC	1	0.8	NC	NC -- NC	1	1.0	NC	NC -- NC
3577	4	3.0	NC	NC -- NC	2	1.2	NC	NC -- NC	2	1.8	NC	NC -- NC
3578	3	3.2	NC	NC -- NC	3	1.6	NC	NC -- NC	0	1.6	NC	NC -- NC
City Total[†]	19	19.7	96	58 -- 150	13	9.0	144	77 -- 246	6	10.7	56	20 -- 122

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

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

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

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

TABLE 7a
Kidney Cancer Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total						Males						Females					
	Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI		
3543	8	7.8	103	44	--	203	2	4.2	NC	NC	--	NC	6	3.6	167	61	--	363
3546	4	7.5	NC	NC	--	NC	1	4.2	NC	NC	--	NC	3	3.4	NC	NC	--	NC
3549	3	7.8	NC	NC	--	NC	0	4.6	NC	NC	--	NC	3	3.2	NC	NC	--	NC
City Total[†]	109	146.3	75 *	61	--	90	57	85.9	66 *	50	--	86	52	60.4	86	64	--	113

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

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

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

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

TABLE 7b
Kidney Cancer Incidence
Cambridge, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	1	2.1	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.0	NC	NC -- NC
3546	0	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.9	NC	NC -- NC
3549	0	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.9	NC	NC -- NC
City Total[†]	30	39.0	77	52 -- 110	18	21.8	83	49 -- 131	12	17.3	70	36 -- 121

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

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

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

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

TABLE 7c
Kidney Cancer Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	3	2.7	NC	NC -- NC	0	1.5	NC	NC -- NC	3	1.2	NC	NC -- NC
3546	3	2.6	NC	NC -- NC	1	1.5	NC	NC -- NC	2	1.2	NC	NC -- NC
3549	1	2.7	NC	NC -- NC	0	1.6	NC	NC -- NC	1	1.1	NC	NC -- NC
City Total[†]	32	51.4	62	* 43 -- 88	14	30.7	46	* 25 -- 76	18	20.7	87	52 -- 138

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

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

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

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

TABLE 7d
Kidney Cancer Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	4	2.7	NC	NC -- NC	2	1.4	NC	NC -- NC	2	1.3	NC	NC -- NC
3546	1	2.9	NC	NC -- NC	0	1.6	NC	NC -- NC	1	1.3	NC	NC -- NC
3549	2	2.9	NC	NC -- NC	0	1.7	NC	NC -- NC	2	1.2	NC	NC -- NC
City Total[†]	47	56.4	83	61 -- 111	25	33.5	75	48 -- 110	22	22.9	96	60 -- 145

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

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

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

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

TABLE 8a
Leukemia Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
3543	7	7.1	98	39	202	2	3.5	NC	NC	NC	5	3.7	137	44	320
3546	6	6.7	90	33	195	4	3.4	NC	NC	NC	2	3.3	NC	NC	NC
3549	5	7.1	71	23	165	5	3.9	129	41	300	0	3.2	NC	NC	NC
City Total†	102	134.1	76 *	62	92	55	72.0	76 *	58	99	47	62.1	76	56	101

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

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

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

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

TABLE 8b
Leukemia Incidence
Cambridge, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	2	2.3	NC	NC -- NC	0	1.1	NC	NC -- NC	2	1.1	NC	NC -- NC
3546	3	2.0	NC	NC -- NC	2	1.1	NC	NC -- NC	1	1.0	NC	NC -- NC
3549	3	2.1	NC	NC -- NC	3	1.1	NC	NC -- NC	0	1.0	NC	NC -- NC
City Total[†]	38	41.7	91	64 -- 125	24	22.1	109	70 -- 162	14	19.6	71	39 -- 120

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

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

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

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

TABLE 8c
Leukemia Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
3543	4	2.1	NC	NC	-- NC	2	1.1	NC	NC	-- NC	2	1.1	NC	NC	-- NC
3546	1	2.0	NC	NC	-- NC	0	1.0	NC	NC	-- NC	1	1.0	NC	NC	-- NC
3549	1	2.2	NC	NC	-- NC	1	1.2	NC	NC	-- NC	0	1.0	NC	NC	-- NC
City Total[†]	27	41.1	66	* 43	-- 96	13	22.3	58	31	-- 100	14	18.8	75	41	-- 125

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

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

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

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

TABLE 8d
Leukemia Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
3543	1	2.5	NC	NC	NC	0	1.2	NC	NC	NC	1	1.3	NC	NC	NC
3546	2	2.6	NC	NC	NC	2	1.3	NC	NC	NC	0	1.3	NC	NC	NC
3549	1	2.7	NC	NC	NC	1	1.5	NC	NC	NC	0	1.2	NC	NC	NC
City Total[†]	37	52.2	71 *	50	98	18	28.0	64	38	102	19	24.1	79	47	123

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

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

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

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

TABLE 9a
Liver Cancer Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	3	1.8	NC	NC -- NC	3	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC
3546	2	1.7	NC	NC -- NC	0	1.2	NC	NC -- NC	2	0.6	NC	NC -- NC
3549	4	1.8	NC	NC -- NC	2	1.3	NC	NC -- NC	2	0.5	NC	NC -- NC
City Total[†]	40	34.2	117	84 -- 159	26	23.9	109	71 -- 160	14	10.3	136	74 -- 227

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

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

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

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

TABLE 9b
Liver Cancer Incidence
Cambridge, Massachusetts
1982--1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	1	0.5	NC	NC -- NC	1	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC
3546	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
3549	2	0.4	NC	NC -- NC	1	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC
City Total[†]	13	8.5	153	82 -- 262	7	5.5	127	51 -- 261	6	3.0	203	74 -- 442

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

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

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

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

TABLE 9c
Liver Cancer Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	1	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
3546	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
3549	1	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
City Total[†]	10	10.7	94	45 -- 173	7	7.5	93	37 -- 192	3	3.1	NC	NC -- NC

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

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

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

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

TABLE 9d
Liver Cancer Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
3546	2	0.8	NC	NC -- NC	0	0.5	NC	NC -- NC	2	0.2	NC	NC -- NC
3549	1	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.2	NC	NC -- NC
City Total[†]	17	15.1	113	66 -- 181	12	10.9	110	57 -- 193	5	4.2	119	38 -- 278

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

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

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

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

TABLE 10a
Lung and Bronchus Cancer Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
3543	41	50.0	82	59	-- 111	24	26.9	89	57	-- 133	17	23.2	73	43	-- 117
3546	39	47.6	82	58	-- 112	19	25.6	74	45	-- 116	20	22.0	91	55	-- 140
3549	51	48.4	105	78	-- 139	31	27.8	111	76	-- 158	20	20.6	97	59	-- 150
City Total†	799	897.4	89 *	83	-- 95	460	509.6	90 *	82	-- 99	339	387.8	87 *	78	-- 97

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

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

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

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

TABLE 10b
Lung and Bronchus Cancer Incidence
Cambridge, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	21	15.6	135	83 -- 206	15	9.4	159	89 -- 262	6	6.2	97	36 -- 212
3546	14	14.4	97	53 -- 163	7	8.8	80	32 -- 164	7	5.6	124	50 -- 256
3549	14	14.5	97	53 -- 162	9	8.7	104	47 -- 197	5	5.8	86	28 -- 201
City Total[†]	276	278.0	99	88 -- 112	179	169.1	106	91 -- 123	97	109.0	89	72 -- 109

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

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

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

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

TABLE 10c
Lung and Bronchus Cancer Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total						Males						Females					
	Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI		Obs	Exp	SIR		95% CI	
3543	12	16.6	72		37 --	127	5	9.0	56		18 --	130	7	7.6	92		37 --	190
3546	11	15.9	69		35 --	124	4	8.6	NC		NC --	NC	7	7.3	96		38 --	198
3549	17	16.2	105		61 --	168	9	9.4	96		44 --	182	8	6.9	117		50 --	230
City Total[†]	251	300.1	84	*	74 --	95	134	171.8	78	*	65 --	92	117	128.4	91		75 --	109

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

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

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

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

TABLE 10d
Lung and Bronchus Cancer Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total						Males						Females					
	Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI			Obs	Exp	SIR	95% CI		
3543	8	16.6	48	*	21	-- 95	4	8.0	NC	NC	--	NC	4	8.6	NC	NC	--	NC
3546	14	16.8	83		46	-- 140	8	8.3	96	41	--	190	6	8.5	71	26	--	154
3549	20	16.9	118		72	-- 183	13	9.0	144	77	--	247	7	7.9	89	35	--	182
City Total[†]	272	320.2	85	*	75	-- 96	147	169.0	87	73	--	102	125	151.2	83	* 69	--	99

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

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

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

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

TABLE 11a
Non-Hodgkin's Lymphoma Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	11	12.9	85	43 -- 153	4	5.7	NC	NC -- NC	7	7.2	97	39 -- 199
3546	10	12.2	82	39 -- 151	2	5.7	NC	NC -- NC	8	6.5	122	53 -- 241
3549	6	12.3	49	18 -- 106	2	6.3	NC	NC -- NC	4	5.9	NC	NC -- NC
City Total[†]	186	242.5	77 *	66 -- 89	88	123.1	72 *	57 -- 88	98	119.4	82	66 -- 100

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

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

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

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

TABLE 11b
Non-Hodgkin's Lymphoma Incidence
Cambridge, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	1	3.6	NC	NC -- NC	0	1.6	NC	NC -- NC	1	2.0	NC	NC -- NC
3546	3	3.2	NC	NC -- NC	1	1.5	NC	NC -- NC	2	1.7	NC	NC -- NC
3549	2	3.2	NC	NC -- NC	0	1.5	NC	NC -- NC	2	1.7	NC	NC -- NC
City Total[†]	48	66.3	72	* 53 -- 96	21	32.0	66	41 -- 100	27	34.4	79	52 -- 114

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

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

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

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

TABLE 11c
Non-Hodgkin's Lymphoma Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	6	4.3	141	51 -- 307	2	1.9	NC	NC -- NC	4	2.4	NC	NC -- NC
3546	4	4.0	NC	NC -- NC	1	1.9	NC	NC -- NC	3	2.2	NC	NC -- NC
3549	1	4.1	NC	NC -- NC	1	2.1	NC	NC -- NC	0	2.0	NC	NC -- NC
City Total[†]	68	81.2	84	65 -- 106	35	41.7	84	59 -- 117	33	39.5	83	57 -- 117

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

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

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

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

TABLE 11d
Non-Hodgkin's Lymphoma Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	4	4.6	NC	NC -- NC	2	2.1	NC	NC -- NC	2	2.6	NC	NC -- NC
3546	3	4.8	NC	NC -- NC	0	2.3	NC	NC -- NC	3	2.5	NC	NC -- NC
3549	3	4.8	NC	NC -- NC	1	2.5	NC	NC -- NC	2	2.3	NC	NC -- NC
City Total[†]	70	96.8	72	* 56 -- 91	32	50.4	64	* 43 -- 90	38	46.4	82	58 -- 112

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

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

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

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

TABLE 12a
Pancreatic Cancer Incidence
Cambridge, Massachusetts
1982–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	9	8.4	107	49 -- 203	3	3.3	NC	NC -- NC	6	5.1	117	43 -- 255
3546	8	7.4	109	47 -- 214	3	3.1	NC	NC -- NC	5	4.3	117	38 -- 272
3549	13	7.0	187	99 -- 320	9	3.3	273	* 125 -- 519	4	3.7	NC	NC -- NC
City Total[†]	139	136.4	102	86 -- 120	67	61.2	110	85 -- 139	72	75.2	96	75 -- 121

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

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

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

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

TABLE 12b
Pancreatic Cancer Incidence
Cambridge, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	4	2.8	NC	NC -- NC	2	1.1	NC	NC -- NC	2	1.7	NC	NC -- NC
3546	4	2.3	NC	NC -- NC	2	1.0	NC	NC -- NC	2	1.3	NC	NC -- NC
3549	5	2.2	224	72 -- 523	4	1.0	NC	NC -- NC	1	1.3	NC	NC -- NC
City Total[†]	51	45.5	112	83 -- 147	24	19.5	123	79 -- 183	27	26.0	104	68 -- 151

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

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

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

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

TABLE 12c
Pancreatic Cancer Incidence
Cambridge, Massachusetts
1988–1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	3	2.6	NC	NC -- NC	1	1.0	NC	NC -- NC	2	1.6	NC	NC -- NC
3546	3	2.3	NC	NC -- NC	1	1.0	NC	NC -- NC	2	1.4	NC	NC -- NC
3549	4	2.2	NC	NC -- NC	3	1.1	NC	NC -- NC	1	1.2	NC	NC -- NC
City Total[†]	45	43.3	104	76 -- 139	24	19.5	123	79 -- 183	21	23.8	88	55 -- 135

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

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

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

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

TABLE 12d
Pancreatic Cancer Incidence
Cambridge, Massachusetts
1994–1999

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
3543	2	2.7	NC	NC -- NC	0	1.1	NC	NC -- NC	2	1.6	NC	NC -- NC
3546	1	2.6	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.5	NC	NC -- NC
3549	4	2.4	NC	NC -- NC	2	1.2	NC	NC -- NC	2	1.3	NC	NC -- NC
City Total[†]	43	48.0	89	65 -- 121	19	22.1	86	52 -- 134	24	26.0	92	59 -- 137

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

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

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

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

TABLE 13a
Cancer Incidence
Arlington, Massachusetts
1982–1999

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	94	99.9	94	76	115	58	56.8	102	78	132	36	43.1	83	58	116
Leukemia	85	86.3	98	79	122	45	44.8	100	73	134	40	41.5	96	69	131
Liver	28	23.4	120	79	173	17	16.0	107	62	171	11	7.5	147	73	264
Lung & Bronchus	544	643.8	84 *	78	92	328	359.1	91	82	102	216	284.7	76 *	66	87
NHL	161	158.7	101	86	118	73	75.0	97	76	122	88	83.7	105	84	130
Pancreatic	102	100.0	102	83	124	46	42.7	108	79	144	56	57.2	98	74	127

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

TABLE 13b
Cancer Incidence
Arlington, Massachusetts
1982–1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	28	27.6	102	67 -- 147	18	15.2	118	70 -- 187	10	12.3	81	39 -- 149
Leukemia	30	27.5	109	74 -- 156	18	14.3	125	74 -- 198	12	13.2	91	47 -- 159
Liver	12	6.0	201	* 104 -- 351	8	3.9	205	88 -- 404	4	2.1	NC	NC -- NC
Lung & Bronchus	174	204.9	85	* 73 -- 99	113	125.6	90	74 -- 108	61	79.3	77	* 59 -- 99
NHL	43	44.7	96	70 -- 130	13	20.8	62	33 -- 107	30	23.9	126	85 -- 179
Pancreatic	44	33.4	132	96 -- 177	25	14.3	174	* 113 -- 257	19	19.0	100	60 -- 156

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

TABLE 13c
Cancer Incidence
Arlington, Massachusetts
1988–1993

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	26	35.0	74	49	109	16	20.3	79	45	128	10	14.7	68	33	125
Leukemia	20	26.1	77	47	118	12	13.8	87	45	152	8	12.3	65	28	128
Liver	4	7.3	NC	NC	NC	2	5.0	NC	NC	NC	2	2.2	NC	NC	NC
Lung & Bronchus	183	214.7	85 *	73	99	106	120.5	88	72	106	77	94.2	82	64	102
NHL	53	52.6	101	76	132	27	24.9	108	71	158	26	27.7	94	61	138
Pancreatic	33	31.6	105	72	147	11	13.5	81	41	146	22	18.1	122	76	184

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

TABLE 13d
Cancer Incidence
Arlington, Massachusetts
1994–1999

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	40	36.3	110	79	150	24	20.9	115	74	171	16	15.5	104	59	168
Leukemia	35	31.9	110	76	153	15	16.5	91	51	150	20	15.3	131	80	201
Liver	12	9.8	123	63	214	7	6.8	103	41	212	5	3.0	168	54	393
Lung & Bronchus	187	221.2	85 *	73	98	109	114.7	95	78	115	78	106.5	73 *	58	91
NHL	65	59.3	110	85	140	33	28.7	115	79	162	32	30.6	104	71	147
Pancreatic	25	34.0	74	48	109	10	14.8	68	33	124	15	19.2	78	44	129

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

TABLE 14a
Cancer Incidence
Watertown, Massachusetts
1982–1999

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	76	70.4	108	85 -- 135	48	39.7	121	89 -- 160	28	30.7	91	61 -- 132
Leukemia	68	61.1	111	86 -- 141	38	31.5	121	85 -- 165	30	29.6	101	68 -- 145
Liver	21	16.5	127	79 -- 195	21	11.2	188	* 116 -- 287	0	5.3	NC	NC -- NC
Lung & Bronchus	423	452.4	93	85 -- 103	233	250.7	93	81 -- 106	190	201.8	94	81 -- 109
NHL	137	113.2	121	* 102 -- 143	63	53.4	118	91 -- 151	74	59.9	124	97 -- 155
Pancreatic	66	70.3	94	73 -- 119	32	29.9	107	73 -- 151	34	40.4	84	58 -- 118

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

TABLE 14b
Cancer Incidence
Watertown, Massachusetts
1982–1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	21	19.0	111	68 -- 169	13	10.5	124	66 -- 212	8	8.5	94	40 -- 185
Leukemia	18	19.1	94	56 -- 149	12	9.9	121	62 -- 211	6	9.1	66	24 -- 143
Liver	5	4.1	121	39 -- 283	5	2.7	186	60 -- 435	0	1.4	NC	NC -- NC
Lung & Bronchus	147	140.6	105	88 -- 123	90	85.7	105	84 -- 129	57	54.9	104	79 -- 135
NHL	40	31.0	129	92 -- 175	17	14.5	118	68 -- 188	23	16.6	139	88 -- 208
Pancreatic	19	23.0	83	50 -- 129	6	9.8	61	22 -- 133	13	13.1	99	53 -- 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, Center for Health Information, Statistics, Research and Evaluation, Massachusetts Department of Public Health.

TABLE 14c
Cancer Incidence
Watertown, Massachusetts
1988–1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Kidney	33	24.7	134	92 -- 188	24	14.3	168	* 108 -- 251	9	10.5	86	39 -- 163
Leukemia	21	18.5	113	70 -- 173	11	9.7	113	56 -- 203	10	8.8	113	54 -- 208
Liver	6	5.1	117	43 -- 254	6	3.5	169	62 -- 368	0	1.6	NC	NC -- NC
Lung & Bronchus	142	151.0	94	79 -- 111	83	84.2	99	79 -- 122	59	66.8	88	67 -- 114
NHL	48	37.6	128	94 -- 169	20	17.8	112	69 -- 174	28	19.8	141	94 -- 204
Pancreatic	22	22.2	99	62 -- 150	12	9.5	127	65 -- 221	10	12.8	78	38 -- 144

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

TABLE 14d
Cancer Incidence
Watertown, Massachusetts
1994–1999

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Kidney	22	26.4	83	52	126	11	15.0	73	37	131	11	11.4	97	48	173
Leukemia	29	23.5	124	83	177	15	12.1	124	69	205	14	11.4	123	67	206
Liver	10	7.1	141	68	259	10	4.9	204	98	376	0	2.2	NC	NC	NC
Lung & Bronchus	134	160.5	83 *	70	99	60	82.5	73 *	56	94	74	78.0	95	74	119
NHL	49	44.0	111	82	147	26	21.2	123	80	180	23	22.9	101	64	151
Pancreatic	25	24.9	100	65	148	14	10.7	131	72	220	11	14.2	77	38	138

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

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

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

Table 15
MDEP 21E Hazardous Material and Oil Releases
Arlington, Belmont, Cambridge, and Watertown, Massachusetts

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0010373	NO LOCATION AID (RESIDNTIAL)	72 HATHAWAY CIR	ARLINGTON	12/29/1993	TWO HR	FUEL OIL #2 (100 GAL)	AST; PIPE	RAO
MAPPED	3-0010409	NO LOCATION AID (RESIDNTIAL)	76 BRANTWOOD RD	ARLINGTON	1/9/1994	TWO HR	FUEL OIL #2 (25 GAL); FUEL OIL #2 (15 GAL)	AST; PIPE	RAO
MAPPED	3-0010500	NO LOCATION AID (RESIDNTIAL)	95 PAUL REVERE RD	ARLINGTON	2/1/1994	TWO HR	FUEL OIL #2 (100 GAL); FUEL OIL #2 (200 GAL)	AST	RAO
MAPPED	3-0010645	NEAR SUMMER ST COMMUNITY SAFETY BUILDING (COMMERCIAL; MUNICIPAL)	112 MYSTIC ST	ARLINGTON	3/7/1994	72 HR	GASOLINE (600 PPMV)	UST	RAO
MAPPED	3-0010856	RTE 2 MWRA PUMP STA (STATE)	JASON ST/SPRING ST	ARLINGTON	4/12/1994	TWO HR	FUEL OIL #2 (600 GAL); FUEL OIL #2 (150 GAL)	AST	RAO
MAPPED	3-0010969	SYMMES HOSPITAL (COMMERCIAL)	HOSPITAL RD	ARLINGTON	5/8/1994	TWO HR	OIL; PETROLEUM BASED OIL	UST	TIER 2
MAPPED	3-0011199	NO LOCATION AID (RESIDNTIAL)	7 MOHAWK RD	ARLINGTON	4/23/1994	72 HR	FUEL OIL #2 (60 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0011481	MIRAK CHEVROLET (COMMERCIAL)	26 HOBBS CT	ARLINGTON	8/17/1994	72 HR	GASOLINE (174 PPMV); GASOLINE (50 PPMV)	UST	RAO
MAPPED	3-0011482	BACK OF GARAGE (COMMERCIAL)	26 HOBBES CT	ARLINGTON	8/17/1994	72 HR	FUEL OIL #2 (53 PPMV); FUEL OIL #2 (50 PPMV)	UST	RAO
NOT MAPPED	3-0011738	NO LOCATION AID (RESIDNTIAL; ROADWAY)	PAUL REVERE RD	ARLINGTON	10/17/1994	TWO HR	OIL (2 GAL); 1,1'-BIPHENYL, CHLORO-DERIVS. (500 MG/L); 1,1'-BIPHENYL, CHLORO-DERIVS. (5 PPM); MINERAL OIL (4 GAL)	TRANSFORM	RAO
MAPPED	3-0011771	FRONT OF BUILDING (COMMERCIAL)	50 GROVE ST	ARLINGTON	10/25/1994	72 HR	FUEL OIL #2 (105 PPMV)	UST	RAO
NOT MAPPED	3-0011921	POLE 83/1 (RESIDNTIAL)	WASHINGTON ST/ARLINGTON ST	ARLINGTON	12/2/1994	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (12 GAL); MINERAL OIL (12 GAL)	TRANSFORM	RAO
MAPPED	3-0012002	POLE #18 (COMMERCIAL; ROADWAY)	SUMMER ST/MYSTIC ST	ARLINGTON	12/24/1994	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (50 GAL); PETROLEUM BASED OIL (30 GAL)	TRANSFORM	RAO
MAPPED	3-0012118	ANDERSON & SONS INC (COMMERCIAL)	895-901 MASSACHUSETTS AVE	ARLINGTON	1/30/1995	72 HR	FUEL OIL #2 (220 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (226 PPMV)	UST	RAO
MAPPED	3-0012170	NO LOCATION AID (COMMERCIAL)	305 BROADWAY	ARLINGTON	2/13/1995	TWO HR	DIESEL FUEL (25 GAL)	VEHICLE	DEF TIER 1B
NOT MAPPED	3-0012342	NEAR WILDWOOD APTS (ROADWAY)	PLEASANT ST	ARLINGTON	4/4/1995	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (40 GAL)	PIPE	RAO
MAPPED	3-0013059	NO LOCATION AID (COMMERCIAL)	1425 MASSACHUSETTS AVE	ARLINGTON	10/19/1995	72 HR	FUEL OIL #2 (100 PPM)	UST	RAO
MAPPED	3-0013084	ARLINGTON GARDENS APTS (RESIDNTIAL)	130 EVERETT ST	ARLINGTON	10/25/1995	72 HR	FUEL OIL #2 (0.5 INCH)	UST	RAO
MAPPED	3-0013738	NO LOCATION AID (RESIDNTIAL)	24 CENTRAL ST	ARLINGTON	5/8/1996	TWO HR	FUEL OIL #2 (15 GAL)	AST; PIPE	RAO
MAPPED	3-0013858	NO LOCATION AID (RESIDNTIAL)	38 TOWERS RD	ARLINGTON	6/6/1996	72 HR	FUEL OIL #2 (100 PPMV)	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0014110	NO LOCATION AID (COMMERCIAL; RESIDNTIAL)	8 WELLINGTON ST	ARLINGTON	12/23/1996	72 HR	FUEL OIL #2 (107 PPMV)	UST	RAO
MAPPED	3-0014443	MBTA PARKING LOT (STATE)	1395-1425 MASS AVE	ARLINGTON	10/29/1996	72 HR	KEROSENE (11 MG/L); GASOLINE; PROPANE, 2-METHOXY-2-METHYL- (130 UG/L); NAPHTHALENE (67 UG/L)	UST	RTN CLOSED
MAPPED	3-0014668	ALPHA AUTO BODY (COMMERCIAL)	30 PARK AVE	ARLINGTON	12/25/1996	TWO HR	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (25 GAL); UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (10 GAL)	DRUMS	RAO
MAPPED	3-0014989	NO LOCATION AID (COMMERCIAL)	30 MILL ST	ARLINGTON	4/10/1997	72 HR	FUEL OIL #2 (102 PPMV); UNKNOWN CHEMICAL OF UNKNOWN TYPE (0.5 INCH)	UST	RAO
MAPPED	3-0015709	SUNOCO (COMMERCIAL)	46 BROADWAY	ARLINGTON	11/11/1997	TWO HR	WASTE OIL; OIL (1 GAL)	4; DRUMS	RTN CLOSED
MAPPED	3-0015826	MA HWY DEPT (STATE)	519 APPLETON ST	ARLINGTON	12/15/1997	72 HR	PETROLEUM BASED OIL (0.6 INCH)	UST	RTN CLOSED
MAPPED	3-0015938	RIDGE ST (RESIDNTIAL)	32 TERESA CIR	ARLINGTON	11/12/1997	TWO HR	FUEL OIL #2 (50 GAL); FUEL OIL #2 (50 GAL)	AST; PIPE	RAO
MAPPED	3-0016615	NO LOCATION AID (RESIDNTIAL)	93 WAVERLY AND 160 RENFREW STS	ARLINGTON	3/19/1998	TWO HR	FUEL OIL #2; FUEL OIL #2 (11000 MG/KG); FUEL OIL #2 (48 MG/L)	LINE; PIPE	RAO
MAPPED	3-0017104	BRACKETT SCHOOL (MUNICIPAL; SCHOOL)	EASTERN AVE	ARLINGTON	7/30/1998	72 HR	FUEL OIL #4; FUEL OIL #4 (1000 GAL); FUEL OIL #2; FUEL OIL #2 (1000 GAL)	PIPE; UST	RAO
MAPPED	3-0017441	NO LOCATION AID (COMMERCIAL)	81 MYSTIC ST	ARLINGTON	10/16/1998	72 HR	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (1043 PPMV); UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (100 PPMV)	UST	TIER 2
MAPPED	3-0017442	NO LOCATION AID (COMMERCIAL)	81 MYSTIC ST	ARLINGTON	10/16/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (125 PPMV); FUEL OIL #2 (100 PPMV)	UST	RTN CLOSED
MAPPED	3-0017565	DUDLEY FUEL (COMMERCIAL)	9 DUDLEY ST PL	ARLINGTON	11/12/1998	72 HR	GASOLINE; UNKNOWN CHEMICAL OF UNKNOWN TYPE; KEROSENE (2000 PPMV); KEROSENE; FUEL OIL #2	UST	RAO
MAPPED	3-0017599	DUDLEY FUEL (COMMERCIAL)	9 DUDLEY ST PL	ARLINGTON	11/19/1998	72 HR	WASTE OIL (122 PPMV); WASTE OIL	UST	RTN CLOSED
MAPPED	3-0017697	BRIGHAMS EAST EDGE OF PARKING LOT (COMMERCIAL)	30 MILL ST	ARLINGTON	12/7/1998	72 HR	FUEL OIL #6; FUEL OIL #6	UST	RTN CLOSED
MAPPED	3-0017698	NO LOCATION AID	140 MYSTIC ST	ARLINGTON	12/8/1998	72 HR	GASOLINE; GASOLINE (100 PPMV); UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0018021	NO LOCATION AID (COMMERCIAL)	440 MASSACHUSETTS AVE REAR	ARLINGTON	2/22/1999	72 HR	GASOLINE (104 PPMV); GASOLINE (101 PPM)	UST	RAO
MAPPED	3-0018209	TELEPHONE POLE AND CATCH BASIN	MASS AVE AND PLEASANT ST	ARLINGTON	4/20/1999	TWO HR	OIL (3 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (2 GAL)	SWITCH	RAO
MAPPED	3-0018766	BOSTON EDISON STATION 59 (COMMERCIAL)	88 MYSTIC ST	ARLINGTON	9/18/1999	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (25 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL)	TRANSFORM	RAO
MAPPED	3-0018828	EVERETT ST (COMMERCIAL)	125 BROADWAY	ARLINGTON	10/6/1999	72 HR	GASOLINE; GASOLINE (0.05 GAL/HR)	UST	TIER 2
MAPPED	3-0019131	NO LOCATION AID (COMMERCIAL)	180 MOUNTAIN AVE	ARLINGTON	1/3/2000	TWO HR	OIL; FUEL OIL #4 (11 GAL)	UNKNOWN	TIER 2
MAPPED	3-0019235	NO LOCATION AID (RESIDNTIAL)	25 MILL ST	ARLINGTON	2/2/2000	TWO HR	FUEL OIL #2 (40 GAL)	VEHICLE	RAO
MAPPED	3-0019499	BRIGHAMS REAR LOADING DOCK (COMMERCIAL)	42 MILL ST	ARLINGTON	5/3/2000	TWO HR	PETROLEUM BASED OIL (50 GAL); PETROLEUM BASED OIL	PIPE	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0019754	DPW YARD (MUNICIPAL)	51 GROVE ST	ARLINGTON	7/21/2000	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (1.2 INCH)	UNKNOWN	TIER 2
MAPPED	3-0019829	RAAB RESIDENCE (RESIDNTIAL)	117 GRAY ST	ARLINGTON	8/10/2000	72 HR	FUEL OIL #2 (252 PPMV); FUEL OIL #2 (101 PPM)	UST	RAO
MAPPED	3-0019873	NO LOCATION AID (RESIDNTIAL)	10 UNIVERSITY RD	ARLINGTON	8/25/2000	72 HR	FUEL OIL #2 (2200 PPMV); FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0020164	NO LOCATION AID (COMMERCIAL)	1395 MASSACHUSETTS AVE	ARLINGTON	11/29/2000	72 HR	GASOLINE (160 PPMV); GASOLINE	UST	RTN CLOSED
MAPPED	3-0020262	NO LOCATION AID (OPENSPACE)	1395 MASSACHUSETTS AVE	ARLINGTON	12/28/2000	72 HR	OIL (222 PPMV); FUEL OIL #2	UST	RTN CLOSED
MAPPED	3-0020732	SUNRISE (COMMERCIAL)	1395 MASSACHUSETTS AVE	ARLINGTON	5/24/2001	72 HR	DIESEL FUEL (4 INCH)	UST	RTN CLOSED
MAPPED	3-0021070	MAGNOLIA ST (RESIDNTIAL)	104 MASS AVE	ARLINGTON	9/11/2001	TWO HR	FUEL OIL #2 (40 GAL); FUEL OIL #2 (39.6 GAL)	AST; PIPE	TIER 2
MAPPED	3-0021092	REAR LOADING DOCK (COMMERCIAL)	42 MILL ST	ARLINGTON	9/18/2001	TWO HR	PETROLEUM BASED OIL (15 GAL); PETROLEUM BASED OIL (11 GAL)	VEHICLE	RAO
NOT MAPPED	3-0021196	NO LOCATION AID (COMMERCIAL)	HOSPITAL RD	ARLINGTON	10/19/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (0.5 INCH); UNKNOWN CHEMICAL OF UNKNOWN TYPE (2.64 INCH); PETROLEUM BASED OIL (2.64 INCH)	UST	RTN CLOSED
MAPPED	3-0021235	DR AND MRS CHARLES H BURKE RESIDENCE (RESIDNTIAL)	7 ARROWHEAD LANE	ARLINGTON	11/4/2001	TWO HR	FUEL OIL #2 (60 GAL); FUEL OIL #2	PIPE	UNCLASSIFIED
MAPPED	3-0021327	NO LOCATION AID (COMMERCIAL)	125 BROADWAY	ARLINGTON	12/13/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (0.5 INCH)	UST	UNCLASSIFIED
MAPPED	3-0021646	INTERSECTION WITH SUMMER ST (COMMERCIAL)	MYSTIC ST	ARLINGTON	4/5/2002	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (500 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (500 GAL)	PIPE	RAO
MAPPED	3-0021658	LOCKLAND AVE (COMMERCIAL)	880 MASSACHUSETTS AVE	ARLINGTON	4/9/2002	72 HR	GASOLINE (180 PPMV)	UST	UNCLASSIFIED
MAPPED	3-0021734	SKATING RINK (MUNICIPAL)	422 SUMMER ST	ARLINGTON	5/5/2002	TWO HR	AMMONIA (50 LBS); AMMONIA (50 LBS)	PIPE; REFRIGERAT; UNIT	UNCLASSIFIED
MAPPED	3-0022274	NO LOCATION AID (RESIDNTIAL)	93 SUNNYSIDE AVE	ARLINGTON	11/4/2002	TWO HR	FUEL OIL #2 (42 GAL); FUEL OIL #2 (40 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022328	NO LOCATION AID (RESIDNTIAL)	5 REED ST	ARLINGTON	11/20/2002	TWO HR	FUEL OIL #2 (100 GAL)	AST	UNCLASSIFIED
MAPPED	3-0022352	NO LOCATION AID (SCHOOL)	869 MASS AVE	ARLINGTON	11/27/2002	TWO HR	FUEL OIL #2 (25 GAL); FUEL OIL #4 (25 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022371	ARLINGTON HIGH SCHOOL (MUNICIPAL; SCHOOL)	869 MASSACHUSETTS AVE	ARLINGTON	12/9/2002	TWO HR	FUEL OIL #4 (6000 GAL); FUEL OIL #4 (3000 GAL)	PIPE	RAO
MAPPED	3-0011536	FMR MDC RINK (MUNICIPAL; STATE)	LAKE ST/RTE 2	BELMONT	8/26/1994	72 HR	FUEL OIL #2 (108 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (50 PPM)	UST	RAO
MAPPED	3-0011922	NO LOCATION AID (RESIDNTIAL)	77 SNAKE HILL RD	BELMONT	12/5/1994	72 HR	FUEL OIL #2 (127 PPMV); FUEL OIL #2 (50 PPMV)	UST	RAO
NOT MAPPED	3-0011938	RTE 60/RTE 2 (ROADWAY)	RTE 60	BELMONT	12/7/1994	TWO HR	LEAD (1200 LBS); SULFURIC ACID	DRUMS; VEHICLE	DEF TIER 1B

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0012253	GETTY GASOLINE STATION	350 PLEASANT ST	BELMONT	3/9/1995	72 HR	WASTE OIL (180 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (180 PPM); ETHENE, TETRACHLORO- (7810 PPB); ETHENE, TRICHLORO- (988 PPB)	UST	RAO
MAPPED	3-0013348	BASEMENT (RESIDNTIAL)	11 ORCHARD ST	BELMONT	1/19/1996	TWO HR	FUEL OIL #2	PIPE	RAO
MAPPED	3-0013550	FMR BEST PETROLEUM (COMMERCIAL)	80 CONCORD ST	BELMONT	3/13/1996	72 HR	FUEL OIL #2 (120 PPMV)	UST	RAO
NOT MAPPED	3-0013935	POLE#3 @ #23 (ROADWAY)	BENTON RD	BELMONT	6/25/1996	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL)	TRANSFORM	RAO
MAPPED	3-0013975	NO LOCATION AID (COMMERCIAL)	337 MILL ST	BELMONT	7/9/1996	72 HR	PETROLEUM BASED OIL (100 PPM)	UST	RAO
MAPPED	3-0014072	CHENEY MIDDLE SCHOOL (MUNICIPAL; SCHOOL)	95 WASHINGTON ST	BELMONT	7/31/1996	72 HR	FUEL OIL #2 (7.5 INCH)	UNKNOWN	RAO
MAPPED	3-0014636	NO LOCATION AID (RESIDNTIAL)	86 MONROE ST	BELMONT	12/17/1996	72 HR	FUEL OIL #2 (143 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (100 PPMV)	UST	RAO
MAPPED	3-0015254	RESIDENCE (RESIDNTIAL)	11 WOODFALL RD	BELMONT	6/27/1997	TWO HR	FUEL OIL #2 (100 GAL)	PIPE	RAO
MAPPED	3-0015893	WINBROOK SCHOOL (SCHOOL)	97 WATERHOUSE RD	BELMONT	1/5/1998	TWO HR	FUEL OIL #4; FUEL OIL #4; UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	UST	RAO
MAPPED	3-0016600	CAMBRIDGE PLATING (COMMERCIAL; INDUSTRIAL)	39 HITTINGER ST	BELMONT	3/15/1998	TWO HR	FUEL OIL #2 (100 GAL); FUEL OIL #2 (20 GAL)	HEATER; SPACE	RAO
MAPPED	3-0016874	MUNICIPAL LIGHT DEPT POLE 13 (RESIDNTIAL)	9 BRETTWOOD RD	BELMONT	6/3/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (30 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (12 GAL)	TRANSFORM	RAO
MAPPED	3-0017080	NO LOCATION AID (RESIDNTIAL)	40 HOWELLS RD	BELMONT	7/23/1998	72 HR	FUEL OIL #2 (103.7 PPMV); FUEL OIL #2 (104 PPM)	UST	RAO
MAPPED	3-0017677	NO LOCATION AID (COMMERCIAL)	297 TRAPELO RD	BELMONT	12/3/1998	72 HR	GASOLINE (553 PPMV); UNKNOWN CHEMICAL OF UNKNOWN TYPE; PETROLEUM BASED OIL (100 PPMV)	UST	RAO
MAPPED	3-0018823	NO LOCATION AID (ROADWAY)	BEACH & MAPLE	BELMONT	10/4/1999	TWO HR	FUEL OIL #2 (60 GAL); FUEL OIL #2 (100 GAL)	VEHICLE	RAO
MAPPED	3-0019570	BELMONT SPRINGS WATER CO (COMMERCIAL)	1010 PLEASANT ST	BELMONT	5/24/2000	72 HR	OIL; DIESEL FUEL	PIPE; UST	RAO
MAPPED	3-0020322	NO LOCATION AID (MUNICIPAL; ROADWAY)	112 STONEY BROOK RD	BELMONT	1/18/2001	TWO HR	FUEL OIL #2 (49 GAL); FUEL OIL #2 (40 GAL)	PIPE; VEHICLE	RAO
MAPPED	3-0020406	NO LOCATION AID (COMMERCIAL; HOSPITAL)	115 MILL ST	BELMONT	2/19/2001	TWO HR	PETROLEUM BASED OIL; FUEL OIL #2 (11 GAL)	UNKNOWN	TIER 2
MAPPED	3-0020576	INTERSECTION OF BRIGHT RD (COMMERCIAL)	82 CONCORD AVE	BELMONT	4/6/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (0.5 INCH); UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RTN CLOSED
MAPPED	3-0020587	NO LOCATION AID (RESIDNTIAL)	36 HILLCREST RD	BELMONT	4/10/2001	72 HR	FUEL OIL #2 (140 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (158 PPM)	UST	RAO
MAPPED	3-0021025	NO LOCATION AID (COMMERCIAL)	270 TRAPELO RD	BELMONT	8/24/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (17 INCH)	UNKNOWN	RTN CLOSED
MAPPED	3-0021120	FORMER MOBIL S/S 1-193 (COMMERCIAL)	337 PLEASANT ST	BELMONT	10/1/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (12 INCH); UNKNOWN CHEMICAL OF UNKNOWN TYPE (1 INCH)	UNKNOWN	RTN CLOSED
MAPPED	3-0021369	NO LOCATION AID (RESIDNTIAL)	26 CEDAR ST	BELMONT	1/3/2002	72 HR	GASOLINE (362 PPMV)	UST	RAO
MAPPED	3-0022041	NO LOCATION AID (COMMERCIAL)	2 LEONARD ST	BELMONT	8/19/2002	72 HR	FUEL OIL #2 (280 PPMV); FUEL OIL #2 (500 PPMV)	UST	UNCLASSIFIED

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0022406	NO LOCATION AID (RESIDNTIAL)	3 CLARKE LANE	BELMONT	12/18/2002	TWO HR	FUEL OIL #2 (10 GAL)	AST	UNCLASSIFIED
MAPPED	3-0022478	NO LOCATION AID (COMMERCIAL)	563 TRAPELO RD	BELMONT	1/9/2003	TWO HR	GASOLINE (18 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (18 GAL)	VEHICLE	UNCLASSIFIED
MAPPED	3-0001442	RIVERSIDE CAMBRIDGE GALLERIA	88 FIRST ST	CAMBRIDGE	7/15/1989	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE; CADMIUM (38 MG/KG)		RAO
MAPPED	3-0010005	CORNER OF HAMPSHIRE ST (COMMERCIAL)	284 NORFOLK ST	CAMBRIDGE	10/1/1993	72 HR	FUEL OIL #6; FUEL OIL #6 (5600 MG/KG)	UST	RAO
MAPPED	3-0010053	HERMAN BLDG #E53 (COMMERCIAL)	30 WADSWORTH ST	CAMBRIDGE	10/6/1993	TWO HR	PETROLEUM BASED OIL (65 GAL); PETROLEUM BASED OIL (85 GAL)	PISTON	RAO
MAPPED	3-0010119	ACROSS FROM MUSEUM OF SCIENCE (STATE)	OBRIEN HWY	CAMBRIDGE	10/29/1993	TWO HR	DIESEL FUEL (5000 GAL); FUEL OIL #2 (200 GAL)	PIPE; UST	RAO
MAPPED	3-0010139	CAMBRIDGE WATER DEPT (MUNICIPAL)	250 FRESH POND PKWY	CAMBRIDGE	11/3/1993	72 HR	FUEL OIL #2; WASTE OIL (150 GAL)	UNKNOWN	RAO
MAPPED	3-0010151	NO LOCATION AID (INDUSTRIAL; ROADWAY)	324 RINDGE RD	CAMBRIDGE	11/4/1993	TWO HR	METHANE, DICHLORO- (55 GAL); METHANE, DICHLORO- (55 GAL)	DRUMS; VEHICLE	RAO
MAPPED	3-0010162	DPW YARD (COMMERCIAL)	147 HAMPSHIRE ST	CAMBRIDGE	11/8/1993	72 HR	DIESEL FUEL (300 PPMV); GASOLINE (300 PPM); DIESEL FUEL (100 PPM)	UST	RAO
MAPPED	3-0010176	BRISTON ARMS APTS (COMMERCIAL)	247 GARDEN ST	CAMBRIDGE	10/5/1993	TWO HR	METHANE; UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	LANDFILL	ADEQUATE REG
MAPPED	3-0010267	NO LOCATION AID (COMMERCIAL)	320 BROADWAY	CAMBRIDGE	10/1/1993	72 HR	WASTE OIL (118 PPMV); FUEL OIL #2 (3.6 PPMV)	PIPE; UST	TIER 2
MAPPED	3-0010304	NO LOCATION AID (COMMERCIAL)	10-12 WENDALL ST	CAMBRIDGE	12/14/1993	72 HR	FUEL OIL #2 (50 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0010338	SHELL SERVICE STATION (COMMERCIAL)	820 MEMORIAL DR	CAMBRIDGE	12/22/1993	72 HR	WASTE OIL	UST	RAO
MAPPED	3-0010374	GULF STATION #118517 (COMMERCIAL)	1725 MASSACHUSETTS AVE	CAMBRIDGE	12/30/1993	72 HR	FUEL OIL #2	PIPE	TIER 2
MAPPED	3-0010446	NEAR BENT ST (COMMERCIAL)	160 SECOND ST	CAMBRIDGE	1/18/1994	72 HR	FUEL OIL #2 (5 INCH); FUEL OIL #2	UST	RAO
MAPPED	3-0010490	REAR OF BLDG (COMMERCIAL; RESIDNTIAL; ROADWAY)	2000 MASSACHUSETTS AVE	CAMBRIDGE	1/29/1994	TWO HR	FUEL OIL #2 (50 GAL); FUEL OIL #2 (300 GAL)	PIPE; UST	RAO
MAPPED	3-0010513	NO LOCATION AID (MUNICIPAL)	250 FRESH POND PKWY	CAMBRIDGE	2/3/1994	TWO HR	SILICATE(2-), HEXAFLUORO-, DIHYDROGEN (5 GAL); SILICATE(2-), HEXAFLUORO-, DIHYDROGEN (5 GAL)	TANK	RAO
MAPPED	3-0010535	NO LOCATION AID (RESIDNTIAL)	343 CONCORD AVE	CAMBRIDGE	9/20/1994	72 HR	GASOLINE; ETHANE, 1,2-DICHLORO-	UST	RAO
MAPPED	3-0010558	MT AUBURN HOSPITAL	330 MT AUBURN HOSPITAL	CAMBRIDGE	10/1/1993	72 HR	FUEL OIL #2	UST	TIER 2
MAPPED	3-0010567	ST PETERS CHURCH	100 CONCORD AVE	CAMBRIDGE	10/1/1993	72 HR	FUEL OIL #2 (21100 MG/KG)	UST	RAO
MAPPED	3-0010568	NO LOCATION AID	64 MOULTON ST	CAMBRIDGE	10/1/1993	72 HR	FUEL OIL #2 (66000 MG/KG)	UST	RAO
MAPPED	3-0010625	CAMBRIDGE WATER DEPT (MUNICIPAL)	250 FRESH POND PKWY	CAMBRIDGE	10/1/1993	TWO HR	FUEL OIL #2	UST	RTN CLOSED
MAPPED	3-0010664	NO LOCATION AID (COMMERCIAL)	357-359 HURON AVE	CAMBRIDGE	3/11/1994	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE; FUEL OIL #2 (28 MG/L); DIESEL FUEL; BTEX (0.06 MG/L)	UNKNOWN	RAO
MAPPED	3-0010711	NO LOCATION AID (COMMERCIAL)	2055 MASS AVE	CAMBRIDGE	3/21/1994	72 HR	GASOLINE (205 PPMV); GASOLINE	PIPE; UST	TIER 2
MAPPED	3-0010760	16 CHAUNCY ST CONDO ASSOC (RESIDNTIAL)	16 CHAUNCY ST	CAMBRIDGE	3/29/1994	TWO HR	FUEL OIL #2 (1 INCH); PETROLEUM BASED OIL	UST	TIER 1C

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0010805	NEVILLE NURSING HOME	650 CONCORD AVE	CAMBRIDGE	4/6/1994	72 HR	GASOLINE (158 PPMV); GASOLINE	UST	RAO
MAPPED	3-0010823	HARVARD UNIVERSITY (COMMERCIAL)	3 SACRAMENTO ST	CAMBRIDGE	4/11/1994	TWO HR	FUEL OIL #2 (60 GAL)	PIPE; TANKER	RAO
MAPPED	3-0010876	NEAR INTERSECTION WITH CONCORD AVE (RESIDNTIAL)	6 FALLON ST	CAMBRIDGE	4/12/1994	TWO HR	FUEL OIL #2 (300 GAL)	AST	RAO
MAPPED	3-0010916	NO LOCATION AID (COMMERCIAL)	173 HARVEY ST	CAMBRIDGE	4/26/1994	72 HR	GASOLINE; FUEL OIL #2 (4460 PPM)	UST	RAO
MAPPED	3-0010928	PAYNE ELEVATOR (INDUSTRIAL)	75 RICHDALE AVE	CAMBRIDGE	4/28/1994	72 HR	FUEL OIL #2 (50 PPMV); FUEL OIL #4 (91 PPM)	UST	RAO
MAPPED	3-0010952	NO LOCATION AID (COMMERCIAL; INDUSTRIAL)	60 MOULTON ST	CAMBRIDGE	5/4/1994	72 HR	FUEL OIL #2 (200 PPMV); BENZENE, 1,3-DIMETHYL- (6.4 PPB); BENZENE, 1,4-DIMETHYL- (6.4 PPB); BENZENE, 1,2-DIMETHYL (9.5 PPB); ETHENE, TRICHLORO- (5.5 PPB); TOTAL PETROLEUM HYDROCARBONS (TPH) (2000 PPM)	UST	RAO
MAPPED	3-0011030	NO LOCATION AID (COMMERCIAL)	10 WARE ST	CAMBRIDGE	5/8/1994	72 HR	FUEL OIL #2 (750 PPM); FUEL OIL #2 (50 PPMV)	UST	RAO
MAPPED	3-0011077	GULF STA (COMMERCIAL)	1725 MASSACHUSETTS AVE	CAMBRIDGE	6/2/1994	72 HR	GASOLINE (1248 PPMV); GASOLINE (1248 PPM)	UST	RTN CLOSED
MAPPED	3-0011110	NO LOCATION AID (RESIDNTIAL)	20 GREY GARDENS WEST	CAMBRIDGE	6/9/1994	72 HR	FUEL OIL #2 (148 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0011113	NO LOCATION AID	210 BENT ST	CAMBRIDGE	6/10/1994	72 HR	KEROSENE (160 PPMV); KEROSENE (160 PPMV)	UST	RAO
MAPPED	3-0011196	NO LOCATION AID (COMMERCIAL)	50 MOULTON ST	CAMBRIDGE	6/23/1994	72 HR	FUEL OIL #2 (102 PPMV); TOTAL PETROLEUM HYDROCARBONS (TPH) (50 PPM)	UST	RAO
MAPPED	3-0011198	NO LOCATION AID (COMMERCIAL)	60 LANDSDOWNE ST	CAMBRIDGE	6/23/1994	72 HR	FUEL OIL #2	UST	RAO
MAPPED	3-0011247	NO LOCATION AID (COMMERCIAL)	222 THIRD ST	CAMBRIDGE	7/9/1994	72 HR	FUEL OIL #2 (50 PPMV); DIESEL FUEL (22000 MG/KG)	UST	RAO
MAPPED	3-0011319	FRESH POND MALL	185 ALEWIFE BROK PKWY	CAMBRIDGE	7/16/1994	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (30 GAL)	ELEVATOR	RAO
MAPPED	3-0011325	@INTERSECTION OF HIGHLAND (RESIDNTIAL)	395 BROADWAY	CAMBRIDGE	7/19/1994	72 HR	FUEL OIL #2 (150 PPMV); FUEL OIL #2 (30 GAL)	UST	RAO
MAPPED	3-0011334	CAMBRIDGE ELECTRIC CO (INDUSTRIAL; SEWER)	46 BLACKSTONE ST	CAMBRIDGE	7/20/1994	TWO HR	FUEL OIL #6 (100 GAL); FUEL OIL #6 (150 GAL)	FUEL OIL; HEATER	RAO
MAPPED	3-0011341	NO LOCATION AID (COMMERCIAL)	1120 MASSACHUSETTS AVE	CAMBRIDGE	7/22/1994	TWO HR	AMMONIA	GAS CYLIND	RAO
MAPPED	3-0011358	MIT POWER PLANT (INDUSTRIAL; SCHOOL)	59 VASSAR ST	CAMBRIDGE	7/26/1994	72 HR	FUEL OIL #2 (0.5 INCH); FUEL OIL #6 (0.5 INCH); FUEL OIL #6	UST	RAO
MAPPED	3-0011533	B & M RAILROAD YARD (INDUSTRIAL)	INDUSTRIAL PARK DR	CAMBRIDGE	8/26/1994	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (1 INCH); FUEL OIL #2 (330000 MG/KG)	UNKNOWN	TIER 2
MAPPED	3-0011646	NO LOCATION AID (RESIDNTIAL)	21 LAKEVIEW AVE	CAMBRIDGE	9/26/1994	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0011657	NO LOCATION AID (COMMERCIAL)	46 BLACKSTONE ST	CAMBRIDGE	9/9/1994	72 HR	DIESEL FUEL (50 PPMV); GASOLINE (50 PPMV); GASOLINE; FUEL OIL #6 (50 PPMV); FUEL OIL #6; FUEL OIL #2	UST	RAO
MAPPED	3-0011759	NO LOCATION AID	25-27 RESERVOIR ST	CAMBRIDGE	10/21/1994	72 HR	FUEL OIL #2	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0011772	NO LOCATION AID (COMMERCIAL)	239 PROSPECT ST	CAMBRIDGE	10/25/1994	72 HR	GASOLINE (83 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (83.4 PPM)	UST	RAO
MAPPED	3-0011809	NO LOCATION AID	28 DANA ST	CAMBRIDGE	11/2/1994	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0012068	BRISTON ARMS APTS (RESIDNTIAL)	247 GARDEN ST	CAMBRIDGE	1/13/1995	TWO HR	METHANE	BURIED SW	ADEQUATE REG
MAPPED	3-0012193	@ HARVARD BOATHOUSE (BOATHOUSE; WATERBODY)	245 MEMORIAL DR	CAMBRIDGE	2/22/1995	TWO HR	JET FUEL (100 GAL)	CRASH; FUELTANK; HELICOPTER	RAO
MAPPED	3-0012210	OFF CONNECTOR RD CORNER OF AMES COURT (COMMERCIAL)	100 BROADWAY	CAMBRIDGE	3/2/1995	72 HR	GASOLINE (200 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (1820 PPM)	UST	RAO
MAPPED	3-0012276	WISE POTATO CHIPS (COMMERCIAL)	141 RINDGE AVE	CAMBRIDGE	3/16/1995	72 HR	GASOLINE (200 PPMV); GASOLINE	UST	RAO
MAPPED	3-0012325	NO LOCATION AID (COMMERCIAL)	432 COLUMBIA ST	CAMBRIDGE	3/30/1995	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (10 GAL); FUEL OIL #6 (640 MG/KG)	TRANSFORM	RAO
MAPPED	3-0012329	HARVARD UNIVERSITY MORSE HALL (SCHOOL)	124 WALKER ST	CAMBRIDGE	3/31/1995	TWO HR	FUEL OIL #4 (30 GAL); FUEL OIL #4 (15 GAL)	FUELTANK	RAO
MAPPED	3-0012440	PORTION OF HARVARD UNIVERSITY (COMMERCIAL)	870 MEMORIAL DR	CAMBRIDGE	5/3/1995	72 HR	FUEL OIL #2 (443 PPMV)	UST	RAO
MAPPED	3-0012519	APARTMENT BUILDING (RESIDNTIAL)	16-19A FOREST ST	CAMBRIDGE	5/26/1995	72 HR	FUEL OIL #2; TOTAL PETROLEUM HYDROCARBONS (TPH)	UST	RAO
MAPPED	3-0012531	NO LOCATION AID (COMMERCIAL)	18-20 MOULTON ST	CAMBRIDGE	5/31/1995	72 HR	FUEL OIL #2 (300 PPMV)	UST	RAO
MAPPED	3-0012611	NO LOCATION AID (COMMERCIAL)	615 CONCORD AVE	CAMBRIDGE	6/23/1995	72 HR	FUEL OIL #2 (130 PPMV); FUEL OIL #2 (180 PPMV)	UST	RAO
MAPPED	3-0012699	NO LOCATION AID (COMMERCIAL)	87 FAWCETT ST	CAMBRIDGE	7/19/1995	72 HR	FUEL OIL #2 (290 PPMV)	UST	RAO
MAPPED	3-0012743	WOK EAST RESTAURANT (COMMERCIAL)	645 CAMBRIDGE ST	CAMBRIDGE	7/27/1995	TWO HR	VEGETABLE OIL (110 GAL)		RAO
MAPPED	3-0012856	CAMBRIDGE BRANDS (COMMERCIAL)	810 MAIN ST	CAMBRIDGE	8/25/1995	TWO HR	AMMONIA (40 GAL)	AST; PIPE	RAO
MAPPED	3-0012881	FMR MIDLAND ROSS SITE (COMMERCIAL)	445 CONCORD AVE	CAMBRIDGE	9/1/1995	72 HR	ETHENE, TRICHLORO- (2500); CHRYSENE (55000); TOTAL PETROLEUM HYDROCARBONS (TPH) (94000 MG/KG)	UNKNOWN	RTN CLOSED
MAPPED	3-0012885	NO LOCATION AID (COMMERCIAL; INDUSTRIAL)	810 MAIN ST	CAMBRIDGE	9/4/1995	TWO HR	AMMONIA (50 LBS)	PIPE	RAO
MAPPED	3-0012892	NO LOCATION AID (COMMERCIAL)	25 ACORN PARK	CAMBRIDGE	9/6/1995	72 HR	FUEL OIL #4 (142 PPMV)	UST	RAO
MAPPED	3-0012966	HAVILAND CANDY INC (COMMERCIAL; INDUSTRIAL)	134 CAMBRIDGE & FIRST STS	CAMBRIDGE	9/23/1995	TWO HR	AMMONIA (1991 LBS); AMMONIA (350 GAL)	PIPE; UNKNOWN	RAO
MAPPED	3-0012975	NO LOCATION AID (COMMERCIAL)	281 BROADWAY	CAMBRIDGE	9/27/1995	72 HR	UNKNOWN CHEMICAL OF TYPE - OIL	UST	RAO
MAPPED	3-0013105	U HAUL (COMMERCIAL)	849 MAIN ST	CAMBRIDGE	11/1/1995	TWO HR	FUEL OIL #2 (0.5 INCH)	AST	STMRET
MAPPED	3-0013203	NO LOCATION AID (RESIDNTIAL)	351 VASSAR ST	CAMBRIDGE	12/1/1995	72 HR	FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0013330	CAMBRIDGE HOUSING AUTHORITY (RESIDNTIAL)	900 CAMBRIDGE ST	CAMBRIDGE	1/13/1996	TWO HR	FUEL OIL #4 (6 INCH)	UST	RAO
MAPPED	3-0013335	JIMMYS FOREIGN AUTO (COMMERCIAL)	2055 MASSACHUSETTS AVE	CAMBRIDGE	1/17/1996	TWO HR	FUEL OIL #2 (50 GAL)	AST	RAO
MAPPED	3-0013599	YARD 7 RAIL FACILITY (RAIL SIDNG)	EAST ST	CAMBRIDGE	3/24/1996	TWO HR	DIESEL FUEL (200 GAL)	VEHICLE	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0013797	NO LOCATION AID (COMMERCIAL)	605 MT AUBURN ST	CAMBRIDGE	5/21/1996	72 HR	GASOLINE (287 PPM)	UST	TIER 2
MAPPED	3-0013866	STAR MARKET PARKING LOT REAR (COMMERCIAL)	699 MT AUBURN ST	CAMBRIDGE	6/6/1996	TWO HR	DIESEL FUEL (20 GAL)	PIPE; VEHICLE	RAO
MAPPED	3-0013933	NO LOCATION AID (COMMERCIAL)	812 MEMORIAL DR	CAMBRIDGE	6/25/1996	72 HR	FUEL OIL #2 (0.5 INCH)	UNKNOWN	RAO
MAPPED	3-0013987	HARRISON & LOYD (COMMERCIAL)	80-88 TROWBRIDGE ST	CAMBRIDGE	7/12/1996	72 HR	FUEL OIL #2 (105 PPMV); FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0014055	NO LOCATION AID (COMMERCIAL)	RIVER ST AND PUTNAM AVE	CAMBRIDGE	7/29/1996	TWO HR	FUEL OIL #2 (11 GAL); TOTAL PETROLEUM HYDROCARBONS (TPH) (1900 GAL)	UST	RAO
MAPPED	3-0014168	NEAR MASS AVE (COMMERCIAL)	844 MAIN ST	CAMBRIDGE	8/28/1996	72 HR	GASOLINE (436 PPMV)	UST	STMRET
MAPPED	3-0014217	GUEST QUARTERS HOTEL (WATERBODY)	WESTERN AVE BRIDGE	CAMBRIDGE	9/10/1996	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	DEF TIER 1B
MAPPED	3-0014310	LOT (COMMERCIAL)	RIVER & PUTNAM ST	CAMBRIDGE	10/8/1996	72 HR	FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0014329	CAMBRIDGE AUTO CLINIC (COMMERCIAL; ROADWAY)	297 CONCORD AVE	CAMBRIDGE	10/14/1996	TWO HR	GASOLINE (100 GAL)	TANKER	RAO
MAPPED	3-0014637	NO LOCATION AID (COMMERCIAL)	1 KENDALL SQ	CAMBRIDGE	12/17/1996	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (5.5 INCH); UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	RAO
MAPPED	3-0014643	RIVER STREET (COMMERCIAL)	44 BLACKSTONE ST	CAMBRIDGE	12/19/1996	TWO HR	FUEL OIL #6 (100 GAL)	AST; PIPE; PUMP SEAL	RAO
MAPPED	3-0014698	POLE 18 (ROADWAY)	58-64 HIGHLAND ST	CAMBRIDGE	1/7/1997	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (8 GAL)	TRANSFORM	RAO
MAPPED	3-0014706	NO LOCATION AID (COMMERCIAL)	287 PROSPECT ST	CAMBRIDGE	1/8/1997	72 HR	GASOLINE (7.5 INCH); PETROLEUM BASED OIL (0.5 INCH)	UNKNOWN	RTN CLOSED
MAPPED	3-0014930	GREENLEAF BLDG (SCHOOL)	76 BRATTLE ST	CAMBRIDGE	3/19/1997	TWO HR	DIESEL FUEL (60 GAL); DIESEL FUEL (25 GAL)	VEHICLE	RAO
MAPPED	3-0014935	ATLANTIC PAPER BOX (COMMERCIAL)	270 ALBANY ST	CAMBRIDGE	3/21/1997	72 HR	FUEL OIL #2 (120 PPMV); FUEL OIL #2 (2500 MG/KG); FUEL OIL #4 (2500 MG/KG)	UST	RAO
MAPPED	3-0015001	NO LOCATION AID (RESIDNTIAL)	169 CHILTON ST	CAMBRIDGE	4/14/1997	TWO HR	FUEL OIL #2 (70 GAL); FUEL OIL #2 (10 GAL)	AST	RAO
MAPPED	3-0015062	KENDALL TANK & BOILER (COMMERCIAL)	275 THIRD ST	CAMBRIDGE	5/1/1997	72 HR	FUEL OIL #2 (14 INCH); FUEL OIL #2 (300 MG/L)	UNKNOWN	RAO
MAPPED	3-0015190	SCHOOL (SCHOOL)	74R FAYERWEATHER ST	CAMBRIDGE	6/11/1997	72 HR	UNKNOWN CHEMICAL OF TYPE - OIL (10 PPM); BENZENE (2.5 PPM); TOTAL PETROLEUM HYDROCARBONS (TPH)	UST	RAO
MAPPED	3-0015211	NO LOCATION AID (RESIDNTIAL)	8 FOLLEN ST	CAMBRIDGE	6/16/1997	72 HR	FUEL OIL #2 (114 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0015243	KENDALL STATION (INDUSTRIAL)	265 FIRST ST	CAMBRIDGE	6/25/1997	TWO HR	FUEL OIL #6 (3000 GAL); FUEL OIL #6 (5000 GAL)	AST; PIPE	RAO
MAPPED	3-0015303	NO LOCATION AID (COMMERCIAL)	603 CONCORD AVE	CAMBRIDGE	7/16/1997	72 HR	WASTE OIL (438 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (438 PPM)	UST	TIER 2
MAPPED	3-0015332	NO LOCATION AID (COMMERCIAL)	30 CAMBRIDGE PARK DR	CAMBRIDGE	7/25/1997	72 HR	FUEL OIL #2 (6 INCH); FUEL OIL #2 (8 INCH)	UNKNOWN	RAO
MAPPED	3-0015351	NO LOCATION AID (COMMERCIAL)	287 PROSPECT ST	CAMBRIDGE	7/29/1997	72 HR	GASOLINE	UST	RTN CLOSED
MAPPED	3-0015367	CAMBRIDGE CHEVROLET (COMMERCIAL)	275 FRESH POND PKWY	CAMBRIDGE	7/31/1997	72 HR	FUEL OIL #2 (140 PPMV)	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0015448	MWRA PRISON PT INDUSTRIAL PARK RD (INDUSTRIAL)	1 MONSIGNOR OBRIEN HWY	CAMBRIDGE	8/20/1997	72 HR	FUEL OIL #2; FUEL OIL #2	UST	RTN CLOSED
NOT MAPPED	3-0015700	NO LOCATION AID (WATERBODY)	MEMORIAL DR	CAMBRIDGE	11/6/1997	TWO HR	GASOLINE	BOAT	RAO
MAPPED	3-0015765	MWRA INDUSTRIAL PARK RD (STATE)	1 MONSIGNER OBRIEN HWY	CAMBRIDGE	11/26/1997	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (3.6 INCH); OIL (3.6 INCH)	UST	RTN CLOSED
MAPPED	3-0015776	MDC (COMMERCIAL; RESIDNTIAL; STATE)	751 MEMORIAL DR	CAMBRIDGE	12/2/1997	72 HR	FUEL OIL #2 (113 PPMV)	UST	RAO
MAPPED	3-0015786	NO LOCATION AID (RESIDNTIAL)	48 KIRKLAND ST	CAMBRIDGE	12/4/1997	72 HR	FUEL OIL #2 (500 PPMV); FUEL OIL #2 (500 PPMV)	UST	RAO
MAPPED	3-0016014	NO LOCATION AID (INDUSTRIAL)	MILLERS RIVER	CAMBRIDGE	11/16/1999	TWO HR	OIL	SETTLING; TANK	DEF TIER 1B
MAPPED	3-0016604	MT AUBURN SER STAT (COMMERCIAL)	605 MT AUBURN ST	CAMBRIDGE	3/17/1998	72 HR	GASOLINE (30 INCH); OIL	UST	TIER 2
MAPPED	3-0016710	NO LOCATION AID (COMMERCIAL)	200 CAMBRIDGE PARK DR	CAMBRIDGE	4/17/1998	TWO HR	ASBESTOS; ASBESTOS (1 LBS)	UNKNOWN	RAO
MAPPED	3-0016735	REAR BIOCHEM BLDG (OPENSPACE; SCHOOL)	7 DIVINITY AVE	CAMBRIDGE	4/28/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (3000 GAL); UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	AST	RAO
MAPPED	3-0016739	CITY OF CAMBRIDGE (MUNICIPAL)	113 GARDEN ST	CAMBRIDGE	4/30/1998	72 HR	GASOLINE (186 PPMV); GASOLINE (112 PPMV)	UST	RAO
MAPPED	3-0016747	NO LOCATION AID (INDUSTRIAL)	315 VASSER ST	CAMBRIDGE	5/1/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (10 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL)	TRANSFORM	RAO
MAPPED	3-0016793	FRESH POND ROTARY (INDUSTRIAL)	CONVERGENCE OF RTES 2 16 AND 3	CAMBRIDGE	5/13/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (2100 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (3000 GAL)	PIPE	RAO
MAPPED	3-0016878	CAMBRIDGE SAVINGS BANK 469500000N325400E	1960 MASSACHUSETTS AVE	CAMBRIDGE	6/4/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (15 INCH); FUEL OIL #2 (0.5 INCH)	UNKNOWN	RTN CLOSED
MAPPED	3-0016883	KENNEDY SQUARE (RESIDNTIAL; ROADWAY)	CRAIGIE ST	CAMBRIDGE	6/5/1998	TWO HR	GASOLINE (10 GAL); DIESEL FUEL (80 GAL)	TANKER	RAO
MAPPED	3-0016997	ACCESS RD (RR TRACKS; SCHOOL)	222 AND 224 ALBANY ST	CAMBRIDGE	7/2/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (40 GAL)	UNKNOWN	RAO
MAPPED	3-0017013	ADJACENT TO 12 GERRYS LANDING (UTILITY)	FRESH POND PKWY @ MT AUBURN ST	CAMBRIDGE	7/8/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (1400 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (8316 GAL)	PIPE	RAO
MAPPED	3-0017107	B&M RAILROAD (INDUSTRIAL)	WATER ST @ B&M RR YARD	CAMBRIDGE	7/31/1998	TWO HR	HYDROCHLORIC ACID (500 GAL)	CAR; RR TANK	RAO
MAPPED	3-0017136	REAR SCISSOR LIFT (COMMERCIAL)	87 CAMBRIDGEPARK DR	CAMBRIDGE	8/10/1998	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); DIESEL FUEL (10 GAL)	AST; PIPE	RAO
MAPPED	3-0017214	BLACKSTONE STATION OUTFALL (WATERBODY)	MEMORIAL DR @ RIVER ST	CAMBRIDGE	8/25/1998	TWO HR	OIL; LUBRICATING OIL (1 GAL)	PIPE	RAO
MAPPED	3-0017229	NO LOCATION AID (COMMERCIAL)	30 CAMBRIDGEPARK DR	CAMBRIDGE	8/28/1998	72 HR	FUEL OIL #2 (330 PPMV)	UST	RAO
MAPPED	3-0017273	CORNER OF MUNROE (COMMERCIAL)	303 THIRD ST	CAMBRIDGE	9/10/1998	72 HR	PETROLEUM BASED OIL (120 PPMV); GASOLINE (100 PPMV); DIESEL FUEL (100 PPMV)	UST	RAO
MAPPED	3-0017298	MAGAZINE BEACH VETERANS MEMORIAL POOL (STATE)	MEMORIAL DR	CAMBRIDGE	9/15/1998	TWO HR	OIL (10 GAL); FUEL OIL #2 (10 GAL)	UNKNOWN	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0017299	NO LOCATION AID (RESIDNTIAL)	38 MIDDLESEX ST	CAMBRIDGE	9/20/1998	72 HR	FUEL OIL #2; FUEL OIL #2 (11000 MG/KG); FUEL OIL #2 (24 MG/L)	PIPE	RAO
MAPPED	3-0017455	NO LOCATION AID (COMMERCIAL)	10 NORTH POINT BLVD	CAMBRIDGE	10/21/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE; GASOLINE (110 PPMV); DIESEL FUEL	UST	TIER 2
MAPPED	3-0017637	NO LOCATION AID (COMMERCIAL)	60 ABERDEEN AVE	CAMBRIDGE	11/25/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (24 INCH); FUEL OIL #6 (0.5 INCH); FUEL OIL #4 (0.5 INCH)	FMR; UST	RAO
MAPPED	3-0017750	BU BOATHOUSE CONSTRUCTION	619 MEMORIAL DR	CAMBRIDGE	12/16/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL; OIL	UNKNOWN	RAO
MAPPED	3-0017753	NO LOCATION AID (COMMERCIAL)	254 MASSACHUSETTS AVE	CAMBRIDGE	12/18/1998	72 HR	FUEL OIL #6 (6 INCH); FUEL OIL #6 (1.5 INCH)	UST	TIER 2
MAPPED	3-0017819	NO LOCATION AID (MUNICIPAL)	48 SIXTH ST	CAMBRIDGE	1/5/1999	72 HR	FUEL OIL #2 (118 PPMV); FUEL OIL #2 (121 PPMV)	UST	RAO
MAPPED	3-0017884	NO LOCATION AID (SCHOOL)	38 OXFORD ST	CAMBRIDGE	1/22/1999	TWO HR	HYDROGEN PEROXIDE (H2O2) (5 GAL)	5 GAL PAIL	RAO
MAPPED	3-0018096	NO LOCATION AID (COMMERCIAL)	75 SIDNEY ST	CAMBRIDGE	3/22/1999	TWO HR	PETROLEUM BASED OIL (10 GAL); PETROLEUM BASED OIL (13 GAL)	PIPE	RAO
MAPPED	3-0018133	RADCLIFFE QUAD ATLETIC BUILDING (SCHOOL)	60 GARDEN ST	CAMBRIDGE	3/29/1999	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (1 INCH)	ELEVATOR; SYSTEM	RAO
MAPPED	3-0018161	NO LOCATION AID (ROADWAY)	BINNEY & THIRD STS	CAMBRIDGE	4/5/1999	TWO HR	DIESEL FUEL (75 GAL); DIESEL FUEL (60 GAL)	VEHICLE	RAO
MAPPED	3-0018261	NO LOCATION AID (COMMERCIAL)	975 MASSACHUSETTS AVE	CAMBRIDGE	5/4/1999	72 HR	GASOLINE (1000 PPMV); GASOLINE (100 PPMV)	UST	RAO
MAPPED	3-0018477	NO LOCATION AID (COMMERCIAL)	985 MEMORIAL DR	CAMBRIDGE	7/2/1999	72 HR	FUEL OIL #2 (138 PPMV); FUEL OIL #2	UST	RAO
MAPPED	3-0018479	NO LOCATION AID (RESIDNTIAL; ROADWAY)	192 ELM ST	CAMBRIDGE	7/6/1999	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (15 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (13 GAL); 1,1'-BIPHENYL, CHLORO-DERIVS. (130 MG/L); 1,1'-BIPHENYL, CHLORO-DERIVS. (131 PPM)	TRANSFORM	RAO
MAPPED	3-0018656	NO LOCATION AID (COMMERCIAL)	1012 CAMBRIDGE PL	CAMBRIDGE	8/18/1999	TWO HR	PETROLEUM BASED OIL (30 GAL); PETROLEUM BASED OIL (10 GAL)	PIPE; VEHICLE	RAO
MAPPED	3-0018804	NO LOCATION AID (COMMERCIAL)	15 CAMBRIDGE CTR	CAMBRIDGE	9/28/1999	72 HR	PETROLEUM BASED OIL (165 PPMV)	UST	RTN CLOSED
MAPPED	3-0018884	BROADWAY (COMMERCIAL)	90 ELLERY ST	CAMBRIDGE	10/26/1999	72 HR	FUEL OIL #2 (116 PPMV); FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0019147	MIT (SCHOOL; UNIVERSITY)	304 VASSAR ST	CAMBRIDGE	1/10/2000	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); PETROLEUM BASED OIL (0.5 INCH)	ELEVATOR	RAO
MAPPED	3-0019168	NO LOCATION AID (SCHOOL)	60 ALBANY ST	CAMBRIDGE	1/16/2000	TWO HR	FUEL OIL #6 (200 GAL); FUEL OIL #6 (100 GAL)	PIPE; UST	RAO
NOT MAPPED	3-0019315	MYSTIC RIVER (WATERBODY)	ALEWIFE BROOK	CAMBRIDGE	2/24/2000	TWO HR	OIL	UNKNOWN	RAO
NOT MAPPED	3-0019649	NO LOCATION AID (ROADWAY)	CAMBRIDGE ST	CAMBRIDGE	6/19/2000	TWO HR	PETROLEUM BASED OIL (20 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	PIPE; VEHICLE	RAO
MAPPED	3-0019837	HAVILAND CANDY INC (INDUSTRIAL)	134 CAMBRIDGE ST	CAMBRIDGE	8/15/2000	TWO HR	AMMONIA; AMMONIA (10 LBS)	PIPE; UNKNOWN	RAO
MAPPED	3-0019919	NO LOCATION AID (COMMERCIAL)	111 CHARLES ST	CAMBRIDGE	9/8/2000	72 HR	GASOLINE (800 PPMV); PETROLEUM BASED OIL (200 PPM)	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0020011	MIT CORNER OF VASSAR AND MAIN STS (COMMERCIAL; SCHOOL)	10 TO 40 VASSAR ST	CAMBRIDGE	10/4/2000	TWO HR	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (100 GAL); KEROSENE (100 GAL)	UST	RTN CLOSED
MAPPED	3-0020020	THIRD ST AND LINSKEY WAY (COMMERCIAL; INDUSTRIAL; WATERBODY)	364 THIRD ST	CAMBRIDGE	10/6/2000	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (5 GAL); OIL	FAILED; SYSTEM; TREATMENT	RTN CLOSED
MAPPED	3-0020057	NEAR INTERSECTION OF HANCOCK AND HARVARD (COMMERCIAL)	149 HANCOCK ST	CAMBRIDGE	10/25/2000	TWO HR	FUEL OIL #2 (10 GAL); FUEL OIL #2 (22 GAL)	PIPE	RAO
MAPPED	3-0020174	MASSACHUSETTS AVE (APARTMENTS; RESIDNTIAL)	9 ELLERY ST	CAMBRIDGE	12/3/2000	TWO HR	FUEL OIL #2 (10 GAL); FUEL OIL #2 (20 GAL)	AST; UST	RAO
MAPPED	3-0020202	NO LOCATION AID (RESIDNTIAL)	20 HIGHLAND AVE	CAMBRIDGE	12/8/2000	72 HR	FUEL OIL #2 (230 PPMV); FUEL OIL #2 (230 PPM)	UST	RAO
MAPPED	3-0020305	NSTAR ELECTRIC POWER SUBSTATION (RESIDNTIAL)	EDMUNDS ST & MASS AVE CORNER	CAMBRIDGE	1/12/2001	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (40 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	TRANSFORM	RAO
MAPPED	3-0020315	NSTAR ELECTRIC POWER SUBSTATION (COMMERCIAL)	EDMUNDS ST & MASS AVE CORNER	CAMBRIDGE	1/13/2001	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (35 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (70 GAL)	TRANSFORM	RAO
MAPPED	3-0020317	SOUTH SIDE OF GILMORE BRIDGE (ROADWAY)	CHARLESTOWN AVE	CAMBRIDGE	1/16/2001	TWO HR	FUEL OIL #6 (15 GAL); FUEL OIL #6 (15 GAL)	TANKER	RAO
MAPPED	3-0020321	DEPT OF PUBLIC WORKS (COMMERCIAL)	147 HAMPSHIRE ST	CAMBRIDGE	1/18/2001	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (15 GAL); DIESEL FUEL (15 GAL)	PIPE; SCHOOLBUS	RAO
MAPPED	3-0020346	CORNER CAMERON AND MASS AVE (COMMERCIAL)	5 CAMERON AVE	CAMBRIDGE	1/24/2001	TWO HR	ETHENE, TETRACHLORO-; ETHENE, TETRACHLORO- (87.5 GAL)	UNKNOWN	RTN CLOSED
MAPPED	3-0020386	NO LOCATION AID (COMMERCIAL)	265 FIRST ST	CAMBRIDGE	2/12/2001	TWO HR	SULFURIC ACID (5 GAL); SULFURIC ACID (20 GAL)	AST; PIPE	RAO
MAPPED	3-0020420	CORNER OF DUDLEY ST (ROADWAY)	2366 MASSACHUSETTS AVE	CAMBRIDGE	2/22/2001	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (12 GAL); DIESEL FUEL (15 GAL)	VEHICLE	RAO
MAPPED	3-0020577	SUNOCO (COMMERCIAL)	515 CONCORD AVE	CAMBRIDGE	4/6/2001	TWO HR	PETROLEUM BASED OIL (40 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE	RTN CLOSED
MAPPED	3-0020609	AT CORNER OF HARVARD ST (COMMERCIAL)	191 TO 193 WINDSOR ST	CAMBRIDGE	4/20/2001	TWO HR	FUEL OIL #2; WASTE OIL (477 PPM)	UST	RAO
MAPPED	3-0020736	US PETROLEUM GAS STATION (COMMERCIAL)	297 CONCORD AVE	CAMBRIDGE	5/25/2001	72 HR	GASOLINE (340 PPMV)	UST	TIER 2
MAPPED	3-0020744	NO LOCATION AID (COMMERCIAL)	820 MEMORIAL DR	CAMBRIDGE	5/29/2001	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE		TIER 2
MAPPED	3-0020881	SIDNEY ST	70 PACIFIC ST	CAMBRIDGE	7/6/2001	TWO HR	DIESEL FUEL (50 GAL); DIESEL FUEL (30 GAL)	VEHICLE	RAO
MAPPED	3-0020988	NSTAR STA (INDUSTRIAL)	TERMINAL RD	CAMBRIDGE	8/9/2001	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (1000 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (1700 GAL)	PIPE	RAO
MAPPED	3-0021004	LOADING DOCK ON BLANCHE ST (COMMERCIAL; INDUSTRIAL)	38 SIDNEY ST	CAMBRIDGE	8/16/2001	TWO HR	SODIUM HYDROXIDE (55 LBS)	DRUMS	RAO
MAPPED	3-0021097	NO LOCATION AID (RESIDNTIAL)	325-345 FRANKLIN ST	CAMBRIDGE	9/20/2001	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (282 PPMV); UNKNOWN CHEMICAL OF UNKNOWN TYPE (282 MG/KG)	UST	RAO
MAPPED	3-0021252	NO LOCATION AID (INDUSTRIAL)	125 BROOKLINE ST	CAMBRIDGE	11/15/2001	72 HR	PETROLEUM BASED OIL (8.5 INCH)	UNKNOWN	UNCLASSIFIED

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0021256	NO LOCATION AID (ROADWAY; STATE)	MEMORIAL DR AT MASS AVE	CAMBRIDGE	11/15/2001	TWO HR	PETROLEUM BASED OIL (20 GAL)	VEHICLE	RAO
MAPPED	3-0021265	MIT (COMMERCIAL; SCHOOL)	60 ALBANY ST	CAMBRIDGE	11/25/2001	TWO HR	FUEL OIL #6 (60 GAL); FUEL OIL #6 (60 GAL)	PIPE; PUMP	RAO
MAPPED	3-0021556	N42 DEGREES E71 DEGREES (INDUSTRIAL)	87 CAMBRIDGE PARK DR	CAMBRIDGE	3/8/2002	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (75 GAL); DIESEL FUEL (30 GAL)	AST	RAO
MAPPED	3-0021574	HARVARD U OXFORD ST PLAYGROUND (OPENSPACE)	FRANCES AVE	CAMBRIDGE	3/14/2002	TWO HR	ARSENIC (46 PPM); ARSENIC (48 MG/KG); LEAD (550 MG/KG)	UNKNOWN	RAO
MAPPED	3-0021751	NECCO (INDUSTRIAL)	254 MASSACHUSETTS AVE	CAMBRIDGE	5/12/2002	TWO HR	AMMONIA; AMMONIA (571 LBS); OIL	PIPE	RAO
MAPPED	3-0021939	ELECTRICAL SERVICE BAY FACING BENT ST (COMMERCIAL)	320 CHARLES ST	CAMBRIDGE	7/15/2002	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (50 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (100 GAL)	TRANSFORM	RAO
MAPPED	3-0021947	EDMUNDS ST (COMMERCIAL)	2485 MASS AVE	CAMBRIDGE	7/18/2002	72 HR	PETROLEUM BASED OIL (12 INCH); PETROLEUM BASED OIL (13.56 INCH)	UNKNOWN	UNCLASSIFIED
MAPPED	3-0022002	WEST OF MT. AUBURN HOSPITAL (STATE; WATERBODY)	GERRYS LANDING ROAD	CAMBRIDGE	8/6/2002	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (2200 GAL); MINERAL OIL (10000 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022050	NO LOCATION AID (ROADWAY)	2192 MASS AVE	CAMBRIDGE	8/25/2002	TWO HR	DIESEL FUEL (100 GAL)	VEHICLE	UNCLASSIFIED
MAPPED	3-0022080	NO LOCATION AID (COMMERCIAL)	148 SYDNEY ST	CAMBRIDGE	9/6/2002	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #2 (18 INCH); OIL (18 INCH)	UST	UNCLASSIFIED
MAPPED	3-0022116	NO LOCATION AID (RESIDNTIAL)	66 HOMER AVE	CAMBRIDGE	9/17/2002	72 HR	FUEL OIL #2 (286 PPMV); TOTAL PETROLEUM HYDROCARBONS (TPH) (286 MG/KG)	UST	UNCLASSIFIED
MAPPED	3-0022185	NO LOCATION AID (COMMERCIAL)	WHEELER ST AND RTE 2	CAMBRIDGE	10/5/2002	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (2000 GAL); MINERAL OIL (1500 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022270	NO LOCATION AID (COMMERCIAL)	740 TO 770 MAIN ST	CAMBRIDGE	11/1/2002	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #6 (100 PPMV)	UST	RAO
MAPPED	3-0022369	NO LOCATION AID (COMMERCIAL)	2505 MASSACHUSETTS AVE	CAMBRIDGE	12/6/2002	72 HR	PETROLEUM BASED OIL (3 INCH); UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (3 INCH)	UST	UNCLASSIFIED
MAPPED	3-0022376	NO LOCATION AID (COMMERCIAL)	127 SMITH PLACE	CAMBRIDGE	12/10/2002	TWO HR	FUEL OIL #2 (20 GAL); FUEL OIL #2 (13300 MG/KG)	AST	UNCLASSIFIED
MAPPED	3-0022441	HARVARD UNIV (SCHOOL)	1737 CAMBRIDGE ST	CAMBRIDGE	1/2/2003	TWO HR	FUEL OIL #2; FUEL OIL #6 (30 GAL)	UST	UNCLASSIFIED
MAPPED	3-0022509	WR GRACE (COMMERCIAL; INDUSTRIAL)	62 WHITTEMORE AVE	CAMBRIDGE	1/22/2003	TWO HR	ASBESTOS	BURIED ACM	UNCLASSIFIED
MAPPED	3-0022639	BLDG 15 (COMMERCIAL)	15 ACORN PARK	CAMBRIDGE	3/3/2003	TWO HR	FUEL OIL #4 (200 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022673	SUNOCO (COMMERCIAL)	266 MASSACHUSETTS AVE	CAMBRIDGE	3/14/2003	TWO HR	GASOLINE (15 GAL)	PIPE	UNCLASSIFIED
MAPPED	3-0022752	HARVARD UNIVERSITY (SCHOOL)	29 GARDEN ST	CAMBRIDGE	4/7/2003	72 HR	OIL	UNKNOWN	UNCLASSIFIED
MAPPED	3-0010080	GETTY SERVICE STATION (COMMERCIAL)	110 GALEN ST	WATERTOWN	10/21/1993	72 HR	WASTE OIL; UNKNOWN CHEMICAL OF TYPE - OIL (0.1 GAL)	UNKNOWN	TIER 2
MAPPED	3-0010154	SUNOCO SERVICE STATION (COMMERCIAL)	170 GALEN ST	WATERTOWN	11/5/1993	72 HR	FUEL OIL #2 (50 PPMV); GASOLINE (50 PPMV); WASTE OIL (50 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (1133 PPM)	UST	TIER 2
MAPPED	3-0010381	BOSTON EDISON SERVICE CENTER (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	12/30/1993	72 HR	OIL (0.48 INCH); UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	RAO
MAPPED	3-0010461	US ARMY RESEARCH LAB (INDUSTRIAL)	WATERTOWN ARSENAL	WATERTOWN	1/19/1994	TWO HR	FUEL OIL #6 (50 GAL)	PIPE	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0010598	MJ ROLLIE (COMMERCIAL)	56 IRVING ST	WATERTOWN	2/18/1994	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (13 GAL); PETROLEUM BASED OIL (17.9 GAL)	DRUMS	RAO
MAPPED	3-0011565	INTERSECTION OF (COMMERCIAL)	N BEACON AND ARSENAL ST	WATERTOWN	9/6/1994	TWO HR	PETROLEUM BASED OIL (1200 GAL); UNKNOWN CHEMICAL OF TYPE - OIL (1226 GAL)	PIPE; UST	TIER 2
MAPPED	3-0011799	OFF AUBURN ST ROUTE 16 REAR OF BUILDING (COMMERCIAL)	30 WASHBURN ST	WATERTOWN	10/26/1994	72 HR	OIL (1.5 INCH); UNKNOWN CHEMICAL OF UNKNOWN TYPE	AST; DRUMS; FUELTANK; PIPE; UST	TIER 2
MAPPED	3-0011882	GAS STATION 22084720303 ON ROUTE 20 (COMMERCIAL)	448 MAIN ST	WATERTOWN	11/22/1994	72 HR	GASOLINE (280 PPMV); GASOLINE (360 PPM)	UST	RAO
MAPPED	3-0012009	NO LOCATION AID (RESIDNTIAL)	31 BACON ST	WATERTOWN	12/27/1994	TWO HR	FUEL OIL #2 (50 GAL); FUEL OIL #2 (200 PPM)	AST	RAO
MAPPED	3-0012132	FMR WATERTOWN ARSENAL NOW ARSENAL PARK (FEDERAL)	ARSENAL ST	WATERTOWN	2/3/1995	TWO HR	ARSENIC (66 MG/KG); ARSENIC (30 MG/KG); ACENAPHTHYLENE, 1,2-DIHYDRO (3.1 UG/G); BENZ[A]ANTHRACENE (1.3 UG/G); BENZO(K)FLUORANTHENE (12 UG/G); BENZ[E]ACEPHENANTHRYLENE (13 UG/G); BENZO[A]PYRENE (9.8 UG/G); CHRYSENE (11 UG/G); INDENO(1,2,3-CD)PYRENE (3.3 UG	UNKNOWN	TIER 1C
MAPPED	3-0012488	UST AREA (COMMERCIAL; INDUSTRIAL)	29 BRIDGE ST REAR	WATERTOWN	5/18/1995	72 HR	FUEL OIL #4 (100 PPMV); FUEL OIL #4	UST	RAO
MAPPED	3-0012808	NO LOCATION AID (INDUSTRIAL)	480 ARSENAL ST	WATERTOWN	10/4/1995	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (18 GAL)	PIPE	RAO
MAPPED	3-0012861	NO LOCATION AID (INDUSTRIAL)	405 ARSENAL ST	WATERTOWN	8/29/1995	TWO HR	MERCURY (3 LBS)	GAUGE	RAO
MAPPED	3-0012996	CROSS ST ROSEDALE RD (COMMERCIAL)	380 PLEASANT ST	WATERTOWN	10/2/1995	72 HR	CUTTING OIL (1.75 INCH)	UNKNOWN	TIER 2
MAPPED	3-0013088	NYNEX CENTRAL OFFICE (COMMERCIAL)	143 WALNUT ST	WATERTOWN	10/26/1995	72 HR	KEROSENE (160 PPMV)	UST	RAO
MAPPED	3-0013339	WET OF SCHOOL ST (COMMERCIAL)	314 ARSENAL ST	WATERTOWN	1/18/1996	72 HR	UNKNOWN CHEMICAL OF TYPE - OIL (120 PPMV)	UST	RAO
MAPPED	3-0013746	NO LOCATION AID (COMMERCIAL)	70-80 GROVE ST	WATERTOWN	5/10/1996	72 HR	PETROLEUM BASED OIL (18 INCH); FUEL OIL #6	UNKNOWN	RTN CLOSED
MAPPED	3-0013776	NO LOCATION AID (COMMERCIAL)	74 ACTON ST	WATERTOWN	5/17/1996	72 HR	FUEL OIL #2; FUEL OIL #4; FUEL OIL #6; PETROLEUM BASED OIL (100 PPM)	UST	RTN CLOSED
MAPPED	3-0014273	ARSENAL PARK (OPENSPACE)	455-663 ARSENAL ST	WATERTOWN	9/25/1996	TWO HR	ARSENIC (209 PPMV); ARSENIC (181 MG/KG)	UNKNOWN	RTN CLOSED
MAPPED	3-0014713	ARSENAL MALL (COMMERCIAL)	485 ARSENAL ST	WATERTOWN	1/9/1997	TWO HR	1,1'-BIPHENYL, CHLORO-DERIVS. (15 PPMV); 1,1'-BIPHENYL, CHLORO-DERIVS. (15 PPM)	UNKNOWN	RTN CLOSED
MAPPED	3-0015315	SCARBOROUGHT REALTY TRUST (COMMERCIAL)	20 BRIDGE ST	WATERTOWN	7/18/1997	72 HR	OIL (8 INCH); FUEL OIL #4 (0.5 INCH)	UST	RTN CLOSED
MAPPED	3-0015418	GETTY SER STA (COMMERCIAL)	110 GALEN ST	WATERTOWN	8/14/1997	72 HR	GASOLINE (260 PPMV)	UST	RTN CLOSED
MAPPED	3-0015541	BLDG 60 (FEDERAL)	395 ARSENAL ST	WATERTOWN	9/18/1997	TWO HR	DIESEL FUEL (125 GAL); DIESEL FUEL (100 GAL)	DRUMS	RAO
MAPPED	3-0015590	YACHT CLUB (COMMERCIAL)	425 CHARLES RIVER RD	WATERTOWN	10/3/1997	72 HR	GASOLINE (450 PPMV); GASOLINE (100 PPMV)	UST	RAO
MAPPED	3-0015600	NO LOCATION AID (RESIDNTIAL)	27 CAREY AVE	WATERTOWN	10/7/1997	72 HR	FUEL OIL #2 (245 PPMV); FUEL OIL #2 (245 PPMV)	UST	RAO
MAPPED	3-0015763	NO LOCATION AID (COMMERCIAL)	656 MAIN ST	WATERTOWN	11/26/1997	72 HR	GASOLINE (100 PPMV); GASOLINE (180 PPMV); NAPHTHALENE (25 PPM)	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0015785	MAIN ST (RESIDNTIAL)	86-88 EDENFIELD AVE	WATERTOWN	12/4/1997	TWO HR	FUEL OIL #2 (100 GAL); FUEL OIL #2 (120 GAL)	AST	RAO
MAPPED	3-0016549	STOP AND SHOP (COMMERCIAL)	700 PLEASANT ST	WATERTOWN	2/27/1998	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	PIPE; UNKNOWN; VEHICLE	RAO
NOT MAPPED	3-0016569	OUTFALL #12 (WATERBODY)	CHARLES RIVER RD	WATERTOWN	3/7/1998	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL		RAO
MAPPED	3-0016601	COOLIDGE SQ SERVICE MELENDY AVE (COMMERCIAL)	575 TO 577 MT AUBURN ST	WATERTOWN	3/16/1998	72 HR	DIESEL FUEL (410 PPMV); GASOLINE (410 PPMV)	UST	RTN CLOSED
MAPPED	3-0016607	42 AND 47 MAIN ST (COMMERCIAL; RESIDNTIAL)	MAIN ST AT BECO MANHOLE 3706	WATERTOWN	3/18/1998	TWO HR	DIESEL FUEL (30 GAL); DIESEL FUEL (20 GAL)	BUS FUEL; TANK	RAO
MAPPED	3-0016821	BETWEEN BRIDGE AND SEYON STS (COMMERCIAL)	555 PLEASANT ST	WATERTOWN	5/18/1998	TWO HR	AMMONIA (17 LBS); AMMONIA (26 LBS)	PIPE	RAO
MAPPED	3-0017084	NO LOCATION AID (COMMERCIAL)	64 GROVE ST	WATERTOWN	7/24/1998	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #2 (100 PPMV)	UST	RAO
MAPPED	3-0017393	ROUTE 20 (ROADWAY)	MAIN & MIDDLE @ WATERTOWN SQ	WATERTOWN	10/8/1998	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (70 GAL)	VEHICLE	RAO
MAPPED	3-0017443	NO LOCATION AID (RESIDNTIAL)	10-12 HUNT ST	WATERTOWN	10/16/1998	TWO HR	FUEL OIL #2 (250 GAL); FUEL OIL #2 (250 GAL)	AST	RAO
MAPPED	3-0017528	NO LOCATION AID (COMMERCIAL)	60 TO 70 NORTH BEACON ST	WATERTOWN	11/5/1998	72 HR	GASOLINE (130 PPMV); GASOLINE (135 PPM)	UST	RTN CLOSED
MAPPED	3-0017608	BARDON TRIMOUNT (COMMERCIAL)	105 COOLIDGE HILL RD	WATERTOWN	11/20/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (310 PPMV); DIESEL FUEL (315 PPMV)	UST	RAO
MAPPED	3-0017620	NO LOCATION AID (COMMERCIAL)	626 ARSENAL ST	WATERTOWN	11/23/1998	72 HR	GASOLINE (1120 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (1170 PPMV)	UST	RAO
MAPPED	3-0017672	INTERSECTION WITH PATTEN ST (COMMERCIAL)	76 ARSENAL ST	WATERTOWN	12/3/1998	72 HR	FUEL OIL #2 (180 PPMV); OIL (226 PPMV)	UST	TIER 2
MAPPED	3-0017719	BOSTON EDISON COMPANY (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	12/10/1998	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (400 PPMV); DIESEL FUEL (373 PPM)	UST	RAO
MAPPED	3-0017833	A RUSSO & SONS INC (COMMERCIAL)	560 PLEASANT ST	WATERTOWN	1/7/1999	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL); DIESEL FUEL (20 GAL)	SADDLETNK; VEHICLE	RAO
MAPPED	3-0017896	FMR EXXON (COMMERCIAL)	14 ARSENAL 11 TO 13 MT AUBURN	WATERTOWN	1/22/1999	72 HR	GASOLINE (513 PPMV); GASOLINE (212 PPMV)	UNKNOWN	RAO
MAPPED	3-0017958	NO LOCATION AID (COMMERCIAL)	14 ARSENAL 11 TO 13 MT AUBURN	WATERTOWN	2/8/1999	TWO HR	GASOLINE; UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0017982	NO LOCATION AID (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	2/12/1999	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL)	TRANSFORM	RAO
MAPPED	3-0018144	EVERETT AVE (RESIDNTIAL)	194 PALFREY ST	WATERTOWN	6/9/1999	TWO HR	FUEL OIL #2 (100 GAL); PETROLEUM BASED OIL (150 GAL)	AST	RAO
MAPPED	3-0018308	AT TAYLOR ST (COMMERCIAL)	49 TO 59 MT AUBURN ST	WATERTOWN	5/17/1999	72 HR	PETROLEUM BASED OIL (16 INCH); PETROLEUM BASED OIL	UST	RTN CLOSED
MAPPED	3-0018471	BIGELOW AVE (COMMERCIAL)	631 MT AUBURN ST	WATERTOWN	7/1/1999	72 HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (12 INCH); ACENAPHTHYLENE, 1,2-DIHYDRO (51 MG/KG); 2-METHYLNAPHTHALENE (131 MG/KG); NAPHTHALENE (8.8 MG/KG)	UNKNOWN	TIER 2
MAPPED	3-0018590	14 ARSENAL ST (COMMERCIAL)	11 TO 13 MT AUBURN ST	WATERTOWN	8/4/1999	TWO HR	GASOLINE	UST	RTN CLOSED
MAPPED	3-0018592	CONSTRUCTION SITE (COMMERCIAL)	9 GALEN ST	WATERTOWN	8/4/1999	72 HR	GASOLINE (1300 PPMV)	UST	RAO

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
MAPPED	3-0018860	HIGH SCHOOL (SCHOOL)	50 COLUMBIA ST	WATERTOWN	10/19/1999	TWO HR	FUEL OIL #2 (100 GAL); FUEL OIL #2 (100 GAL)	PIPE	RAO
MAPPED	3-0018876	BIGELOW AVE (COMMERCIAL)	631 MT AUBURN ST	WATERTOWN	10/25/1999	72 HR	FUEL OIL #2 (100 PPMV); FUEL OIL #2 (100 PPMV)	UST	RTN CLOSED
MAPPED	3-0018880	ORCHARD ST (COMMERCIAL)	917 BELMONT ST	WATERTOWN	10/25/1999	TWO HR	DIESEL FUEL (150 GAL); DIESEL FUEL (100 GAL)	VEHICLE	RAO
MAPPED	3-0019013	NO LOCATION AID (COMMERCIAL)	75 NORTH BEACON ST	WATERTOWN	11/29/1999	72 HR	GASOLINE (192 PPMV); GASOLINE (100 PPMV)	UST	RAO
MAPPED	3-0019344	ARSENAL PARK (COMMERCIAL)	ARSENAL ST	WATERTOWN	3/9/2000	TWO HR	ASBESTOS	ASBESTOS; PIPE	DEF TIER 1B
MAPPED	3-0019544	NEAR NONANTUM RD (COMMERCIAL)	2 GALEN ST	WATERTOWN	5/15/2000	72 HR	FUEL OIL #2 (2 INCH); UNKNOWN CHEMICAL OF TYPE - OIL (2 INCH)	UST	TIER 2
MAPPED	3-0019625	INTERSECT WITH WILLIAMS ST (COMMERCIAL)	170 GALEN ST	WATERTOWN	6/14/2000	72 HR	UNKNOWN CHEMICAL OF TYPE - OIL (29 PPM); GASOLINE	UNKNOWN	RTN CLOSED
MAPPED	3-0019683	NO LOCATION AID (OPENSPACE)	158 WALTHAM ST	WATERTOWN	4/11/2002	72 HR	GASOLINE (2000 PPMV); GASOLINE (100 PPM)	UST	RAO
MAPPED	3-0019698	NO LOCATION AID (COMMERCIAL)	76 ARSENAL ST	WATERTOWN	7/7/2000	72 HR	OIL (6 INCH)	UNKNOWN	RTN CLOSED
MAPPED	3-0020350	BIRCH ST PRIVATE (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	1/25/2001	72 HR	NAPHTHALENE, 2-CHLORO- (300 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (300 PPM)	UST	RAO
MAPPED	3-0020433	BIRCH ST PRIVATE (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	3/2/2001	TWO HR	FUEL OIL #6 (60 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (60 GAL)	UST	RAO
MAPPED	3-0020501	WATERTOWN MALL REAR OF BEST BUY (COMMERCIAL)	550 ARSENAL ST	WATERTOWN	3/20/2001	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL (100 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (200 GAL)	TRANSFORM	RAO
MAPPED	3-0020623	OFF BIRTH ST PRIVATE (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	4/24/2001	72 HR	GASOLINE (200 PPMV); UNKNOWN CHEMICAL OF TYPE - OIL (350 PPM)	UST	RAO
MAPPED	3-0020765	OFF BIRCH ST PRIVATE (COMMERCIAL)	480 ARSENAL ST	WATERTOWN	6/6/2001	72 HR	PETROLEUM BASED OIL; UNKNOWN CHEMICAL OF TYPE - OIL (350 PPM)	UST	RAO
MAPPED	3-0020833	HIGH SCHOOL BOILER ROOM (WATERBODY)	50 COLUMBIA ST	WATERTOWN	6/23/2001	TWO HR	FUEL OIL #2 (1000 GAL); FUEL OIL #2 (600 GAL)	PIPE	RAO
MAPPED	3-0021258	NO LOCATION AID (COMMERCIAL)	270 PLEASANT ST	WATERTOWN	11/16/2001	72 HR	FUEL OIL #6 (36 INCH); FUEL OIL #6	UST	UNCLASSIFIED
MAPPED	3-0021328	NO LOCATION AID (OPENSPACE)	30 CALIFORNIA ST	WATERTOWN	12/12/2001	TWO HR	PETROLEUM BASED OIL	UNKNOWN	RAO
MAPPED	3-0021419	NO LOCATION AID (INDUSTRIAL)	58 IRVING ST	WATERTOWN	1/23/2002	TWO HR	FUEL OIL #2; FUEL OIL #2 (30 GAL)	UST	RAO
MAPPED	3-0021509	NO LOCATION AID (RESIDNTIAL)	110 COOLIDGE HILL RD	WATERTOWN	2/22/2002	72 HR	PETROLEUM BASED OIL (18 INCH); OIL (18.72 INCH)	UNKNOWN	UNCLASSIFIED
MAPPED	3-0021773	WATERTOWN YACHT CLUB (WATERBODY)	425 CHARLES RIVER RD	WATERTOWN	5/20/2002	TWO HR	UNKNOWN CHEMICAL OF TYPE - OIL; UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	RAO
MAPPED	3-0021799	HESS STATION NO 21526 (COMMERCIAL)	NORTH BEACON & ARSENAL STS	WATERTOWN	5/31/2002	72 HR	GASOLINE (3 INCH)	UST	UNCLASSIFIED
MAPPED	3-0021937	NO LOCATION AID (INDUSTRIAL)	58 IRVING ST	WATERTOWN	7/15/2002	72 HR	FUEL OIL #2 (0.05 GAL/HR); FUEL OIL #2	UST	UNCLASSIFIED
NOT MAPPED	3-0022045	NO LOCATION AID (MUNICIPAL)	ARSENAL ST	WATERTOWN	8/22/2002	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (30 GAL)	SADDLE; TANK	UNCLASSIFIED
MAPPED	3-0022172	NO LOCATION AID (RESIDNTIAL; ROADWAY)	CUSHMAN AND FAYETTE STS	WATERTOWN	10/3/2002	TWO HR	UNKNOWN CHEMICAL OF UNKNOWN TYPE (40 GAL); UNKNOWN CHEMICAL OF UNKNOWN TYPE (38 GAL)	TRANSFORM	UNCLASSIFIED

MAPPED/ NOT MAPPED	RTN	LOCATION AID	ADDRESS	TOWN	DATE	CATEGORY	MATERIALS	SOURCES	STATUS
NOT MAPPED	3-0022685	NO LOCATION AID (STATE)	SCHOOL ST E OF	WATERTOWN	3/20/2003	TWO HR	ARSENIC (190 PPM)	UNKNOWN	UNCLASSIFIED

Source: MDEP Bureau of Waste Site Cleanup. 2003. Downloadable Site Lists. http://www.state.ma.us/dep/bwsc/sites/sdown.htm (information contained in table is presented as downloaded).

Notes:
RTN - Release Tracking Number. Unique ID number assigned to releases not remediated by October 1993 and to those occuring October 1993-present
Location Aid - Place name of release
Address - Street location of release
Date - Date of release (releases prior to October 1993), or date release was reported to MDEP (for releases occurring October 1993-present)
Cateogory - Reporting category of release
Materials - Chemical(s) in release
Sources - Origin(s) of release contamination
Status - Current remediation status of release. Definitions: ADQREG Adequately Regulated; DEFT1B Default Tier 1B; DEPMOU DEP Memorandum of Understanding; DEPNDS Not a Disposal Site (DEP); DEPNFA No Further Action (DEP Determined); DPS Downgradient Property Status; DPSTRM Downgradient Property Status Terminated; INVSUB Submittal Invalidated by DEP; LSPNFA LSP No Further Action; PENNDS Pending Not a Disposal Site; PENNFA Pending No Further Action; RAO Release Action Outcome; RAONR Response Action Outcome Not Required; REMOPS Remedy Operation Status; SPECPR Special Project; STMRET Response Action Outcome Statement Retracted; TCLASS Tier Classification; TIER1A Tier 1A; TIER1B Tier 1B; TIER1C Tier 1C; TIERII Tier II; UNCLSS Unclassified; WCSPRM Waiver Completion Statement Permanent.

Appendix B

Cancer Incidence Coding Definitions

Appendix B
Coding Definitions of Cancer Site/Type*

Cancer Site / Type	ICD-O-1 and Other Pre-ICD-O-2 Codes		ICD-O-2 Codes	
	Site code	**Histology code**	**Site code**	**Histology code**
Kidney and Renal Pelvis	189.0, 189.1	except 9590–9980	C64.9, C65.9	except 9590–9989
Leukemia	140.0–199.9	includes O9800–O9943, O9951, P9803–P9943, B9803–B9943	1. C00.0–C80.9 **and** 2. C42.0, C42.1, C42.4	1. includes 9800–9822, 9824–9826, 9828–9941 2. includes 9823, 9827
Liver	155.0	except 9590–9980	C22.0	except 9590–9989
Lung and Bronchus	162.2–162.9	except 9050–9053, 9590–9980	C34.0–C34.9	except 9590–9989
Non-Hodgkin's Lymphoma (NHL)	140.0–199.9	includes O9590–O9642, O9670–O9710, O9750, P9593–P9643, P9693–P9713, P9753, B9593–B9643, B9703	1. C00.0–C80.9 **and** 2. All sites except C42.0, C42.1, C42.4	1. includes 9590–9595, 9670–9717 2. includes 9823, 9827
Pancreas	157.0–157.9	except 9590–9980	C25.0–C25.9	except 9590–9989

*Note: Includes invasive tumors only, selected by excluding in situ stages J0, S0, TTISNXM0, TTANXMX, TTANXM0, TTAN0MX, TTISN0M0, TTISNXMX, TTISN0MX, TTISN0M0, and TTIN0M0 (1982–1994 data) or by specifying behavior code (1995–1999 data).

Appendix C

Risk Factor Information for Selected Cancer Types

Kidney cancer involves a number of tumor types located in various areas of the kidney and renal system. Renal cell cancer (which affects the main area of the kidney) accounts for over 90% of all malignant kidney tumors (ACS 2006). The American Cancer Society estimates that there will be approximately 38,890 cases of kidney and upper urinary tract cancer, resulting in more than 12,840 deaths in 2006 (ACS 2006). Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in individuals between 55 and 84 years of age (ACS 2006). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

Since 1970, U.S. incidence rates for renal cell cancer have risen between 2% and 4% annually among the four major race and gender groups (i.e., white males, white females, black males, and black females) (Chow et al. 1999; McLaughlin et al. 1996). Rapid increases in incidence among blacks as compared to among whites have resulted in an excess of the disease among blacks; age-adjusted incidence rates between 1975 and 1995 for white men, white women, black men, and black women were 9.6, 4.4, 11.1, and 4.9 per 100,000 person-years, respectively (Chow et al. 1999). Rising incidence rates may be partially due to the increased availability of screening for kidney cancer.

The etiology of kidney cancer is not fully understood. However, a number of environmental, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by about 40% (ACS 2006). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed (Yuan et al. 1998).

Virtually every study that has examined body weight and renal cell cancer has observed a positive association. Some studies suggest that obesity is a factor in 20% of people who develop kidney cancer (ACS 2006). A diet high in protein (meat, animal fats, milk products, margarine and oils) has been implicated in epidemiological studies as a risk factor for renal cell carcinoma (McLaughlin et al. 1996). Consumption of adequate amounts of fruits and vegetables lowers the risk of renal cell cancer. In addition, use of diuretics and antihypertensive medications are associated with increased risk of renal cell carcinoma. However, hypertension has also been linked to kidney cancer and it is not clear whether the disease or the medications used to treat them is the cause (ACS 2000). Long-term use of pain relievers such as phenacetin (and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (McLaughlin et al. 1996).

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al. 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS 2006; McLaughlin et al. 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS 2006; Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS 2006). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution

workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al. 1997; McLaughlin et al. 1996).

Wilms' tumor is the most common type of kidney cancer affecting children and accounts for approximately 5% to 6% of all kidney cancers and about 6% of all childhood cancers. This cancer is more common among African Americans than other races and among females than males. Wilms' tumor most often occurs in children under the age of 7 years. The causes of Wilms' tumor are not known, but certain birth defect syndromes and other genetic risk factors (such as family history or genetic mutations) are connected with this cancer. However, most children who develop Wilms' tumor do not have any known birth defects or inherited gene changes. No environmental risk factors, either before or after a child's birth, have been shown to be associated with the development of Wilms' tumor (ACS 2006a).

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Leukemia is the general term that includes a group of different cancers that occur in the blood forming organs and result in the formation of abnormal amounts and types of white blood cells in the blood and bone marrow. Individuals with leukemia generally maintain abnormally high amounts of leukocytes or white blood cells in their blood. This condition results in an individual's inability to maintain certain body functions, particularly a person's ability to combat infection.

In 2006, leukemia is expected to affect approximately 35,070 individuals in the United States (20,000 males and 15,070 females) in the United States, resulting in 22,280 deaths. Acute cases of leukemia are slightly more common that chronic, 15,860 and 14,520 respectively. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML (approximately 11,700 cases) and CLL (approximately 9,560 cases). Incidences of ALL have increased approximately 1.8% per year since 1988 while incidences of CLL have decreased approximately 1.9% each year since 1988. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority (74%) of these cases are of the ALL type (ACS 2006a).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals, and 1300 (one-third) of total cases of ALL will occur in adults. ALL risk is lowest for adults aged 25 through 50 and then begins to pick up (ACS 2006b). The increase in incidence among older individuals begins at approximately 40-50 years of age, peaking at about age 85 (Linet and Cartwright 1996). ALL is more common among whites than African Americans and among males than females (Weinstein and Tarbell 1997). Exposure to high-dose radiation (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents) is a known environmental risk factor associated with the development of ALL (ACS 2006b). Significant radiation exposure (e.g., diagnostic x-rays) within the first few months of development may carry up to a 5-fold increased risk of developing ALL (ACS 2006b). However, few studies report an increased risk of leukemia associated with residing in proximity to nuclear plants or occupational exposure to low-dose radiation (Linet and Cartwright 1996; Scheinberg et al. 1997). There is conflicting evidence about whether exposure to electromagnetic fields (EMF) plays a role in the development of ALL, however, most studies to date have found little or no risk (ACS 2006b).

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2006c). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al. 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS 2006c). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection. However, no specific virus has been identified as related to ALL (Linet and Cartwright 1996). Reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care do not support this hypothesis (Greaves MF 1997; Kinlen and Balkwill 2001; Rosenbaum et al. 2000).

Although AML can occur in children (usually during the first two years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS 2006d). This type of leukemia is more common among males than among females but affects African Americans and whites at similar rates (Scheinberg et al. 1997). High-dose radiation exposure (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents), long-term occupational exposure to benzene (a chemical in gasoline and cigarette smoke), and exposure to certain chemotherapy drugs, especially alkylating agents (e.g., mechlorethamine, cyclophosphamide), have been associated with an increased risk of developing AML among both children and adults (ACS 2006d). The development of childhood AML is suspected to be related to parental exposure to pesticides and other chemicals, although findings are inconsistent (Linet and Cartwright 1996). Studies have suggested a link between electromagnetic field (EMF) exposure (e.g., from power lines) and leukemia (Minder and Pfluger 2001; Schuz et al. 2001). However, there is conflicting evidence regarding EMF exposure and leukemia and it is clear that most cases are not related to EMF (Kleinerman et al. 2000).

Other possible risk factors related to the development of AML include cigarette smoking and genetic disorders. It is estimated that approximately one-fifth of cases of AML are caused by smoking (Scheinberg et al. 1997). Also, a small number of AML cases can be attributed to rare inherited disorders, such as Down's syndrome (ACS 2006d). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al. 2001).

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 2006e). Twice as many men as women are affected by this type of leukemia (Deisseroth et al. 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS 1999; Weinstein and Tarbell 1997). It is thought that individuals with a family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright 1996). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al. 1997).

Of all the leukemias, CML is among the least understood. While this disease can occur at any age, CML is extremely rare in children (about 2% of leukemias in children) and the average age of diagnosis is 40 to 50 years (ACS 2006f). Incidence rates are higher in males than in females, but unlike the other leukemia types, rates are higher in blacks than in whites in the U.S. (Linet and Cartwright 1996). High-dose radiation exposure may increase the risk of developing CML (ACS 2006f). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell 1997).

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

In some developing countries, HCC is most common type of cancer diagnosed particularly in East Asia and Africa. Incidence in the United States had been increasing up to 1999. Recently, the rate has become more stable (ACS 2006a). Rates of HCC in the U.S. had increased by 70% during the 1980s and 1990s (Yu et al. 2000). Similar trends were observed in Canada and Western Europe. The primary reason for the higher rates observed during those years was the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag 2001; El-Serag and Mason 2000).

Men are at least three times more likely to develop HCC than women. Much of this is likely due to differences in lifestyle factors which increase a person's risk for developing liver cancer (ACS 2006a). Although 85% of individuals diagnosed with liver cancer are between 45 and 85 years of age, the disease can occur in persons of any age (ACS 2006a).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS 2006a). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al. 2000). In the United States, HBV accounts for less than a quarter of the cases and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS 2006a).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that is the result of scar tissue formation on the liver, which can lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al. 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS 2006a). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS 2006a). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al. 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan 2000). Vinyl chloride, a known human

carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2006a; London and McGlynn 1996). These chemicals may also increase the risk of cholangiocarcinoma and HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS 2006a). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS 2006a; ATSDR 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS 2006a; London and McGlynn 1996). Long-term anabolic steroid use may slightly increase the risk of HCC (ACS 2006a). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al. 2000; London and McGlynn 1996).

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Lung cancer generally arises in the epithelial tissue of the lung. Several different histologic or cell types of lung cancer have been observed. The various types of lung cancer occur in different regions of the lung and each type is associated with slightly different risk factors (Blot and Fraumeni 1996). The most common type of lung cancer in the United States today is adenocarcinoma which accounts for about 40% of all lung cancers (ACS 2005). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Lung cancer is very rare in people under the age of 40. The incidence is greater among men than women (probably because men are more likely to be smokers than women) and among blacks than whites (Blot and Fraumeni 1996). The American Cancer Society estimates that lung and bronchus cancer will be diagnosed in 174,470 people (92,700 cases in men and 81,770 in women) in the U.S. in 2006, accounting for about 12% of all new cancer diagnoses. For purposes of treatment, lung cancer is divided into two clinical groups: small cell lung cancer (13%) and non-small cell lung cancer (87%) (ACS 2006). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS 2005). In Massachusetts, an estimated 4,070 individuals will be diagnosed with lung and bronchus cancer in 2006. Incidence rates for lung and bronchus cancer in Massachusetts from 1998 through 2002 were 86.5 per 100,000 and 60.4 per 100,000 for males and females, respectively (ACS 2006). Nationwide, the incidence rate declined significantly in men during the 1990s, most likely as a result of decreased smoking rates over the past 30 years. Rates for women are approaching a plateau, after a long period of increase. This is likely because decreasing smoking patterns among women have lagged behind those of men (ACS 2006). Trends in lung cancer incidence suggest that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals have higher rates of smoking than individuals of other groups (Blot and Fraumeni 1996).

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

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS 2005). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (Samet and Eradze 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni 1996; Pohlabeln et al. 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS 2005; Blot and Fraumeni 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al. 2001; Steenland and Sanderson 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni 1996).

As noted above, exposure to radon (a naturally occurring radioactive gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer among miners. Recently, a number of studies have demonstrated that exposure to elevated levels of residential radon may also increase lung cancer risk (Lubin and Boice 1997; Kreienbrock et al. 2001; Tomasek et al. 2001). Epidemiological evidence suggests that radon may be the second leading cause of lung cancer after smoking (Samet and Eradze 2000). However, actual lung cancer risk is determined by cumulative lifetime exposure to indoor radon. Therefore, normal patterns of residential mobility suggest that most people living in high-radon homes experience lifetime exposures equivalent to residing in homes with lower radon levels (Warner et al. 1996).

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

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

Diet has also been implicated in the etiology of lung cancer, however, the exact relationship is unclear. Diets high in fruits and vegetables decrease lung cancer risk, but the reasons for this are unknown (Brownson et al. 1998). A study showed a positive association between total fat, monounsaturated fat, and saturated fat and lung cancer among males, however, this effect was not observed among women (Bandera et al. 1997).

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Lymphomas are cancers involving the cells of the lymphatic system. The majority of lymphomas involve the lymph nodes and spleen but the disease may also affect other areas within the body. Non-Hodgkin's lymphoma (NHL) is a classification of all lymphomas except Hodgkin's disease. Thus NHL is a mixed group of diseases that is characterized by the malignant increase in specific cells of the immune system (B or T lymphocytes). B-cell lymphomas are more common than T-cell lymphomas, accounting for about 85% of all cases of NHL (ACS 2006a). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller 1996). NHL can occur at any age. However, the average age at diagnosis is in the early 60s and the incidence of this disease generally increases with age. This disease is more common in men than in women and affects whites more often than African Americans or Asian Americans (ACS 2006a). The American Cancer Society estimates that approximately 58,870 Americans will be diagnosed with NHL in 2006 with 30,680 diagnoses occurring among males and 28,190 diagnoses occurring among females (ACS 2006b).

Overall, between 1973 and 1997, the incidence of NHL in the U.S. grew 81% (Garber 2001), although over the past 20 years, the incidence rate appears to have stabilized (ACS 2006b). In Massachusetts, the incidence of NHL increased 50% during 1982-1997 from 10.5 cases per 100,000 to 15.7 cases per 100,000 (MCR 1997, 2000). The increase in NHL incidence has been attributed to better diagnosis, greater exposure to causative agents, and, to a lesser extent, the increasing incidence of AIDS-related lymphomas (Devesa and Fears 1992; Scherr and Mueller 1996). Although the primary factors related to the development of NHL include conditions that suppress the immune system, viral infections, and certain occupational exposures, these factors are thought to account for only a portion of the increase observed in this cancer type (Scherr and Mueller 1996).

NHL is more common among people who have abnormal or compromised immune systems, such as those with inherited diseases that suppress the immune system, individuals with autoimmune disorders, and people taking immunosuppressant drugs following organ transplants. Genetic predisposition (e.g., inherited immune deficiencies) only accounts for a small proportion of NHL cases (Scherr and Mueller 1996). AIDS patients have a 100- to 300-fold higher risk for NHL than the general population (again, these cases account for only a minor part of overall NHL incidence) (Garber 2001). NHL has also been reported to occur more frequently among individuals with conditions that require medical treatment resulting in suppression of the immune system, such as cancer chemotherapy. However, current evidence suggests that the development of NHL is related to suppression of the individual's immune system as a result of treatment, rather than the treatment itself (Scherr and Mueller 1996).

Several viruses have been shown to play a role in the development of NHL. Among organ transplant recipients, suppression of the immune system required for acceptance of the transplant leads to a loss of control or the reactivation of viruses that have been dormant in the body [e.g., Epstein - Barr virus (EBV) and herpes virus infections]. In addition, because cancer-causing viruses are known to cause lymphomas in various animals, it has been proposed that these types of viruses may also be associated with the development of NHL among humans without compromised immune systems. Infection with the human T-cell leukemia/lymphoma virus (HTLV-I) is known to cause T-cell lymphoma among adults. However, this is a relatively rare infection and most likely contributes only a small amount to the total incidence of NHL (Scherr and Mueller 1996). EBV infection is common among the general population and has been shown to play a role in the development of most cases of transplant and AIDS related NHL. Although viruses are causal factors for some subtypes of NHL, to date, studies have shown that the role of EBV in the development of NHL in the general population may not be large (Scherr and Mueller 1996). Moreover, the high prevalence of EBV in the general population suggests that EBV may be only one of several factors in the development of this cancer.

Recent studies have found that a type of bacteria, Helicobacter pylori, a common cause of stomach ulcers, can also cause some lymphomas of the stomach (ACS 2006). An important implication of this finding is that treatment with antibiotics could prevent some NHL of the stomach.

Some occupations have been associated with an increased risk of developing NHL, such as occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm 1990, 1993; Tatham et al. 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and organophosphate insecticides (Wigle et al. 1990; Zahm et al. 1990; Zahm et al. 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD alone have not demonstrated an increased risk of NHL (Scherr and Mueller 1996). An elevated risk for NHL development has also been noted among fence workers, orchard workers, and meat workers. High-dose exposure to benzene has been associated with NHL (ACS 2006a). However, a recent international cohort study indicated that petroleum workers exposed to benzene were not at an increased risk of NHL (Wong and Raabe 2000).

In addition, epidemiological studies of long-term users of permanent hair coloring products have suggested an increased incidence of NHL (Zahm et al. 1992; Scherr and Mueller 1996). However, a population based study found no association between the use of hair color products and an increased risk of developing NHL. The researchers further stated that results from this study and previous studies, including experimental animal studies, provide little convincing evidence linking NHL with normal use of hair dye (Holly et al. 1998).

Although radiation (e.g., nuclear explosions or radioactive fallout from reactor accidents) has been implicated in the development of some cancers, including NHL (ACS 2006a), there is little evidence for an increased risk of lymphoma due to radiation (Scherr and Mueller 1996).

Studies have suggested that contamination of drinking water with nitrate may be associated with an increased risk of NHL (Ward et al. 1996). Nitrate forms N-nitroso compounds which are known carcinogens and can be found in smoked or salt-dried fish, bacon, sausages, other cured meats, beer, pickled vegetables, and mushrooms.

Smoking has also been suggested to increase the risk of NHL. A study that evaluated the history of tobacco use and deaths from NHL determined that people who had ever smoked had a two-fold increase of dying from NHL as compared to those who never smoked. Further, a four-fold increase was found among the heaviest smokers (Linet et al. 1992). In addition, a more recent study that primarily examined occupation and NHL risk found a significant association with high levels of cigarette smoking and all NHL types (Tatham et al. 1997). However, a review of five cohort studies and 14 case-control studies concludes that results of epidemiological studies have been inconsistent and that smoking has not been determined to be a definitive risk factor in the development of NHL (Peach and Barnett 2000).

A Danish study has linked the use of tricyclic and tetracyclic antidepressants to NHL. However, more research is needed on this possible association (Dalton et al. 2000).

Although NHL is associated with a number of risk factors, the causes of this disease remain unknown. Most patients with NHL do not have any known risk factors (ACS 2006a).

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The American Cancer Society estimates that approximately 33,730 people in the U.S. (17,150 men and 16,580 women) will develop pancreatic cancer in 2006. This disease accounts for approximately 2% of all new cases of cancer in both men and women, but between 5% and 6% of all cancer deaths (ACS 2006a). This discrepancy has been attributed to detection of pancreatic cancer at an advanced stage and the short median survival time for this cancer of approximately three months. Between 1920 and 1965, mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people. These increases are believed to be due, in part, to improved diagnosis during this time period (Anderson et al. 1996). However, over the past 25 years, incidence rates have declined slowly but consistently in men and a slight decline in rates among women has been observed since the mid-1980s. Further, since the 1970s, men have experienced a slight decrease in mortality from pancreatic cancer, although rates among women have not dropped (ACS 2006a). The risk of developing pancreatic cancer increases with age and the majority of cases occur between age 60 and 80. Men are approximately 20% more likely to develop pancreatic cancer than are women (ACS 2006b).

Very little is known about what causes pancreatic cancer and how to prevent it. However, a number of risk factors have been identified. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2006b). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al. 1996).

Certain medical conditions, such as chronic pancreatitis, diabetes mellitus, and cirrhosis, have been associated with pancreatic cancer, but the reasons for these associations are largely unknown. More recently, a possible role for the bacteria Helicobacter pylori, which causes ulcers and some gastric cancers, has been suggested in the development of pancreatic cancer (ACS 2006b; Stolzenberg-Solomon et al. 2001). Some researchers also believe that excess stomach acid may increase the risk of pancreatic cancer (ACS 2006b).

There is also some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer may be associated with animal protein and fat consumption as evidenced by higher rates of this cancer in countries whose populations eat a diet high in fat (ACS 2006a). Decreased risks for the disease are usually associated with fruit and vegetable consumption (ACS 2006). Obesity is also a risk factor for pancreatic cancer, and very overweight people are 20% more likely to develop pancreatic cancer (ACS 2006b). Although older studies suggested that coffee and alcohol consumption may be risk factors, more recent studies do not support this association (Michaud et al. 2001).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS 2006b; Ji et al. 2001; Porta et al. 1999). Exposure to certain dyes and chemicals related to gasoline, in addition to asbestos and ionizing radiation, has also been associated with the development of pancreatic cancer in some studies. However, other studies have found no link between these agents and pancreatic cancer (ACS 2006b; Anderson et al. 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this claim (Ojajarvi et al. 2000). Although occupational exposures may have played a role in the incidence of this cancer in the past, currently most newly diagnosed patients with pancreatic cancer do not have evidence of a specific chemical exposure or relevant occupational history (Evans et al. 1997).

Finally, pancreatic cancer seems to run in some families. According to the American Cancer Society, an inherited tendency to develop pancreatic cancer may account for as many as 10% of cases. Also, inherited DNA mutations that increase risk of developing pancreatic cancer can also increase the risk of developing other cancers. For example, some people with inherited BRCA2 mutations (which increases risk of breast cancer), an inherited tendency for melanoma (skin cancer), or an inherited tendency for colorectal cancer are also at an increased risk of developing pancreatic cancer (ACS 2006b). Pancreatic cancer has been observed in both familial clusterings among siblings as well as in individuals of consecutive generations (Anderson et al. 1996).

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Appendix D

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

General Terms

Absorption
The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Anaerobic
Requiring the absence of oxygen [compare with aerobic].

Analyte

A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP [see Community Assistance Panel.]

Cancer
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

CERCLA [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure]

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)

A group of people from a community and from health and environmental agencies who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Delayed health effect
A disease or an injury that happens as a result of exposures that might have occurred in the past.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see route of exposure].

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit

The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease prevention
Measures used to prevent a disease or reduce its severity.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

DOD
United States Department of Defense.

DOE
United States Department of Energy.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction
A method of estimating the amount of people's past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Half-life (t½)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remain.

Hazard
A source of potential harm from past, current, or future exposures.

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to evaluate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.

Health statistics review

The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

mg/cm2
Milligram per square centimeter (of a surface).

mg/m3
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, a condition, or an injury) is stated.

Mutagen
A substance that causes mutations (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard

A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see National Priorities List for Uncontrolled Hazardous Waste Sites]

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevalence survey
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public availability session
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are no public health hazard,

no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Public health statement
The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public health surveillance
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [see Resource Conservation and Recovery Act (1976, 1984)]

Receptor population
People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)
This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA

RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or an environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Stakeholder

A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Surveillance [see public health surveillance]

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen

A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)

National Center for Environmental Health (CDC)
(http://www.cdc.gov/nceh/dls/report/glossary.htm)

National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

For more information on the work of ATSDR, please contact:

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Appendix E

Response to Public Comments

Appendix E

Response to Public Comments on "Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999"

Listed below are comments received from the public regarding the Public Health Assessment (PHA) for Cambridge Plating Company in Belmont, Massachusetts. The public comment period ended on December 15, 2005. MDPH and ATSDR received written comments from two residents of Belmont. In addition, a number of comments were received from individuals who attended the October 20, 2005 public meeting. All comments received are summarized with responses provided below. Where possible, these comments were listed together and a single response has been provided.

Comment 1: *Due to limited surface soil sampling data available for Cambridge Plating, it is unknown whether past emissions of chromium to air could have resulted in elevated levels of chromium in surface soils in other areas surrounding the facility. Without this data, it was not possible for you to evaluate this potential surface soil exposure pathway. Please define the specific sample data MA DEP. The sample needs to include depth, location, and number of samples. Also, would soil samples from residents north of the facility be useful?*

Comment 2: *Purecoat has caused the release of chromium in to the air from vents on or near the roof of the facility. Chromium has been determined to be a human carcinogen. In the air, chromium compounds are present mostly as fine dust particles, which eventually settle over land and water. There is the potential that chromium could be on nearby properties, even if the Purecoat property appears to be clean. There is a known chromium contaminated site at the north side of the facility that is not fenced in. Children who have unique vulnerabilities to toxic emissions have been observed on the site. It is not yet known if this area has been fully cleaned up. We still do not know how much contamination is in the ground, and how it got there. This is why additional studies should be done.*

Comment 3: *Airborne chromium may have been dispersed and contamination may not necessarily be near the plant. Off-site sampling for chromium should be done to evaluate dispersal of airborne emissions from the roof.*

Comment 4: *Was the chromium found on site and near the railroad tracks hexavalent chromium?*

Response:

In November 2005, Cambridge Plating submitted additional soil sampling results for both total and hexavalent chromium (chromium VI) in response to a requirement by the Massachusetts Department of Environmental Protection (MDEP) (OHI 2005a). Ten on-site locations were sampled to meet the MDEP requirement including three locations along Hittinger Street, three along the MBTA railroad tracks, two along the fence adjacent to the baseball field, and two along Brighton Street. These locations were required by MDEP based on air emissions modeling results indicating that areas of maximum impact would be located on the site; this modeling was done in order to determine whether off-site sampling would be necessary (J. Miano, MDEP, personal communication, 2006). All OHI samples were collected from surface soils at depths between 0 and 6 inches and analyzed for both total and hexavalent forms of chromium. Total chromium was detected in each of the ten samples at concentrations ranging from 23 to 220 milligrams per kilogram (mg/kg) (OHI 2005a). The maximum detected concentration of total chromium was below the Reference Dose Media Evaluation Guide (RMEG) value for adult exposure to chromium VI (2,000 mg/kg) and similar to the RMEG value for childhood exposure to chromium VI (200 mg/kg). RMEGs are developed by the Agency for Toxic Substances and Disease Registries (ATSDR) and represent the concentration in soil at which daily human exposure is unlikely to result in adverse noncarcinogenic effects. ATSDR comparison values for total chromium were not available, so comparison values for chromium VI were used. Chromium VI was detected in one of the ten samples at 1.2 mg/kg (OHI 2005a), well below both the adult and child RMEG values for chromium VI.

To evaluate community concerns about potential exposure to chromium that might be present in off-site surface soils, exposure estimates were calculated for incidental soil ingestion using the maximum total chromium (220 mg/kg) and maximum chromium VI (1.2 mg/kg) concentrations detected in on-site surface soils samples collected at Cambridge Plating at depths of 0 to 6 inches. As previously mentioned, the highest concentrations in soil would be on the site itself. Therefore, the use of the highest measured concentration in on-site soil represents a conservative assumption for evaluating off-site health risks.

Assuming an older child trespassed on the site, incidentally ingested 200 mg/kg of soil per day, 1 day per week for 26 weeks of the year for 5 years, the exposure doses would be below the United States Environmental Protection Agency (EPA) Chronic Reference Doses for both chromium III and chromium VI [4, 5]. The EPA reference dose is defined as

[4] ATSDR Minimal Risk Levels and EPA cancer slope factors were not available for total chromium

an estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans. Therefore, the chromium present in the soil is unlikely to cause adverse health effects in nearby residents.

Because true on-site surface soil samples were previously unavailable and samples collected from 0 to 4 feet and presented in the 2005 PHA report included soil samples with higher concentrations of total chromium and chromium VI, the same exposure assumptions listed above were also used to evaluate the previous chromium concentrations measured in soil. Specifically, using the maximum concentrations of total (3,900 mg/kg) and hexavalent chromium (4.6 mg/kg) detected in all on-site surface soil samples collected at depths of 0 to 4 feet below ground surface, the estimated exposures are also below the EPA Chronic Reference Doses for both chromium III and chromium VI.[6] In addition, other considerations suggest that these exposure estimates would be even lower. First, it is unlikely that the trespasser would be exposed to the maximum concentration of chromium 1 day each week for 26 weeks of each year for 5 years. Second, soil containing the maximum concentration of total chromium (3,900 mg/kg) was excavated and removed from the site in 2004 (OHI 2005b). Finally, more recent soil samples taken around the perimeter of the property confirm that levels of chromium in

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[5] *Calculation using the maximum total chromium value in surface soil:*

$$\text{Noncancer Effects Exposure Factor} = \frac{(1\,\text{day/week})\,(26\,\text{weeks/year})\,(5\,\text{years})}{(5\,\text{years})\,(365\,\text{days/year})} = 0.07$$

$$\text{Noncancer Effects Exposure Dose(Older Child)} = \frac{(\text{max contaminant concentration})\,(\text{ingestion rate})\,(\text{exposure factor})\,(1\text{kg}/10^6\,\text{mg})}{\text{body weight}}$$

$$= \frac{(220\,\text{mg/kg})\,(200\,\text{mg/day})\,(0.07)\,(1\text{kg}/10^6\,\text{mg})}{35\,\text{kg}} = 8.8x10^{-5}\,mg\,/\,kg\,/\,day$$

$$\text{EPA Chronic Rfd for Chromium VI} = 0.003 mg\,/\,kg\,/\,day$$

$$\text{EPA Chronic Rfd for Chromium III} = 1.5 mg\,/\,kg\,/\,day$$

[6] *Calculation using the maximum hexavalent chromium value in surface soil:*

$$\text{Noncancer Effects Exposure Dose(Older Child)} = \frac{(\text{max contaminant concentration})\,(\text{ingestion rate})\,(\text{exposure factor})\,(1\text{kg}/10^6\,\text{mg})}{\text{body weight}}$$

$$= \frac{(1.2\,\text{mg/kg})\,(200\,\text{mg/day})\,(0.07)\,(1\text{kg}/10^6\,\text{mg})}{35\,\text{kg}} = 4.8x10^{-7}\,mg\,/\,kg\,/\,day$$

$$\text{EPA Chronic Rfd for Chromium VI} = 0.003 mg\,/\,kg\,/\,day$$

$$\text{EPA Chronic Rfd for Chromium III} = 1.5 mg\,/\,kg\,/\,day$$

surface soil, soil that residents may come in contact with, are unlikely to cause adverse health effects for nearby residents.

Based on air emissions modeling results, MDEP predicted that the areas with the highest concentrations of chromium would likely be located on-site. Therefore, given that adverse health effects are unlikely for trespassers exposed to on-site surface soil, based on both reasonable and conservative exposure estimates, it appears unlikely that exposure through incidental ingestion to chromium potentially present in off-site surface soil would result in adverse health effects for residents living in close proximity to Cambridge Plating.

In June 2007, in response to the MDPH recommendation in the 2005 public comment release of the Cambridge Plating Public Health Assessment, the MDEP sampled surface soil at off-site locations near Cambridge Plating to confirm the results of air emissions modeling and to determine if elevated levels of chromium were present in off-site soil (MDEP 2007). The seven samples were located near the tennis courts and ball field east of the facility, near residences north of the facility, near the intersection of Hittinger and Baker Streets south of the facility, and at three locations along Brighton Street northwest, west, and southwest of the facility. Hexavalent chromium was not detected in any of the seven surface soil samples (Figure 4). Total chromium measurements ranged from 12–46 mg/kg (Table 1). These concentrations of chromium were lower than ATSDR comparison values of 200 mg/kg for hexavalent chromium and are not likely to pose a health threat to nearby residents. These concentrations are also below levels of chromium considered typical for soil in the eastern United States (Shacklette and Boerngen 1984).

Table 1: Off-Site Surface Soil Sampling Results (June 2007)

Sample Name	Hexavalent Chromium (mg/kg)	Total Chromium (mg/kg)
1-Ball Field	Not Detected	26
2-Tennis Courts	Not Detected	44
3-Residences - North	Not Detected	20
4-Brighton St - Vale Rd	Not Detected	19
5-Condos - End of Vale	Not Detected	12
6-Brighton St - Flanders Rd	Not Detected	46
7-Hittinger St - Baker St - South	Not Detected	14

This additional information was incorporated as part of the final Cambridge Plating Public Health Assessment report (see sections IV and V).

Comment 5: *Some neighbors have vegetable gardens – could this be another exposure pathway?*

Response:
Studies of chromium uptake into plants have shown that only a small fraction of chromium present in soils typically reaches the above ground portion of edible plants

(ATSDR 2000). However, to evaluate community concerns about potential exposure to chromium through ingestion of fruits and vegetables grown in nearby gardens, exposure estimates were calculated using the maximum total chromium value measured during recent off-site surface soil sampling discussed in the response above (exposure estimates shown below under Calculations). Sampling conducted during June 2007 at several off-site locations indicated no detectable levels of hexavalent chromium and a maximum total chromium concentration of 46 mg/kg.

Conservative estimates for consumption of fruits and vegetables by households in the northeast region of the United States were used to calculate exposure estimates (EPA 1997). The EPA suggests the use of a bioconcentration factor to represent the amount of chromium in soil that is actually taken up by the roots of the plant and the use of a dry weight to wet weight conversion factor to determine the concentration of chromium in a typical garden plant (EPA 1999). Calculations can be seen below and show that the human exposure dose of chromium from fruits and vegetables is well below levels thought by the EPA to cause harm in humans. This indicates that regular ingestion of fruits and vegetables grown in the vicinity of Cambridge Plating would be unlikely to result in adverse health effects for residents.

Equation for uptake of chromium from soil to plants

Hexavalent Chromium in Plants = C x BCF x CF

where:

C = Contaminant Concentration in soil (mg/kg)
BCF = Bioconcentration Factor (unitless)
CF = Dry weight to wet weight conversion factor (unitless)

Equation for exposure dose from ingestion of homegrown fruits or vegetables

D = CL x CR x EF x PH / BW

where:

D = Exposure Dose (mg/kg/day)
CL = Contaminant Concentration in plant (mg/g)
CR = Consumption Rate of Food Group (g/day)
EF = Exposure Factor (unitless)
PH = Percentage of food that is homegrown
BW = Body weight (kg)

Calculations

Hexavalent Chromium in Plants = C x BCF x CF
= 46 mg/kg x 0.0075 x 0.12
= 0.0414 mg/kg or 0.0000414 mg/g

Calculation for ingestion of homegrown fruits:

$$\text{Exposure Dose(Adult)} = \frac{(\text{Contaminant concentration})\,(\text{Consumption rate})\,(\text{Exposure Factor})\,(\%\ \text{of food that is homegrown})}{\text{body weight}}$$

$$= \frac{(0.0000414\ \text{mg/g})\,(129.6\ \text{g/day})\,(1)\,(0.5\%)}{70\ \text{kg}} = 0.0000004 mg/kg-day$$

Calculation for ingestion of homegrown vegetables:

$$\text{Exposure Dose(Adult)} = \frac{(\text{Contaminant concentration})\,(\text{Consumption rate})\,(\text{Exposure Factor})\,(\%\ \text{of food that is homegrown})}{\text{body weight}}$$

$$= \frac{(0.0000414\ \text{mg/g})\,(469.2\ \text{g/day})\,(1)\,(3.8\%)}{70\ \text{kg}} = 0.00001 mg/kg-day$$

The total human exposure dose of chromium from contaminated garden produce is equal to the sum of the exposure dose from homegrown fruit and the exposure dose from homegrown vegetables. Therefore, the total human exposure dose from chromium in garden produce is 0.00001 mg/kg-day based on levels measured in recent off-site soil samples. The EPA Chronic Reference Dose (Rfd) is 0.003 mg/kg-day. Because the total exposure dose of chromium present in garden fruits and vegetables is well below the EPA Rfd, it is unlikely to cause adverse health effects in residents.

Comment 6: *People spend a lot of time at the softball field and at Clay Pit Pond. Is it safe to be playing softball or walking around the pond? Has soil been tested at the ball field and near the pond?*

Response:
As discussed in the responses above, two of the ten soil samples taken in November 2005 were located along the fence adjacent to the baseball field. Surface soil samples CR-1 and CR-3 were located along the fence that borders the right outfield of the field adjacent to Cambridge Plating. These soil samples had no detectable levels of hexavalent chromium and had concentrations of total chromium of 51 mg/kg and 24 mg/kg, respectively. A surface soil sample taken at the ball field in June 2007 also had no detectable level of hexavalent chromium and had a low total chromium concentration of 26 mg/kg. These concentrations are all below concentrations of chromium considered typical for soil in the eastern United States (Shacklette and Boerngen 1984).

Incidental ingestion and dermal contact with soils near Clay Pit Pond could be possible for children or adults who may walk or play around the pond. However, MDEP modeling based on air emissions indicated higher concentrations on-site and lower concentrations at points off-site, such as at Clay Pit Pond. The off-site soil sampling confirmed the modeling and indicated that soil samples from points nearest to Clay Pit Pond, such as samples taken near the tennis courts and from the ball field, show levels of chromium are below comparison values. Contaminant levels below these comparison

values are not likely to pose a health threat to residents (see further discussion in Section IV of the PHA).

A majority of the contaminants measured on-site were below comparison values or were within the range of concentrations considered typical for soil of this area and are not likely to pose a health threat. A small number of contaminants were measured above comparison values. However, in addition to the likelihood that site-related contaminant concentrations are lower at Clay Pit Pond than in on-site soil, it is important to consider that comparison values are based on a residential exposure scenario, and it is unlikely that a visitor to Clay Pit Pond would have had contact with surface soil for a comparable frequency and duration of time.

Comment 7: *What about children who have been seen playing in puddles that could be contaminated by rooftop drainage and/or sump pump discharge?*

Response:
It is possible that individuals who trespassed on the property could have or have had infrequent contact with rooftop drainage or with sump pump discharge. Because data are not available for rooftop water or sump pump discharge, it is not possible to quantitatively evaluate this scenario. Reports in the Belmont Health Department files indicate that young adults have trespassed on the Cambridge Plating property in the past; specifically, there was an incidence of youth climbing on the roof of the building (Belmont Department of Health 2003a, 2003b). However, because this is a working industrial facility it is not expected that these individuals would be trespassing on a regular basis or for an extended period of time. Therefore, it is unlikely that children who might trespass on the site would have contact with rooftop drainage or sump pump discharge for a sufficient frequency and duration of time to result in health effects.

Comment 8: *On page 10, paragraph 3, it refers to a chromating process still in operation. Is this hexavalent chromium? What is the process and what fugitive emissions would be expected?*

Response:
According to MDEP, there is still a chromating process in operation at Cambridge Plating. This process is operated at ambient conditions and uses hexavalent chromium (C. Buttaro, MDEP, personal communication, 2007). However, as stated in the 2005 public comment release of the public health assessment titled "*Evaluation of Environmental Concerns and Cancer Incidence in Belmont and Surrounding Communities, Middlesex County, Massachusetts, 1982–1999,*" the chromating process is not heated and air emissions are not expected (R. Crystal, U.S. EPA Region 1, personal communication, 2005).

Comment 9: *Purecoat has caused the release of TCE to the soil and groundwater on its property. TCE has been identified as a probable human carcinogen. TCE breaks down into more hazardous compounds such as vinyl chloride. Groundwater does leave the Purecoat facility. All the testing to date has only been conducted on the Purecoat property. No testing has occurred in the neighborhoods adjacent to the facility, the ball field, or the tennis courts. At some superfund sites, TCE has been found to volatilize from*

the groundwater and soils and accumulate in the basements of properties neighboring the site.

Comment 10: *Preventing further exposure should be very important, as we do not yet know what kind of contaminants are in the ground, if it has spread off-site, or if vapor intrusion to indoor air of nearby homes has occurred. Further assessment should be a priority, to ensure the health and safety of our community.*

Response:
To evaluate whether the levels of TCE detected in groundwater at the site could contaminate indoor air in nearby residences, MDPH asked ATSDR to run a model incorporating site-specific information on groundwater, soil, and housing for the area surrounding the facility (ATSDR 2003). On the basis of the Johnson-Ettinger modeling results provided by ATSDR and soil gas and groundwater investigations at Cambridge Plating required by MDEP, high levels of VOCs, including TCE, detected in groundwater near the building (monitoring wells MW-03 and CC-105) do not appear to have migrated south toward the site boundary at sufficient concentrations to raise health concerns related to VOCs in indoor air in nearby homes along Hittinger Street.

In November 2005, Cambridge Plating submitted additional groundwater characterization and groundwater sampling results in response to a requirement by the MDEP (OHI 2005a). This additional investigation was performed in response to an MDPH recommendation to conduct "additional characterization of VOCs in groundwater in the northern portion of the site…to ensure elevated levels of TCE detected in monitoring wells located close to the Cambridge Plating building are not presenting a health concern for indoor air in homes north of the site (MDPH 2005)." Three on-site deep monitoring wells were installed and sampled to meet the MDEP requirement. Three existing groundwater monitoring wells on the property were also sampled in 2005. In addition to groundwater sampling locations around the property, a groundwater sample was taken from beneath the facility to meet MDEP's requirement for the investigation of TCE sources in the vicinity of the degreasing area or boiler room. Of the seven new groundwater samples taken on-site, three sampled deep groundwater (30-35 feet below ground surface) and four sampled shallower groundwater.

Review of all available shallow groundwater data for TCE and its breakdown products indicates that TCE concentrations measured in the area of the loading dock (the location previously determined to contain the highest levels of VOCs on-site) are lower than levels measured in 2004, concentrations of cis-1,2-dichloroethene are similar to levels measured in 2004, and concentrations of vinyl chloride are greater than levels measured in 2004 (OHI 2005a).

The new information also clarified groundwater conditions and allowed the MDEP to better determine potential health risks to residents living in the vicinity of Cambridge Plating, including those living in homes north of the facility.

MDEP determined that the new data confirmed that shallow groundwater flow is generally in a south-southwest direction across the site. According to the MDEP, it is unlikely that VOCs found at high concentrations near the loading dock would flow upgradient (i.e., against the flow of groundwater) and be present at concentrations that could present a health hazard to residents in homes north of the site.

Additionally, in an *Interim Deadline* letter dated May 20, 2005, MDEP required that Cambridge Plating Company obtain a groundwater sample from beneath the floor of the facility in the TCE degreasing area or boiler room to investigate the source of elevated levels of VOCs in the area of the loading dock on the southern side of the building. The groundwater sample was taken 10 feet below the floor of the degreasing area by OHI Engineering, Inc. The groundwater sample showed low levels of TCE (15 ug/L) and other VOCs. Therefore, OHI Engineering, Inc. concluded and MDEP concurred that there did not appear to be an ongoing source of VOC contamination beneath the building.

This additional information was incorporated as part of the revised PHA report (see Section V).

Comment 11: *The purpose of the public health action plan is to ensure that this health assessment is not only a definite potential public health hazard, but to provide a plan of action. Who will implement your recommendations?*

Response:
As suggested in the first three recommendations in the 2005 PHA report, MDEP required additional characterization of groundwater and of groundwater flow patterns, and additional surface soil sampling at the Cambridge Plating facility. In November 2005, Cambridge Plating Company submitted results for all of the MDEP requirements. The additional environmental data were reviewed by MDPH and are summarized in Response to Comments 1-4, 6, 9, and 10. The fourth recommendation related to the review of additional environmental data on historical air emissions or ambient air quality related to Cambridge Plating, if available. No additional historical data have become available. The final recommendation suggested a collaboration of efforts to reduce odor and nuisance concerns. Correspondence with the Belmont Department of Health indicates that "odors from the company have subsided and the complaints greatly decreased (Belmont Department of Health 2007)."

Comment 12: *Purecoat has potassium cyanide at the facility. This material can be used to make hydrogen cyanide by merely mixing it with acid. Hydrogen cyanide is what is used in gas chambers, and also listed as a chemical warfare agent. They also store acid. If there were another fire, or accident at the facility, these materials mixed together could cause a cloud of deadly gas. Purecoat should be required to do a consequence analysis of such a release since we have a company, which has release potentially cancer causing substances, and store materials, which could lead to release of a chemical warfare agent, right in the middle of a residential neighborhood, next to a public school, and little league field.*

Response:
The Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 was passed in response to concerns regarding the environmental and safety hazards posed by the storage and handling of toxic chemicals. EPCRA requires that those facilities storing hazardous chemicals, such as potassium cyanide, submit an annual inventory report for those chemicals to the State Emergency Response Commission (SERC), the Local Emergency Planning Committee (LEPC) and local fire department by March 1 of each year. The LEPC must then develop an annually reviewed emergency response plan and provide information about chemicals in the community to residents. In Belmont, the LEPC By-Laws state that members of this committee shall include representatives from the following categories: fire, police, health, transportation, public works, school departments, Hazmat Team, Belmont Emergency Management Agency, hospitals, Emergency Medical Services (EMS), covered facilities (such as Cambridge Plating), media, and community groups. Residents seeking further information on the emergency response plan in the Town of Belmont should contact the LEPC at 617-993-2203 or see http://www.town.belmont.ma.us/Public_Documents/BelmontMA_EMS/LEPC.

Comment 13: *What did childhood cancer look like near Cambridge Plating?*

Response:
Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–2003 were obtained for Belmont census tracts (CTs) 3571 and 3572 from the Massachusetts Cancer Registry (MCR). Cambridge Plating is located in CT 3572 close to the border of CT 3571. During the 22-year time period from 1982–2003, there were four diagnoses of cancer among children under 19 years of age living in Belmont census tracts (CT) 3571 and 3572. As discussed below, no apparent geographic or temporal concentrations of cancer diagnoses were observed in any one area of Belmont CTs 3571 and 3572. Standardized incidence ratios (SIRs) and 95% confidence intervals were not calculated for childhood cancer types due to small numbers of observed diagnoses.

Address at the time of diagnosis for each child diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2005). This allowed for an evaluation of the spatial distribution of individual diagnoses at a smaller geographic level within a census tract (e.g., neighborhoods). The geographic distribution was determined using a qualitative evaluation of the point pattern of cancer diagnoses in Belmont CTs 3571 and 3572. In instances where the address information from the MCR was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using Registry of Motor Vehicle records and telephone books issued within 2 years of a child's diagnosis. In accordance with Massachusetts laws aimed at protecting the confidentiality of patients (M.G.L. c.111. s 24A), maps of the locations of individuals with cancer cannot be provided.

The addresses at the time of diagnosis for each child ranged from 0.3 to 0.6 miles away from the Cambridge Plating facility. Based on a review of address at the time of diagnosis for each child and given that each child was diagnosed with a different type of

cancer during different years, no apparent geographic or temporal concentrations of cancer diagnoses (of any type) were observed in any one area of Belmont CTs 3571 and 3572.

Comment 14: *Why have you not looked at trends in cancer incidence over the past five years?*

Response:
After the release of the 2005 PHA, the MDPH conducted additional analyses of cancer incidence in Belmont, Cambridge, Arlington, and Watertown in order to incorporate newly available MCR data. (The 2005 PHA contained cancer incidence data covering 1982 through 1999.) Due to the volume of information received by the MCR, the large number of reporting facilities, and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of 2 years prior to the current date. At the time of this analysis, the years 2000-2003 constituted the period for which the most recent and complete cancer incidence data were available from the MCR. The updated analysis for 2000–2003 focused on the CTs immediately surrounding the facility. Town-wide cancer incidence rates for 2000–2003 and a summary of results are also provided for Arlington, Belmont, Cambridge, and Watertown. This information was incorporated as part of the revised PHA report (see Appendix F).

Comment 15: *Since the year 2000, there seems to be elevated cancer trends with specific occurrences in the following types: Breast, Brain, Bladder, and Stomach. Why have these cancers been overlooked?*

Response:
For 2005 PHA report, the MDPH selected six cancer types (i.e., kidney, leukemia, liver, lung and bronchus, NHL, and pancreas) for evaluation 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., TCE, chromium). In order to address community concerns about breast, brain, bladder and stomach cancers, cancer incidence rates and a summary of results are provided as part of the revised PHA report (see Appendix F). The additional analysis for these cancer types focused on the CTs immediately surrounding the facility.

Comment 16: *Breast cancer is the most common form of cancer in females nationally. Both genetic and environmental factors are believed to play a role in a woman's risk of developing breast cancer. This cancer has a variety of risk factors that may not make it easy to evaluate in your study. However, it appears that breast cancer levels are elevated in specific areas of town that may warrant a closer look. All the known risk factors for breast cancer, family history, early menstruation, and having children late in life, to name a few, account for less than half of all breast cancer cases.*

Response:

To address community concerns about breast cancer, cancer incidence rates and a summary of results are provided as part of the revised PHA report (see Appendix F).

Comment 17: *Many teachers employed at Belmont High School have been diagnosed with cancer, but do not live in the town; they are exposed to Cambridge Plating on a daily basis, were they included in the study? What about residents who relocated, and were diagnosed out of the area?*

Response:
The cancer incidence evaluation included all individuals diagnosed with the types of cancer evaluated while living in the communities of Belmont, Cambridge, Arlington, and Watertown during 1982–2003. While some of the teachers and staff at Belmont High School with cancer may live or have lived in one of these four communities, individuals with cancer are reported to the MCR according to place of residence at the time of diagnosis. The MCR contains some information on a person's occupation (e.g., job title); however, it is not possible from the MCR to determine the exact location of employment for each individual diagnosed with cancer. In addition, age group and gender-specific population data are required to calculate expected rates and SIRs. Because of the need for age group and gender-specific population data, the census tract is the smallest geographic area for which cancer rates can be accurately calculated.

Although residents might move in and out of the area evaluated during the 22–year period of the study, presumably these migrations balance one another over the course of time.

Comment 18: *What about individuals who are not treated in hospitals, where they are required to report cancer incidences?*

Response:
The MDPH was able to look at the patterns of cancer in Belmont and the surrounding communities by using data collected by the MCR. The MCR is a population-based surveillance system that collects information on all Massachusetts residents diagnosed with cancer, and state law requires that all new cancer diagnoses be reported to the MCR within 6 months of diagnosis, regardless of where the diagnoses occur. According to state regulations, all health care facilities, including but not limited to hospitals and health care providers who provide diagnosis, evaluation, treatment, medical support or palliative services to patients with malignant disease or benign brain-related tumor disease shall report to the MCR. A health care facility means "any facility or institution, whether public or private, proprietary or not for profit, including but not limited to hospitals, including general hospitals, free-standing radiation therapy and outpatient oncology centers, nursing homes, hospices, all pathology and cytology laboratories, including hospital laboratories, health maintenance organizations and other outpatient facilities such as free-standing surgical centers, which diagnose, evaluate or provide cancer treatment to cancer patients (105 CMR 301)."

Estimates of MCR completeness in reporting have increased steadily since the registry was established in 1982. In its early years, the MCR was estimated to include 90-95% of all reportable cancer cases in Massachusetts. More recently, efforts to improve case ascertainment have increased completeness to more than 95%, and the MCR is considered by the North American Association of Central Cancer Registries to have achieved the gold standard for certification in recent years (MDPH 2006).

Comment 19: *The Massachusetts Department of Environmental Protection (MDEP) recommended a wording change in the recommendation regarding future characterization of groundwater conditions and flow patterns in the event of construction or development of the Cambridge Plating property, specifically they recommended the citation of the applicable regulations.*

Response:
MDPH changed the text from "…should be required by MDEP…" to "…should be conducted in accordance with MDEP regulations, the MCP 310 CMR 40.000…"

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Appendix F

Additional Analyses in Response to Public Comments

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

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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.

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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).

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Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 25% of all reported primary brain tumors and the majority of spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the neurons of the cerebellum and are most often seen in children. Also, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS 2006a). The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,820 people (7,260 men and 5,560 women) will die from this disease in 2006 (ACS 2006).

Brain and spinal cord cancers account for over 20% of malignant tumors diagnosed among children aged 0-14 (ACS 2006b). About half of all childhood brain tumors are astrocytomas and 25% are primitive neuroectodermal tumors (PNET), which spread along the spinal cord and the meninges (ACS 2006b). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme[)]. Incidence rates are higher in males than in females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack 1996).

Despite numerous scientific and medical investigations, and analyses, the causes of brain cancer are still largely unknown. Among the possible risk factors investigated in relation to this type of cancer are ionizing radiation, electromagnetic fields, occupational exposures, exposure to N-nitroso compounds, head trauma, and genetic disorders.

The most established risk factor (and only established environmental risk factor) for brain tumors (either cancerous or non-cancerous) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain tumors are caused by radiation to the head from the treatment of other cancers (ACS 2006a). Meningiomas are the most common type of tumors that occur from this type of exposure, but gliomas may also occur (Preston-Martin and Mack 1996). Among adults, the risk of developing meningiomas has been associated with full-mouth dental x-rays taken decades ago when radiation doses were higher than today. Although the relationship between low-dose radiation exposure and increased risk of brain tumors has been debated in several studies, prenatal exposure from diagnostic x-rays has been related to an increase in childhood brain tumors (Preston-Martin and Mack 1996).

In recent years, there has been increasing public concern and scientific interest regarding the relationship of electromagnetic fields (EMF) to brain cancer. However, results from recent epidemiological investigations provide little or no evidence of an association between residential EMF exposure (e.g., from power lines and home appliances) and brain tumors (Kheifets 2001). Studies also suggest that the use of handheld cellular telephones is not associated with an increased risk of primary brain cancer (Muscat et al. 2000). However, given the relatively recent use of cellular phones, evidence is preliminary and few studies have been conducted.

Other environmental factors such as exposure to vinyl chloride (used in the manufacturing of some plastics) and aspartame (a sugar substitute) have been suggested as possible risk factors for brain cancer but no conclusive evidence exists implicating these factors (ACS 2006a). Although some occupational studies have suggested that electrical and electric utility workers may be at a slightly increased risk of brain cancer, these studies have important limitations, such as exposure misclassifications and a lack of

dose-response relationships (Kheifets 2001). Some researchers have also reported an increased risk of brain tumors in adults among veterinarians and farmers. Exposures to farm animals and pets have been considered as possible risk factors because of their association with bacteria, pesticides, solvents, and certain animal oncogenic (cancer-related) viruses (Yeni-Komshian and Holly 2000). However, the relationship between farm life and brain cancer remains controversial.

Recent reports have proposed a link between occupational exposure to lead and brain cancer risk, but further analytic studies are warranted to test this hypothesis (Cocco et al. 1998). In a case-control study, the concentrations of metal and non-metal compounds in brain biopsies from patients with primary brain tumors were compared to results from an analysis of tumor-free brain tissue. Statistically significant associations were observed between the presence of brain tumors and the concentrations of silicon, magnesium, and calcium (Hadfield et al. 1998). However, further research using a larger sample size is needed to determine whether exposure to these elements plays a role in the development of brain cancer. Other occupations that may be associated with elevated risks include workers in certain health professions (e.g., pathologists and physicians), agricultural workers, workers in the nuclear industry, and workers in the rubber industry, although specific exposures have not been established (Preston-Martin and Mack 1996). Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, metal, construction, and electric workers) and the onset of brain tumors in their children have provided inconsistent results (Preston-Martin and Mack 1996).

The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A study concluded that an increased risk of pediatric brain tumor may be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin 2001). However, the role of nitrites and cured meats in the development of brain cancer remains controversial (Blot et al. 1999; Bunin 2000). Because most people have continuous, low level exposure to N-nitroso

compounds throughout their lives, further studies, especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin 1996).

Injury to the head has been suggested as a possible risk factor for later development of brain tumors but most researchers agree that there is no conclusive evidence for an association (ACS 2006a). Head trauma is most strongly associated with the development of meningiomas compared with other types of brain tumor. Several studies have found an increased risk in women with histories of head trauma; in men who boxed; and in men with a previous history of head injuries. Gliomas are the most common type of childhood brain tumor and have been positively associated with trauma at birth (e.g., Cesarean section, prolonged labor, and forceps delivery). However, other studies have found no association (Preston-Martin and Mack 1996).

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-Martin and Mack 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS 2006b). Of those afflicted with the disease, about 5-10% will develop a central nervous system tumor (Preston-Martin and Mack 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS 2006b). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack 1996).

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

In the year 2006, approximately 212,920 women in the U.S. will be diagnosed with breast cancer (ACS 2006). Worldwide, female breast cancer incidence has increased, mainly among women in older age groups whose proportion of the population continues to increase as well (van Dijck et al. 1997). A woman's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon the well-established risk factors for breast cancer. These include increased age, an early age at menarche (menstruation) and/or late age at menopause, late age at first full-term pregnancy, family history of breast cancer, and high levels of estrogen. Other risk factors that may contribute to a woman's risk include benign breast disease and lifestyle factors such as diet, body weight, lack of physical activity, consumption of alcohol, and exposure to cigarette smoke. Data on whether one's risk may be affected by exposure to environmental chemicals or radiation remains inconclusive. However, studies are continuing to investigate these factors and their relationship to breast cancer.

Family history of breast cancer does affect one's risk for developing the disease. Epidemiological studies have found that females who have a first-degree relative with premenopausal breast cancer experience a three-fold greater risk. However, no increase in risk has been found for females with a first degree relative with postmenopausal breast cancer. If women have a first-degree relative with bilateral breast cancer (cancer in both breasts) at any age, then their risk increases five-fold. Moreover, if a woman has a mother, sister or daughter with bilateral premenopausal breast cancer, their risk increases nine-fold (Broeders and Verbeek 1997). In addition, twins have a higher risk of breast cancer compared to non-twins (Weiss et al. 1997).

A personal history of benign breast disease is also associated with development of invasive breast cancer. Chronic cystic or fibrocystic disease is the most commonly diagnosed benign breast disease. Women with cystic breast disease experience a 2-3 fold increase in risk for breast cancer (Henderson et al. 1996).

According to recent studies, approximately 5 to 10% of breast cancers can be attributed to inherited mutations in breast cancer-related genes. Most of these mutations occur in the BRCA1 and BRCA2 genes. Women who inherit BRCA1 or BRCA2 gene mutations have up to an 80% chance of developing breast cancer at some point in their lifetimes (ACS 2006).

Cumulative exposure of the breast tissue to estrogen and progesterone hormones may be one of the greatest contributors to risk for breast cancer (Henderson et al. 1996). Researchers suspect that early exposures to a high level of estrogen, even during fetal development, may add to one's risk of developing breast cancer later in life. Other studies have found that factors associated with increased levels of estrogen (i.e., neonatal jaundice, severe prematurity, and being a fraternal twin) may contribute to an elevated risk of developing breast cancer (Ekbom et al. 1997). Conversely, studies have revealed that women whose mothers experienced toxemia during pregnancy (a condition associated with low levels of estrogen) had a significantly reduced risk of developing breast cancer. Use of estrogen replacement therapy is another factor associated with increased hormone levels and it has been found to confer a modest (less than two-fold) elevation in risk when used for 10-15 years or longer (Kelsey 1993). Similarly, more recent use of oral contraceptives or use for 12 years or longer seems to confer a modest increase in risk for bilateral breast cancer in premenopausal women (Ursin et al. 1998).

Cumulative lifetime exposure to estrogen may also be increased by certain reproductive events during one's life. Women who experience menarche at an early age (before age 12) have a 20% increase in risk compared to women who experience menarche at 14 years of age or older (Broeders and Verbeek 1997; Harris et al. 1992; ACS 2006). Women who experience menopause at a later age (after the age of 55) have a slightly elevated risk for developing the disease (ACS 2006). Furthermore, the increased

cumulative exposure from the combined effect of early menarche and late menopause has been associated with elevated risk (Lipworth 1995). In fact, women who have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than women with 30 years or less of menstrual activity (Henderson et al. 1996). Other reproductive events have also shown a linear association with risk for breast cancer (Wohlfahrt 2001). Specifically, women who gave birth for the first time before age 18 experience one-third the risk of women who have carried their first full-term pregnancy after age 30 (Boyle and Leake 1988). The protective effect of earlier first full-term pregnancy appears to result from the reduced effect of circulating hormones on breast tissue after pregnancy (Kelsey 1993).

Diet, and particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, a hypothesis exists that the type of fat in a woman's diet may be more important than her total fat intake (ACS 2006; Wynder et al. 1997). Monounsaturated fats (olive oil and canola oil) are associated with lower risk while polyunsaturated (corn oil, tub margarine) and saturated fats (from animal sources) are linked to an elevated risk. However, when factoring in a woman's weight with her dietary intake, the effect on risk becomes less clear (ACS 1998). Many studies indicate that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey 1993), probably due to fat tissue as the principal source of estrogen after menopause (McTiernan 1997). Therefore, regular physical activity and a reduced body weight may decrease one's exposure to the hormones believed to play an important role in increasing breast cancer risk (Thune et al. 1997).

Aside from diet, regular alcohol consumption has also been associated with increased risk for breast cancer (Swanson et al. 1997; ACS 2006). Women who consumed one alcoholic beverage per day experienced a slight increase in risk (approximately 10%) compared to non-drinkers, however those who consumed 2 to 5 drinks per day experienced a 1.5 times increased risk (Ellison et al. 2001; ACS 2006). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al. 1997).

To date, no specific environmental factor, other than ionizing radiation, has been identified as a cause of breast cancer. The role of cigarette smoking in the development of breast cancer is unclear. Some studies suggest a relationship between passive smoking and increased risk for breast cancer; however, confirming this relationship has been difficult due to the lack of consistent results from studies investigating first-hand smoke exposure (Laden and Hunter 1998).

Studies on exposure to high doses of ionizing radiation demonstrate a strong association with breast cancer risk. These studies have been conducted in atomic bomb survivors from Japan as well as patients that have been subjected to radiotherapy in treatments for other conditions (i.e., Hodgkin'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.

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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.

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