Public Health Assessment

Evaluation of Cancer, 1982–2000, and Environmental Concerns Related to the Old Colony Railroad Site and Alloy Castings Company

Old Colony Railroad Site
EPA Facility ID: MAD981061567

East Bridgewater, Plymouth County, Massachusetts

Prepared by:
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Under a Cooperative Agreement with:
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I. SUMMARY

At the request of Representative Kathleen Teahan, the East Bridgewater Board of Health, and concerned residents, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health (CEH), conducted an evaluation of cancer and possible environmental exposures in East Bridgewater, Massachusetts. The evaluation was initiated based on community concerns about possible environmental exposures and cancer in relation to the Old Colony Railroad (OCRR) site, which consists of three smaller properties, Precise Engineering, Eastern States Steel, and an old Massachusetts Bay Transportation Authority (MBTA) railroad bed. Various manufacturing operations occupied the Precise Engineering property from about 1920 to 1991 and the Eastern States Steel property from 1851 to the late 1990s. The MBTA railroad bed operated from 1847 to at least the 1930s and was originally part of the Old Colony Railroad. The OCRR site is located at the corner of Cook and West Union Streets. Community concerns also focused on cancer in the Ashley Drive neighborhood and possible environmental exposures related to Alloy Castings Company, an active aluminum foundry operating since 1948 at 151 West Union Street. This project was conducted under a cooperative agreement with the United States Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct public health assessments in Massachusetts.

The investigation provides a review of available environmental data for the OCRR site, considers potential ways people may come in contact with chemicals from this facility, and evaluates the pattern of cancer in East Bridgewater with a particular focus on neighborhoods near areas of community environmental concern. Information about Alloy Castings Company was obtained from the Massachusetts Department of Environmental Protection and the East Bridgewater Board of Health; however, there was no available environmental data related to the company. Therefore, evaluation of community concerns in relation to Alloy Castings Company was limited to a qualitative evaluation of the geographic pattern of cancer diagnoses in neighborhood areas surrounding the company.

Using data from the Massachusetts Cancer Registry (MCR), incidence rates for eight cancer types were calculated for the town of East Bridgewater as a whole as well as for each of the three

census tracts (CTs) that divide the town. Available information about risk factors, including environmental factors, related to the development of cancer was considered. In general, the eight cancer types evaluated occurred near expected rates from 1982 to 2000, the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis, for the town of East Bridgewater as a whole and for the census tracts (CTs) that comprise the town. In addition, none of the cancer types thought to be associated with exposures to contaminants of concern identified at the OCRR site were statistically significantly elevated in CT 5231, the census tract that contains both the site and Alloy Castings Company. Leukemia was statistically significantly elevated among females in East Bridgewater as a whole from 1988 to 1993. However, no apparent geographic concentrations of cases were noted. In addition, a variety of different subtypes of leukemia were represented. This indicates the occurrence of different diseases because the four major subtypes and rarer subtypes each have different risk factors related to their development.

Review of the geographic distribution of each of the cancer types in East Bridgewater revealed no apparent spatial patterns at the neighborhood level. Further, no unusual concentrations of individuals diagnosed with cancer were observed in the vicinity of the OCRR site, Alloy Castings Company, in the Ashley Drive area, or in any other area of East Bridgewater.

There are some potential exposure pathways that may have occurred in the past related to the OCRR site. Past intermittent exposures to onsite surface soil, surface water, and sediment and offsite surface soil may have been possible for trespassers, former workers, or nearby residents. However, based on the contaminant levels detected and the frequency and duration of contact expected, it is unlikely that potential exposures related to the OCRR site would have resulted in adverse health effects for trespassers, former workers, or nearby residents. While soil, surface water, and sediment in some areas of the OCRR site were contaminated in the past, the United States Environmental Protection Agency (EPA) performed contaminant removal activities at the site in 2001. Therefore, exposure to contamination in soil, surface water, or sediment in the present and future for individuals visiting or residing in neighborhoods adjacent to the site is unlikely. Potential exposure to vinyl chloride volatilized to indoor air may be possible for downgradient Spring Street residents if this contaminant is present in shallow groundwater beneath homes at concentrations similar to those detected in onsite monitoring wells in 1988.

Because offsite groundwater conditions are unknown, it would be beneficial to address this data gap with additional investigation, for example, hydrogeological modeling or groundwater sampling at downgradient locations. Based on criteria established by ATSDR, the OCRR site would be classified as posing an Indeterminate Health Hazard in the past, present, and future.

Based on the information reviewed in this evaluation, including available environmental data for the OCRR site 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 containing the OCRR site and Alloy Castings Company or in the town of East Bridgewater as a whole during the 19-year time period, 1982–2000.

II. BACKGROUND AND STATEMENT OF ISSUES

At the request of Representative Kathleen Teahan, the East Bridgewater Board of Health, and concerned residents, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health, conducted an evaluation of cancer and possible environmental exposures in East Bridgewater, Massachusetts. The Old Colony Railroad site is located at the corner of West Union and Cook Streets and consists of three distinct properties: Precise Engineering, Eastern States Steel, and an old Massachusetts Bay Transportation Authority (MBTA) railroad bed. Various manufacturing operations occupied the Precise Engineering site from about 1920 to 1991 and the Eastern States Steel property from 1851 to the late 1990s. The MBTA railroad bed operated from 1847 to at least the 1930s and was originally part of the Old Colony Railroad. For the purposes of this report, all former activities at this location are referred to as the Old Colony Railroad (OCRR) site. Community concerns also focused on cancer in the Ashley Drive neighborhood and possible environmental exposures related to Alloy Castings Company, an active aluminum foundry operating since 1948 at 151 West Union Street. This project was conducted under a cooperative agreement with the United States Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct public health assessments in Massachusetts. Refer to Figure 1 for the locations of the OCRR site, Alloy Castings Company, and the census tracts that divide the town of East Bridgewater.

This investigation provides a review of potential exposure pathways to chemicals from the OCRR site as well as a review of the pattern of cancer in East Bridgewater through comparison of the incidence of eight cancer types with the incidence of these cancers in the state of Massachusetts as a whole. Additionally, available information about risk factors, including environmental factors, related to the development of cancer was evaluated. To evaluate concerns about potential environmental exposures from the OCRR site, MDPH contacted Region 1 of the United States Environmental Protection Agency (EPA) and the Massachusetts Department of Environmental Protection (MDEP) to obtain and review available environmental data. Information about Alloy Castings Company was obtained from the MDEP and the East Bridgewater Board of Health; however, there was no available environmental data related to the company. Therefore, evaluation of community concerns in relation to this company was limited

to a qualitative evaluation of the geographic pattern of cancer diagnoses in neighborhood areas surrounding the company.

Cancer incidence rates were calculated for the town of East Bridgewater during the years 1982–2000, 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. The town of East Bridgewater is divided into three smaller areas, or census tracts (CTs). A census tract is a smaller geographic subdivision of a city or town designated by the United States Census Bureau. Because age group and gender specific population information is necessary to calculate cancer incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated. Both the OCRR site and Alloy Castings Company are located in CT 5231, in the southwest area of the town. East Bridgewater CT 5231 comprises an area of approximately 25 square kilometers and has a total population of 2,872 (U.S. Census Bureau 2000). Refer to Figure 1.

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, to determine if a common etiology (i.e., cause associated with the development) is possible, and may 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 any one individual's cancer diagnosis. The purpose of this evaluation is to report the findings on the patterns of cancer in East Bridgewater, with a particular focus on the neighborhoods in the vicinity of the OCRR site and the Alloy Castings Company, and discuss them in the context of the available environmental information to determine whether recommendations for further public health action are needed.

III. OBJECTIVES

The specific objectives of this investigation were as follows:

- To evaluate opportunities for environmental exposure(s) to nearby residents to contamination identified at the OCRR site;

- To evaluate the incidence rates of eight cancer types (leukemia, non-Hodgkin's lymphoma and cancers of the bladder, brain and central nervous system [CNS], breast, kidney, liver, and lung and bronchus) in East Bridgewater and in areas near the OCRR site and Alloy Castings Company to determine if cancer is occurring more or less often than expected;

- To evaluate the geographic distribution of individuals diagnosed with cancer in East Bridgewater in order to determine if there are any patterns in particular areas of town or in relation to areas of potential environmental concern;

- To review available descriptive information from the Massachusetts Cancer Registry (MCR) for individuals diagnosed with cancer in East Bridgewater to see if there are any particular characteristics related to known or suspected risk factors, including environmental factors, for developing these diseases; and

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

IV. COMMUNITY ENVIRONMENTAL CONCERNS

In order to address community environmental concerns related to the OCRR site, the United States EPA Region I Office in Boston, Massachusetts, and the Southeast Regional Office of the MDEP in Lakeville, Massachusetts, were contacted to obtain and review available environmental information pertaining to the site. Information related to Alloy Castings Company was obtained from MDEP and the East Bridgewater Board of Health. In addition, information regarding other

potential environmental sources located in the area and listed with MDEP as a hazardous release or spill location was reviewed (MDEP 2003c).

The public health assessment titled "Evaluation of Cancer, 1982–2000, and Environmental Concerns Related to the Old Colony Railroad Site and Alloy Castings Company in East Bridgewater, Plymouth County, Massachusetts" was released on December 26, 2006, for a 30-day public comment period. No public comments were received by the MDPH during the public comment period.

A. Old Colony Railroad Site

The OCRR site is located in southeastern Massachusetts at the intersection of Cook and West Union Streets in the town of East Bridgewater, Plymouth County, Massachusetts (Figure 2). The site is 26 miles south of Boston and is situated in a mixed commercial and residential area, approximately 1/4 mile from the center of East Bridgewater. Two abandoned businesses, Precise Engineering and Eastern States Steel, and an inactive Massachusetts Bay Transportation Authority (MBTA) bed comprise the site. Various manufacturing operations occupied the Precise Engineering property from about 1920 to 1991 and the Eastern States Steel property from 1851 to the late 1990s. The MBTA railroad bed operated from 1847 to at least the 1930s and was originally part of the Old Colony Railroad. The inoperative railroad line bisects the 8-acre site. The site is bordered to the north by West Union Street, the west by Cook Street, and the southeast by Spring Street. Approximately 531 people live within a 1/4 mile radius, and 2,567 people live within 1 mile (U.S. EPA 2001). Cook Street homes are located across the street from the site. Nine residential properties on Spring Street and one residential property on West Union Street directly abut the site. A post office is located across from the site on West Union Street.

The topography of the OCRR site is generally level. A seasonal drainage ditch extends from the southeast corner of the property, adjacent to the former Precise Engineering manufacturing building, to the southwest corner and terminates just south of the abandoned Eastern States Steel building (Figure 2) (Roy F. Weston, Inc. 2001). The drainage ditch is on the Precise Engineering property, but is outside the fence that surrounds the site, and is adjacent to a wooded, marshy area on Spring Street residential properties (R. Haworth, EPA, personal communication, 2005). Groundwater in the shallow portion of the aquifer exists approximately 2 feet below the ground

surface and flows in a south, southwestern direction (SEA Consultants 1998). Groundwater in the deeper aquifer, approximately 20 to 25 feet below the surface, has a more westerly component than the shallow groundwater. The site is located within a Zone II wellhead protection area, the area of an aquifer that could be pumped through a well under 180-day drought conditions. Limited removal activities for the entire OCRR site were completed by the EPA in June 2001. Prior to removal activities, access to the site was unrestricted due to inadequate fencing (Roy F. Weston, Inc. 2001). Presently, the buildings and equipment on the site are in disrepair and an 800-foot section of the fencing has fallen down (R. Haworth, EPA, personal communication, 2005).

It is possible that former operations at Eastern States Steel and Precise Engineering, as well as contaminants from the MBTA railroad bed, resulted in the presence of contaminants in various areas within the total 8-acre area of the OCRR site. For example, Horsley and Witten, Inc. reported that groundwater beneath the Eastern States Steel might be impacted by migration of contaminants from the abutting Precise Engineering property or MBTA railroad bed (Horsley and Witten, Inc. 1998).

1. Massachusetts Bay Transportation Authority Railroad Bed

The MBTA railroad bed extends from the southwest corner to the northeast corner of the OCRR site (Figure 2) and was part of the Old Colony Railroad line to Boston, Massachusetts, that began operating in 1847 (K. Grabau, East Bridgewater Selectmen's Office, personal communication, 2005). The railroad transported passengers, including Civil War troops in the 1860s, to and from East Bridgewater. Passenger service ended in 1925. Freight service to the Brockton Edison Company plant on Spring Street continued until approximately the 1930s or 1940s. At present, some residents are interested in creating a recreational rail trail on the now inactive MBTA railroad bed (G. Martin, MDEP, personal communication, 2005).

The Precise Engineering property and five Spring Street residences border the MBTA railroad bed to the east. To the west, it is bordered by the Eastern States Steel property. Soil sampling of the railroad bed was conducted in 1999 by Roy F. Weston, Inc. Levels of lead and arsenic were identified in surface soil above screening values established by ATSDR and MDEP. Due to the

historical use of the pesticide lead arsenate along railroad lines, lead and arsenic are commonly detected on and near railroad beds in Massachusetts (MDEP 2003c).

2. *Precise Engineering*

The Precise Engineering property at 24 West Union Street, East Bridgewater, occupies 2.26 acres of the OCRR site. The property is bordered on the north by West Union Street and one residential property on West Union Street, to the west by the MBTA railroad bed and Eastern States Steel property, and to the southeast by five Spring Street residences (Figure 2). A factory on the Precise Engineering property manufactured wooden shoe lasts (i.e., the solid form around which a shoe is molded) from as early as 1920 until it manufactured plastic shoe lasts beginning in the 1960s. Small boats were assembled on the property from 1976 to 1978. Precise Engineering operated on the site as a metalworking and stamping facility from 1978 until the property was abandoned in the early 1990s. In 2001, the EPA conducted limited removal actions at the OCRR site in order to reduce potential exposures to site contaminants. At present, the Precise Engineering portion of the OCRR site is an inactive industrial facility comprising a one-story brick factory building, paved area, and surrounding acreage.

In 1987, site investigators observed petroleum-impacted soil and groundwater adjacent to an abandoned underground fuel storage tank on the Precise Engineering property (SEA Consultants 1996). They also detected a strong chemical odor adjacent to a truck body historically used to store tetrachlorethylene (PCE). Later in 1987, the site was reported to MDEP. Briggs Associates confirmed the presence of PCE and xylenes in soil and groundwater in 1988. Also in 1988, the property owner removed approximately 100 drums of pigments, paints, cutting oils, toluene, and tetrachloroethane from the building (SEA Consultants 1998).

During a 1988 investigation, Briggs Associates found more areas of volatile organic compounds (VOCs) and petroleum hydrocarbon contamination in subsurface soils near the underground storage tank (SEA Consultants 1998). In 1996, surface water samples collected at Precise Engineering confirmed the presence of VOCs in the drainage ditch (Cambridge Environmental 1998). When polychlorinated biphenyls (PCBs) and arsenic in a soil pile and trichloroethylene (TCE) in groundwater were detected at elevated levels, the soil pile was covered with a tarp

(SEA Consultants 1996). The underground storage tank was removed in 1996 (Cambridge Environmental 1998).

SEA Consultants completed additional site assessments in 1997 and 1998. Investigators noted signs of illegal entry into the factory building. A former employee reported an historical crane fuel oil release near the former underground storage tank (SEA Consultants 1998). The former underground storage tank was identified as the probable source of polycyclic aromatic hydrocarbon (PAH) contamination in sediments of the adjacent drainage ditch. Investigators concluded that offsite sources, namely other portions of the OCRR site, probably contributed to contamination on the Precise Engineering property.

In preparation for removal actions at the OCRR site, Roy F. Weston, Inc. sampled onsite and some offsite soil in 1999 (Roy F. Weston, Inc. 2001). Lead and arsenic were detected in surface soil in the railroad bed, drainage ditch, and adjacent Spring Street residences. During removal activities, which began in August 2000 and were completed in June 2001, buried military munitions were discovered and removed from an area near the building on the Precise Engineering property and at an undeveloped residential property adjacent to the building.

According to the MDEP, the Town of East Bridgewater is currently conducting an assessment of the presence of VOCs in groundwater to the south of the OCRR site (G. Martin, MDEP, personal communication, 2007).

3. Eastern States Steel

The inactive Eastern States Steel facility is located at 36 Cook Street in East Bridgewater. The property consists of approximately 4.5 acres of the 8-acre OCRR site (Figure 2). The property is bordered on the north by West Union Street, the west by Cook Street, the southeast by the inoperative MBTA railroad bed, and the northeast by Precise Engineering. Railroad locomotives were manufactured on the property beginning in 1851 (K. Grabau, East Bridgewater Selectmen's Office, personal communication, 2005). The facility also functioned as a foundry and most recently as a new and scrap metal operation (Roy F. Weston, Inc 2001). The metal operation was active until the late 1990s (K. Grabau, East Bridgewater Selectmen's Office, personal communication, 2005).

Structures on the Eastern States Steel property include a wood frame house once used as office space, a small warehouse in the northwest corner of the property, a main brick building, and several attached brick structures (Cambridge Analytical Associates 1986). Prior to removal activities, there were numerous metal shaving piles south of the main building (Roy F. Weston, Inc. 2001). A soil pile extended along the southeastern portion of the main building for approximately 50 feet.

In 1986, Cambridge Analytical Associates reported the presence of heavy metals and PAHs in soil on the Eastern States Steel property. Two unregistered underground fuel storage tanks located to the west of the main building were removed that year. The presence of residual petroleum hydrocarbons from the tanks was confirmed in 1990 (Kupferman and Weber, Inc. 1990).

During a fire inspection in August 1996, the East Bridgewater Fire Department discovered several drums of chlorinated solvents, calcium carbide, and waste oil (Horsley and Witten, Inc. 1997). MDEP immediately issued a Notice of Responsibility to the property owner (MDEP 1997), who completed removal of the drums from the property in October 1996 (Horsley and Witten, Inc. 1997).

In April 1997, MDEP issued a second Notice of Responsibility to the owner of Eastern States Steel and ordered an Imminent Hazard Evaluation of the site after reviewing the 1986 Cambridge Analytical Associates environmental site assessment (Horsley and Witten, Inc. 1997). In June 1997, Horsley and Witten, Inc. conducted a limited site investigation and confirmed the presence of PCB- and arsenic-contaminated soil piles behind the main building. In August 1997, Horsley and Witten, Inc. collected soil samples from test pits in the area of the former underground storage tanks and the locations sampled by Cambridge Analytical Associates in 1986. Results revealed the presence of petroleum hydrocarbons in the area of the former tanks and elevated levels of PAHs and lead at the southern tip of the property.

In a 1999 investigation prior to EPA removal activities at the OCRR site, Roy F. Weston, Inc. reported elevated levels of lead, arsenic, and PCBs in surface soil surrounding the Eastern States Steel main building (Roy F. Weston, Inc. 2001). During the assessment, investigators also observed several young people on the property and were told that groups of young people often

enter the accessible buildings. During removal activities from August 2000 to June 2001, buried military munitions were discovered and removed from seven locations on the Eastern States Steel property.

B. Alloy Castings Company

Alloy Castings Company is located at 151 West Union Street, approximately 1/10 of a mile northwest of the OCRR site (Figure 2). A residential neighborhood of 13 homes on Ashley Drive is located to the northwest and across a stream from Alloy Castings Company.

Alloy Castings Company is an active aluminum foundry that manufactures decorative light poles, fire alarms, and traffic light bases for municipalities. The company has been in operation since 1948. Community concerns related to this facility focus on intermittent odorous stack emissions, and residents in the surrounding neighborhood have reported unpleasant odors to the Board of Health (R. Fillbrick, East Bridgewater Board of Health, personal communication, 2004). According to the Board of Health, Alloy Castings Company sometimes uses a particular material that emits a very pungent odor; however, the company attempts to limit this to days when wind conditions carry the odor away from residences. In 2000, MDEP inspected the facility for air quality and hazardous waste and found no violations (A. Antonelli, MDEP, personal communication, 2004). The inspection was random and was not prompted by specific community concerns. Since the facility does not discharge industrial wastewater to the stream adjacent to the property and because air emissions from the company fall under the 1-ton per year regulatory threshold, there were no available environmental data to evaluate for Alloy Castings Company.

C. Massachusetts Department of Environmental Protection 21E Hazardous Material and Oil 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, MDEP administers investigation and clean-up of hazardous material and oil release sites, known as “21E sites”, in the Commonwealth.

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 trucks and other vehicles, 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. Information on hazardous material and oil releases is available from 1977 to the present, from the MDEP Bureau of Waste Site Cleanup (MDEP 2003c). However, records prior to 1984 are known to contain significant data gaps.

Hazardous material and oil releases are *potential* sources of exposure to contamination. More detailed information is needed to determine whether individuals residing near these reported release areas were actually exposed to contaminants. This includes 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.

In addition to the evaluation of environmental data associated with the OCRR site, MDPH reviewed the most recent information regarding oil or other hazardous material releases for the town of East Bridgewater and mapped the approximate location of release sites with sufficient address information using a geographic information system (Figure 3) (ESRI 2002). A total of 71 releases were reported in the town of East Bridgewater from 1987 to 2003. The majority of these releases were mapped to an address in town; however, approximately 11% of the releases (n = 8) could not be mapped due to insufficient address information. There were five releases reported at properties on the OCRR site and none at Alloy Castings Company. The full list of releases recorded as "21E sites" in East Bridgewater is shown in Table 1.

V. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with the OCRR site and the properties that comprise the site, MDPH reviewed information from several reports on file with MDEP and EPA. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify those substances that are either not expected to result in adverse health effects or 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 United States EPA Region III (U.S. EPA 2004a) or the applicable groundwater and soil standards developed by MDEP (2003a), 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 based on health guidelines and assumed exposure situations that represent conservative estimates of human exposure. Chemical concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, chemical concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a compound to impact one's health, it must not only be present in the environmental media, but one must also come in contact with the compound. Therefore, if a concentration of a chemical is greater than the appropriate comparison value, the potential for exposure to the chemical should be 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 if an adverse health effect from this chemical could occur.

The ATSDR compiled levels of metals and PAHs that are considered normal for soil of urban and suburban communities (ATSDR 2003c). The United States Geological Survey (USGS) identified levels of metals that are considered typical for soil in the eastern United States (Shacklette and Boerngen 1984), and the MDEP compiled soil background data that are specific to Massachusetts (MDEP 2002). These levels are "background" and were used along with comparison values for both metals and PAHs in this analysis.

A. Soil

In the absence of excavation activities, opportunities for exposures to chemicals are generally expected to be greater for surface soil than for subsurface soil. Hence, surface and subsurface soil sampling results are addressed separately. A summary of the maximum concentration of contaminants detected above comparison values in surface and subsurface soil is presented in Table 2.

1. Surface Soils

From 1999 to 2000, 148 surface samples from the top 3 inches of soil were collected in preparation for EPA removal activities from areas throughout the entire OCRR site. A 50-foot by 50-foot sampling grid was constructed, and a sample was collected at each grid point. Additionally, 201 samples were collected from the top 3 inches of soil at the soil piles, drainage ditch, adjacent residential properties, and other areas with stained soil and stressed vegetation. All samples were analyzed for lead, arsenic, and polychlorinated biphenyls (PCBs), and all three contaminants were detected onsite at concentrations exceeding comparison values.

Near the Eastern States Steel office building, the highest lead level detected in surface soil at the site was 7,840 parts per million (ppm), which was above the MDEP action level of 300 ppm and typical soil background levels for lead (less than 10–300 ppm) (Table 2) (USGS 1984). The highest levels of lead, well above the action level and background levels, in surface soil were concentrated along the western and southern perimeter of the Eastern States Steel main building and around the outbuildings and concrete slabs along Cook Street. The area north of the main Eastern States Steel building and the soil piles in this area also tested well above the action level and background levels for lead. Samples from the southern corner of the Precise Engineering property and the large soil pile on that parcel, in addition to a sample from the southern end of the MBTA railroad bed, were well above the action level and background levels for lead. Other areas of the site generally tested above both the action level and background levels for lead.

The highest concentration of arsenic (201 ppm) in surface soil was located near the northern section of the MBTA railroad bed. This concentration was above the Cancer Risk Evaluation Guide (CREG) value (0.5 ppm) and background levels [mean of 7.4 ppm (USGS 1984), 90th

percentile of 20 ppm (MDEP 2002)]. In general, the highest arsenic levels in surface soil exceeded background levels and were located at the Eastern States Steel soil pile and on or near the railroad bed.

The highest concentration of PCBs (22,500 ppm) found in surface or subsurface soil at the OCRR site occurred in the northeastern corner of the Eastern States Steel property. The next highest PCB level measured in site surface soil (1,400 ppm) was located in the same area. The CREG for PCBs is 0.4 ppm. PCBs were also detected above the CREG value in soil samples collected west of the main Eastern States Steel building and among the outbuildings in this area (up to 840 ppm).

Twenty-five samples from the top 3 inches of soil were taken from the backyards of eight adjacent residential properties on Spring Street (one of which was undeveloped) and one residential property on West Union Street that is adjacent to the Precise Engineering Building. Each sample was analyzed for lead, arsenic, and PCBs. Eight additional samples were analyzed for lead only. Lead was detected above the MDEP action level (300 ppm) and typical background levels (less than 10–300 ppm) at 4,780 ppm at the undeveloped residential property, which is next to the Precise Engineering building. Lead was detected at this undeveloped property above the action level and background levels in four samples in the immediate vicinity of buried military munitions and was not measured above the action level at any other residential property.

The maximum concentration of arsenic measured on residential property was detected above the CREG value (0.5 ppm) at 58 ppm and background levels [mean of 7.4 ppm (USGS 1984), 90th percentile of 20 ppm (MDEP 2002)] at a Spring Street residence adjacent to the MBTA railroad bed. Arsenic was also detected above the CREG at five other Spring Street residences (maximum concentrations of 4.4 ppm, 5.9 ppm, 12.2 ppm, 20.4 ppm, 43.8 ppm) and at the West Union Street residence (maximum concentration of 5.8 ppm). Arsenic levels detected in residential soil were highest in backyards adjacent to the southern edge of the railroad bed, where a maximum concentration of arsenic (120 ppm) in railroad bed surface soil was located. Elevated levels of arsenic are common on and near railroad beds because of the use of arsenic-based herbicides to control weed growth (MDEP 2003c). In all, 18 out of 25 surface soil

samples from the nine residential backyards tested had arsenic levels above the CREG. According to the available sampling data, PCBs were not detected above the CREG value (0.4 ppm) in any sample from a residential property along Spring Street or from the West Union residence.

Seven samples from the top 3 inches of soil were also taken from an area directly adjacent to the street in the front yards of Cook Street residences (R. Haworth, EPA, personal communication, 2005). They were analyzed for lead, arsenic, and PCBs. The highest lead level in these seven samples was 243 ppm, which is below the action level of 300 ppm and within typical background levels (less than 10–300 ppm). The highest concentration of arsenic was 11.2 ppm, which is above the CREG (0.5 ppm), but within typical background levels of this metal (less than 0.1–73 ppm). There were also three detections of PCBs slightly above the CREG (0.4 ppm) that ranged from 0.42–0.84 ppm. However, these concentrations were below the MDEP action level (2 ppm) for PCBs.

As part of the EPA removal activities that were completed in 2001, surface and subsurface soil with PCB concentrations exceeding 50 ppm (which occurred onsite only) was excavated and transported offsite for disposal. Specifically, PCB-contaminated soil was removed from square grids measuring 25 feet on each side, and additional excavation was performed based on samples from the bottom and top edges of each excavated grid. The entire site was covered with geotextile fabric, and an average of 10 inches of clean soil (excluding the depth of clean soil added to excavated areas) was spread across the site. Clean soil cover was much deeper in areas where the soil piles and soil with PCBs above 50 ppm had been excavated. At three adjacent Spring Street residential properties, soil with concentrations of lead or arsenic exceeding MDEP action levels (30 ppm for arsenic and 300 ppm for lead) was excavated and replaced with clean soil (PCBs were not detected above the MDEP action level of 2 ppm at any residential property) (R. Haworth, EPA, personal communication, 2002). Subsurface soil was removed as necessary based on soil testing of the excavated bottom until all samples were below MDEP action levels (these sample results were unavailable). The three Spring Street properties include the undeveloped parcel next to the Precise Engineering building where buried military munitions were discovered and two residential backyards adjacent to the MBTA railroad bed. While neither lead, arsenic, nor PCBs were detected in surface soil at the West Union residential

property, half of the backyard surface soil on this property was excavated and replaced with clean soil because arsenic (75.5 ppm) was detected on the Precise Engineering property directly adjacent to this backyard at a level above the MDEP action level of 30 ppm (R. Haworth, EPA, personal communication, 2002).

2. Subsurface Soils

A total of 64 subsurface soil samples up to 48 feet deep were tested for lead during environmental investigations at the OCRR site. In 1997, a composite sample of the first foot of soil from the large soil pile southwest of the Precise Engineering building had a lead level of 18,100 ppm, which was well above the MDEP action level (300 ppm) and background levels (less than 10–300 ppm) (Table 2). This was the highest level measured at the entire OCRR site. Subsurface lead levels above the action level and background levels were located at the large Precise Engineering soil pile, the Eastern States Steel soil pile, and the soil piles in the northern area of the site.

Twenty subsurface soil samples were analyzed for cadmium and 22 were analyzed for total chromium from depths up to 48 feet. A composite sample from a depth of 0–1 feet from the large Precise Engineering soil pile had maximum concentrations detected at the 8-acre site for cadmium (20.7 ppm) and total chromium (428 ppm). The maximum detected concentration of cadmium was above the chronic Environmental Media Evaluation Guide (EMEG) for childhood exposure (10 ppm) and below the chronic EMEG for an adult (100 ppm). This level also exceeded cadmium background levels (0.01–1 ppm) (ATSDR 2003c). Cadmium contamination greater than background levels was located primarily in soil piles. The maximum concentration of total chromium detected (428 ppm) was above the hexavalent chromium Reference Dose Media Evaluation Guide (RMEG) for a child (200 ppm) and below the RMEG for adult exposure (2,000 ppm). This concentration was within background levels for total chromium (1–1,000 ppm) (USGS 1984). Only one other sample, taken from the same soil pile, exceeded the RMEG for childhood exposure.

Nine soil samples from various depths up to 48 feet were tested for antimony. Antimony (26.3 ppm) was detected from a depth of 0–1 feet in the large Precise Engineering soil pile. This level exceeded the child RMEG (20 ppm), but was below the adult RMEG (300 ppm). This

concentration also exceeded backgrounds levels for antimony (less than 1–8.8 ppm) (USGS 1984). Two other subsurface soil samples, located in the same soil pile, exceeded background levels.

Investigations of the OCRR site analyzed 25 subsurface soil samples ranging up to 48 feet in depth for several PAHs. Indeno(1,2,3-c,d)pyrene (29 ppm) was detected above the comparison value, but within background levels (8.0–61 ppm for urban soil), in 1985 in a composite sample from test pits 0–7 feet deep at Eastern States Steel (ATSDR 2003c). For indeno(1,2,3-c,d)pyrene in residential soil, the EPA Risk Based Concentration (RBC) is 0.87 ppm. South of the main Eastern States Steel building, five PAHs were detected in 1997 above comparison values in a soil sample collected from 4–8 feet deep. Benzo(a)anthracene (33 ppm) was detected above the EPA RBC for residential soil (0.87 ppm), but within background levels (0.169–59 ppm for urban soil) (ATSDR 2003c). Benzo(a)pyrene (42 ppm) was detected above the CREG value (0.1 ppm) and above background levels (0.165–0.22 ppm for urban soil) (ATSDR 2003c). Benzo(b)-fluoranthene (65 ppm) was detected above the EPA RBC for residential soil (0.87 ppm) and slightly above background levels (15–62 ppm for urban soil) (ATSDR 2003c). Benzo(k)-fluoranthene was detected (27 ppm) above the EPA RBC for residential soil (8.7 ppm) and slightly above background levels (0.3–26 ppm for urban soil) (ATSDR 2003c). Dibenzo(a,h)-anthracene was detected (11 ppm) above the EPA RBC for residential soil (0.087 ppm).

Thirteen subsurface soil samples were analyzed for the PCB Arochlor 1254 at the OCRR site. A sample from 6 inches below the surface of the soil pile behind the main Eastern States Steel building had an elevated concentration of Arochlor 1254 (310 ppm), which is above both the child (1 ppm) and adult (10 ppm) chronic EMEG. One other sample from the Eastern States Steel soil pile had a concentration of Arochlor 1254 (4.3 ppm) above the child EMEG. Arochlor 1254 was not detected at Precise Engineering. Another sample from 6 inches below the surface of the Eastern States Steel soil pile had a concentration of 2.7 ppm for Arochlor 1260, which exceeds the CREG value for total PCBs (0.4 ppm).

A 1997 investigation at Eastern States Steel focused on subsurface soil near the former underground storage tanks. Concentrations of arsenic (18,100 ppm) and Arochlor 1248 (1,000 ppm) in soil 4 feet below the surface exceeded comparison values. The CREG value for arsenic

is 0.5 ppm. The CREG value for total PCBs is 0.4 ppm. Subsurface arsenic levels also exceeded background levels (<0.1–73 ppm).

In 1999, 38 subsurface soil samples were collected from locations on the OCRR site in preparation for the EPA removal activities that occurred from 2000 to 2001. The highest level of PCBs detected in subsurface soil (1,300 ppm) was collected from the northern part of the site, to the west of the railroad bed, and exceeded the CREG value (0.4 ppm). The soil sampled was 0–1.5 feet below the surface, in the same grid location as the surface soil sample that tested the highest for PCBs (22,500 ppm) at the OCRR site. As part of the EPA removal activities, subsurface soil with PCB concentrations exceeding 50 ppm (occurred only onsite) was excavated and transported offsite for disposal. Specifically, PCB-contaminated soil was removed from square grids measuring 25 feet on a side, and additional excavation was performed based on testing of samples from the bottom and top edges of each excavated grid. In all, seven 25-feet by 25-feet grids were excavated. Three grids were excavated to a depth of 1 foot, two grids to a depth of 1.5 feet, one grid to a depth of 2 feet, and one grid to a depth of 2.5 feet.

B. Groundwater

The OCRR site lies on the edge of the Zone II groundwater protection area for East Bridgewater Well #5. A Zone II is defined as an area of an aquifer that contributes water to a well under the most severe pumping and recharge conditions that can be realistically anticipated. It is the area of an aquifer that might be pumped under 180-day drought conditions. Well #5 is one of five wells operated by the East Bridgewater Water Department and is about 1.6 miles to the southeast of the OCRR site (S. McCann, East Bridgewater Water Department, personal communication, 2004). This single well operates continuously. The other municipal wells are farther away in the southeastern part of the town.

No drinking water wells exist on the OCRR site, and there are no known private wells in the area. Residents and businesses along the surrounding streets are connected to the municipal water supply. Based on a hydrogeological survey, site groundwater in the shallow aquifer is approximately 2 feet below the surface and appears to flow in a south-southwestern direction at a rate of 2 to 5 feet per year (SEA Consultants 1998). Groundwater flow in the deeper aquifer,

between 20 and 25 feet below the surface, has a more westerly component than the shallow groundwater.

Table 3 summarizes the maximum concentrations of contaminants detected in onsite groundwater samples that exceeded comparison values. Because ATSDR comparison values do not exist for groundwater, drinking water comparison values were used as screening values. In 1988, six shallow monitoring wells (OW-1 through OW-6) were sampled at Precise Engineering for VOCs. Some VOCs, including 1,2-trans-dichloroethylene (trans-DCE), TCE, and vinyl chloride, were detected above comparison values in 1988 in a monitoring well (OW-3) about 20 feet west of the southwest corner of the Precise Engineering building and former underground storage tank. DCE (trans-) (1,020 ppb) was detected below the child (2,000 ppb) and adult (7,000 ppb) Intermediate EMEG, but above the child (200 ppb) and adult (700 ppb) RMEG. TCE (313 ppb) was detected above the EPA RBC for tap water of 0.026 ppb. Vinyl chloride (1,170 ppb) was detected above the EPA Maximum Contaminant Level Goal (MCLG) for drinking water (0 ppb). In a monitoring well (OW-2) near the former chlorinated solvent storage area, benzene was detected above the comparison value (CREG = 0.6 ppb) at 18.4 ppb. A composite sample was collected from three of the monitoring wells (OW-1, OW-2, and OW-3) and analyzed for metals. Arsenic was detected at 10 ppb, which is above the CREG (0.02 ppb) and equal to the EPA Maximum Contaminant Level (MCL). Mercury was detected (140 ppb) above the EPA MCL of 2 ppb for inorganic mercury.

In 1996, three of the shallow monitoring wells at Precise Engineering that were sampled in 1988 (OW-3, OW-4, OW-5) were tested for VOCs, PCBs, and metals. Cadmium (60 ppb) was measured above the child (2 ppb) and adult (7 ppb) chronic EMEG for drinking water. Chromium (90 ppb) was detected above the child RMEG (30 ppb) and below the adult RMEG (300 ppb) for hexavalent chromium. Lead was detected at 100 ppb, which exceeds the EPA MCLG of 0 ppb. These three metals were detected in the same well (OW-5) on the Precise Engineering property. The exact location of this well is unknown. DCE (cis-) was detected above the EPA RBC for tap water (61 ppb) at 65 ppb in monitoring well OW-3, which was about 20 feet west of the southwest corner of the Precise Engineering building and the former underground storage tank.

In 1997, five shallow groundwater samples (from 12–18 feet deep) and three deep groundwater samples (one 48-feet deep and two 71-feet deep) were collected from eight new monitoring wells at Precise Engineering. One existing shallow monitoring well (OW-4) was also sampled. All samples were analyzed for VOCs. Additionally, two shallow wells were analyzed for polycyclic aromatic hydrocarbons (PAHs), and one shallow well was analyzed for PAHs, PCBs, and metals. PCE was detected in one of the shallow wells (SEA-2S) in the northeast corner of the property near the railroad bed at 186 ppb, which is above the RMEG for children (100 ppb) and below the adult RMEG (400 ppb). TCE was also detected in the same well at 9.9 ppb, which is above the EPA RBC for tap water (0.026 ppb). Monitoring well SEA-2S was the most upgradient well of those sampled in 1997 on the Precise Engineering property. At the most downgradient well (SEA-5S) sampled in 1997, PCE was detected at 5.7 ppb and TCE was not detected; therefore, investigators suggested that VOCs might be coming from an offsite source (SEA Consultants 1998). MDEP confirmed that, in addition to potential onsite sources, VOCs in groundwater at Precise Engineering may have also originated from wastewater that was improperly disposed of at a nearby dry cleaner (G. Martin, MDEP, personal communication, 2005).

No other contaminant was detected above comparison values in the monitoring wells that were sampled in 1997. Therefore, based on the available data, it appears that contaminant concentrations in groundwater at Precise Engineering decreased from the time groundwater was first sampled in 1988 to when it was last sampled in 1997.

VOCs and metals have never been detected in East Bridgewater Well #5 (S. McCann, East Bridgewater Water Department, personal communication, 2005). The water department regularly tests Well #5 for VOCs and metals, according to state and federal laws, and does not maintain monitoring waivers for these contaminants.

C. Surface Water

Because ATSDR comparison values do not exist for surface water, drinking water comparison values were used as screening values. This is a conservative evaluation because guidelines for chemicals in drinking water assume adults ingest two liters of water per day. Exposures to chemicals present in surface water not used for drinking water purposes would be expected to be less than exposures to chemicals in drinking water.

A seasonal drainage ditch extends from the eastern edge of the site to the western portion along the southern boundary of the Precise Engineering property. It is on the Precise Engineering property, but is outside the fence that surrounds the site, and is adjacent to a wooded area on Spring Street residential properties (R. Haworth, EPA, personal communication, 2005). In 1988, two surface water samples were collected from an upstream and downstream location and analyzed for VOCs (SEA Consultants 1998). Only toluene was detected in the upstream sample and at a level below comparison values. DCE, PCE, TCE, and vinyl chloride were detected at concentrations exceeding comparison values in the downstream sample only. DCE was measured at 145 ppb, which is above the EPA MCL for drinking water (100 ppb) and below both the Intermediate EMEG (2,000 ppb) and RMEG (200 ppb) for children. PCE was detected (176 ppb) above the child RMEG of 100 ppb and below the adult RMEG of 400 ppb. TCE was measured in the downstream sample at 59.7 ppb, which exceeds the MCLG of 0 ppb and the MCL of 5 ppb. Vinyl chloride was detected (14.5 ppb) above the MCLG value (0 ppb). Site investigators concluded that contaminated onsite groundwater discharging to the drainage ditch impacted the downstream sample (SEA Consultants 1998). A summary of the maximum concentrations of contaminants detected in surface water samples that exceeded comparison values is located in Table 4.

In 1997, three surface water samples (upstream, midstream, downstream) were collected from the drainage ditch and analyzed for VOCs. No VOCs were detected in the upstream sample. TCE, PCE, and DCE were detected in the midstream sample, which was located adjacent to the former solvent storage area and near the crane fuel oil release. TCE and PCE levels slightly exceeded comparison values. TCE was detected (6.3 ppb) above the MCLG value (0 ppb) and the MCL value (5 ppb). PCE was measured at 16.8 ppb, which exceeds the MCL of 5 ppb, but is below the RMEG for children (100 ppb). DCE was the only VOC detected in the downstream sample, and it was detected below comparison values. Thus, it appears that contaminant concentrations in surface water have decreased from the time the drainage ditch was sampled in 1988 to when it was sampled again in 1997.

D. Sediment

Because ATSDR comparison values do not exist for sediment, soil comparison values were used. A summary of the maximum concentrations of contaminants detected in onsite sediment samples that exceeded comparison values for soil is presented in Table 5. In 1997, three sediment samples from upstream, mid-stream, and downstream locations in the drainage ditch were tested for PAHs, metals, PCBs, and VOCs. PAHs were reported in each sample at levels above comparison values. The mid-stream sample had the highest PAH concentrations. In this sample, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and dibenzo(a,h)anthracene levels exceeded comparison values. Benzo(a)anthracene was measured at 8.72 ppm, which exceeds the EPA RBC for residential soil (0.87 ppm), but is within background levels (0.169–9 ppm for urban soil. Benzo(a)pyrene (19.7 ppm) was detected above the CREG value (0.1 ppm) and above background levels (0.165–0.22 ppm for urban soil). Benzo(b)fluoranthene (30.9 ppm) was detected above the RBC for residential soil (0.87 ppm), but within background levels (15–62 ppm for urban soil). Dibenzo(a,h)anthracene (49.4 ppm) was also detected above the RBC for residential soil (0.087 ppm). Lead was reported in the upstream sample at 436 ppm, which exceeds the MDEP soil cleanup standard of 300 ppm, but is within background levels for urban soil (160–840 ppm). Arsenic was detected in the downstream sample at 42.6 ppm, which exceeds the CREG value (0.5 ppm), but is within soil background levels (0.1–73 ppm).

During EPA removal activities from 2000 to 2001, the drainage ditch bank adjacent to Spring Street residences was reconstructed using clean soil. During a subsequent cleanup by the MDEP, soil from the segment near the former underground storage tank was excavated up to the foundation of the Precise Engineering building in order to control the problem of subsurface petroleum leaching into the ditch.

VI. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS AND HEALTH CONCERNS

An evaluation of potential pathways of exposure was conducted to determine whether releases or activities at the OCRR site could be impacting residents of East Bridgewater in the past, present, or future. Exposure to a chemical must first occur before any potential adverse health effects can

result. Five conditions must be present for exposure to occur. First, there must be a source of that 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 contact the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, and dermal absorption. Finally, 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 1993a).

A completed exposure pathway indicates that exposure to humans occurred in the past, is occurring in the present, or will occur in the future. A completed exposure pathway exists when all of the five elements are present. A potential exposure pathway exists when one or more of the five elements is missing or uncertain and indicates that exposure to a contaminant could have occurred in the past, could 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.

To evaluate the potential for health effects, ATSDR Minimal Risk Levels (MRLs) were compared to exposure estimates for the contaminants of concern at the OCRR site. Specifically, the contaminants of concern are lead, arsenic, and PCBs in soil and TCE in surface water. The MRL is an estimate of daily exposure to a contaminant below which noncancer, adverse health outcomes are unlikely to occur. In addition, exposure estimates for contaminants of concern were combined with EPA cancer slope factors provided by ATSDR to evaluate potential cancer risk.

A. Exposure to Soil

Prior to cleanup by the EPA, access to the OCRR site was unrestricted. There is evidence that nearby residents, youths in particular, accessed the site. In the past, trespassers may have been exposed through incidental ingestion of or dermal contact with contaminants such as lead, arsenic, and PCBs detected in onsite surface soil at levels above comparison values. However, it is important to consider that comparison values are based on a residential exposure scenario, and

it is unlikely that a trespasser would have had contact with onsite surface soil for a comparable frequency and duration of time.

The EPA and the International Agency for Research on Cancer (IARC) have classified PCBs as a probable human carcinogen. Although scientists do not know if PCBs can cause cancer in humans, some studies have found that rats exposed to PCBs developed liver cancer. Studies investigating a possible association between environmental exposure to PCBs in the general population and development of breast cancer and NHL are inconclusive. Assuming that an older child trespassed on the site and incidentally ingested the maximum concentration of PCBs detected in surface soil (22,500 ppm) for 2 days every week for 26 weeks over 10 years, they could have been exposed to PCBs at a level that could have presented an increased cancer risk[1]. However, these exposure assumptions are conservative, and it is very unlikely that a trespasser would have had consistent contact with soil containing the highest concentration of PCBs, which was located in one particular area of the OCRR site. It is more likely that soil with a range of contaminant concentrations could have been ingested over time. Under a more realistic assumption that an older child who trespassed regularly on the site could have been exposed to the average concentration of PCBs (85 ppm), an increased cancer risk would not have been likely[2].

Arsenic is classified as a known human carcinogen by the EPA and IARC, and ingestion of inorganic arsenic has been reported to increase the risk of developing cancers of the bladder,

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[1] Cancer Effects Exposure Factor = $\frac{(2 \text{ days/week}) (26 \text{ weeks/year}) (10 \text{ years})}{(70 \text{ years}) (365 \text{ days/year})} = 0.02$

Cancer Effects Exposure Dose = $\frac{(\text{max. contaminant concentration}) (\text{ingestion rate}) (\text{exposure factor}) (1 \text{ kg}/10^6 \text{ mg})}{\text{body weight}}$

$= \frac{(22{,}500 \text{ mg/kg}) (200 \text{ mg/day}) (0.02) (1 \text{ kg}/10^6 \text{ mg})}{35 \text{ kg}} = 2.6 \times 10^{-3} \text{ mg/kg/day}$

Cancer Risk = Cancer Effects Exposure Dose x Cancer Slope Factor

$= 2.6 \times 10^{-3} \text{ mg/kg/day} \times 2.0 (\text{mg/kg/day})^{-1} = 5 \times 10^{-3}$

[2] Cancer Effects Exposure Dose = $\frac{(85 \text{ mg/kg}) (200 \text{ mg/day}) (0.02) (1 \text{ kg}/10^6 \text{ mg})}{35 \text{ kg}} = 1 \times 10^{-5} \text{ mg/kg/day}$

Cancer Risk = $1 \times 10^{-5} \text{ mg/kg/day} \times 2.0 (\text{mg/kg/day})^{-1} = 2 \times 10^{-5}$

kidney, liver, lung and certain types of skin cancer (ATSDR 1993b, 2003c). If a trespasser inadvertently ingested surface soil with the maximum concentration of arsenic detected on the site (210 ppm) for 2 days every week for 26 weeks over 10 years, the exposure would not be expected to result in an increased cancer risk[3]. Although an increased cancer risk is not expected, Section VII provides an evaluation of cancers possibly associated with arsenic exposure (i.e., bladder, kidney, liver, and lung). The MCR does not collect data on individuals diagnosed with the types of skin cancer that have been identified in the scientific literature as being associated with arsenic exposure (i.e., non-melanoma skin cancer); therefore, this cancer type was not able to be evaluated in relation to the OCRR site. If a trespasser inadvertently ingested surface soil with the maximum concentration of arsenic under the same exposure conditions described above, the level would be below ATSDR's MRL[4]. Therefore, noncancer health effects to a trespasser would also not be expected.

In humans, the main target for lead toxicity is the nervous system. Lead exposure is of most concern for young children because children exposed to lead, primarily due to the presence of lead paint in housing, may experience neurological damage (including learning disabilities) and behavioral changes. In order to evaluate potential health concerns related to exposure opportunities to lead in soil at the OCRR site, MDPH used the U.S EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children. This model is widely used throughout the country to predict blood lead levels based on lead intake via several sources (e.g., soil, food, water). Environmental data specific to a site are input into the model to predict blood lead levels for young children (aged 6 months to 7 years). The model generally uses typical or average concentrations in the various source media, assumes daily exposures, and predicts blood lead concentrations based on chronic exposures (e.g., 1 year or more).

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[3] Cancer Effects Exposure Dose = $\frac{(210\ mg/kg)\ (200\ mg/day)\ (0.02)\ (1\ kg/10^6\ mg)}{35\ kg} = 2.4 \times 10^{-5}$ mg/kg/day

Cancer Risk = 2.4×10^{-5} mg/kg/day x 1.5 $(mg/kg/day)^{-1} = 4 \times 10^{-5}$

[4] Noncancer Effects Exposure Factor = $\frac{(2\ days/week)\ (26\ weeks/year)\ (10\ years)}{(10\ years)\ (365\ days/year)} = 0.14$

Noncancer Effects Exposure Dose = $\frac{(210\ mg/kg)\ (200\ mg/day)\ (0.14)\ (1\ kg/10^6\ mg)}{35\ kg} = 1.7 \times 10^{-4}$ mg/kg/day

Chronic MRL = 3×10^{-4} mg/kg/day

At the OCRR site, the average soil lead concentration was 560 ppm. It is unlikely that children under the age of 7 years would have trespassed on a daily basis on the site in the past. However, to be conservative, MDPH ran the IEUBK model with the assumptions that daily exposure occurred at the site and that half of a child's typical daily incidental soil ingestion occurred during the time spent on the site. Using these assumptions, the model predicted that 4 percent of children under the age of 7 years trespassing/playing on the site would be predicted to have blood lead levels greater than 10 micrograms per deciliter (µg/dL), which the Centers for Disease Control and Prevention (CDC) define as a level of concern (ATSDR 2004). The predicted mean blood lead concentration was 4.4 µg/dL. If we assumed that children played on the site for 180 days per year, the model predicted that 2% of children under the age of 7 years would have blood lead levels at 10 µg/dL or greater and that the mean blood lead concentration would be 3.7 µg/dL. Thus, it appears unlikely that young children would have had blood lead levels above the current CDC level of concern given historical exposure opportunities at the site. In addition, the predictions from both scenarios are below the EPA Office of Solid Waste and Emergency Response specified level of protectiveness of no more than 5% risk of an elevated blood lead level (U.S. EPA 2002).

In addition to using the IEUBK model, data on blood lead levels in children living near the site were obtained from the CEH Childhood Lead Poisoning Prevention Program (CLPPP). CLPPP was established for the prevention, screening, diagnosis, and treatment of lead poisoning in children residing in Massachusetts. The Massachusetts Lead Law requires that all children be tested for blood lead levels once between the ages of 9 months and 12 months, and again at ages 2 and 3 (CLPPP 2005).

Between 1991, the first year that CLPPP testing data are readily available, and June 2005, there were two children with elevated blood lead levels (i.e., greater than 10 µg/dL) living on streets very near the site. One of the two children had an initial screening result of 19 µg/dL with a capillary test that was not confirmed with a follow-up venous test; therefore, it is unknown whether the initial result would have been confirmed with the more reliable and accurate venous sample. The second child was tested with a venous test and had a blood lead level of 19 µg/dL. When retested 3 months later with a venous test, the child had a blood lead level of 10 µg/dL.

The most important source of elevated blood lead levels in Massachusetts children is lead paint in older homes (CLPPP 2005). Many homes built before 1978 have lead paint on the interior and exterior of the building (CLPPP 2005). Available data from CLPPP indicate that the residence of one of the children with an elevated blood lead level near the OCRR site had some lead abatement performed 8 years before the child's blood lead test, indicating that the residence had lead paint in the past (CLPPP 1991). Although abatement resulted in compliance with the Massachusetts Lead Law at that time, available information did not indicate whether all lead paint was removed or whether other violations may have surfaced in subsequent years due to further degradation of remaining lead paint. No information on the residence of the other child in terms of the presence of lead paint was available; however, 36% of housing units in East Bridgewater were built before 1950 (U.S. Census Bureau 1990). The housing stock in the vicinity of the site appeared to be of a mid-1900s vintage (D. LaPointe, CEH, site visit, 2004); therefore, it is possible that houses in the vicinity of the site have had lead paint. In addition, the percentage of blood lead test results that exceeded a level of concern (10 ug/dL) in children living near the site appeared to be no different than that observed townwide. Specifically, of all test results reported to CLPPP for blood lead level from 1991 to June 2005 among East Bridgewater children, 10% (n = 515) were equal to or greater than 10 µg/dL. Of all test results during the same time period among children residing within approximately 1/4 of a mile of the OCRR site, 11% (n = 9) were equal to or greater than 10 µg/dL. Thus, it does not appear that children living close to the OCRR site are experiencing higher blood lead levels when compared to the town as a whole.

Lead is classified by IARC as a possible human carcinogen based on some evidence of kidney cancer in animals (ATSDR 2003c). Because there is no cancer slope factor for evaluating potential cancer risk from exposure to lead, the incidence and pattern of kidney cancer were evaluated in Section VII for the town of East Bridgewater as a whole and in relation to the OCRR site.

Because the entire site was covered with clean soil during EPA removal activities, present and future ingestion of contaminants in onsite surface soils by trespassers were eliminated as exposure pathways.

When the facilities on the OCRR site were in operation, employees may have been exposed through incidental ingestion to contaminants such as PCBs and arsenic (lead is of concern for children and has not been conclusively linked to cancer in humans). Assuming that an employee inadvertently ingested surface soil with the maximum PCB concentration (22,500 ppm) for 5 days a week, for 50 weeks, over 30 years, the site could have presented a moderate risk of cancer to some exposed workers[5]. However, the exposure assumptions are conservative, and it is unlikely that an employee would have consistently ingested soil with the maximum PCB concentration detected onsite. If an employee was exposed to the average concentration of PCBs (85 ppm) under the same conditions, exposure would not pose an increased cancer risk for workers[6].

For employees who might have inadvertently ingested the maximum concentration of arsenic detected at the OCRR site for 5 days a week, for 50 weeks, over 30 years, no increased cancer risk would be expected[7]. The same exposure conditions would also be unlikely to result in adverse noncancer health effects[8].

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[5] Cancer Effects Exposure Factor = $\frac{(5 \text{ days/week}) (50 \text{ weeks/year}) (30 \text{ years})}{(70 \text{ years}) (365 \text{ days/year})} = 0.29$

Cancer Effects Exposure Dose = $\frac{(22{,}500 \text{ mg/kg}) (50 \text{ mg/day}) (0.29) (1 \text{ kg}/10^6 \text{ mg})}{70 \text{ kg}} = 4.7 \times 10^{-3}$ mg/kg/day

Cancer Risk = 4.7×10^{-3} mg/kg/day x 2.0 $(\text{mg/kg/day})^{-1} = 9 \times 10^{-3}$

[6] Cancer Effects Exposure Dose = $\frac{(85 \text{ mg/kg}) (50 \text{ mg/day}) (0.29) (1 \text{ kg}/10^6 \text{ mg})}{70 \text{ kg}} = 1.8 \times 10^{-5}$ mg/kg/day

Cancer Risk = 1.8×10^{-5} mg/kg/day x 2.0 $(\text{mg/kg/day})^{-1} = 4 \times 10^{-5}$

[7] Cancer Effects Exposure Dose = $\frac{(210 \text{ mg/kg}) (50 \text{ mg/day}) (0.29) (1 \text{ kg}/10^6 \text{ mg})}{70 \text{ kg}} = 4.4 \times 10^{-5}$ mg/kg/day

Cancer Risk = 4.4×10^{-5} mg/kg/day x 1.5 $(\text{mg/kg/day})^{-1} = 7 \times 10^{-5}$

[8] Noncancer Effects Exposure Factor = $\frac{(5 \text{ days/week}) (50 \text{ weeks/year}) (30 \text{ years})}{(30 \text{ years}) (365 \text{ days/year})} = 0.68$

Noncancer Effects Exposure Dose = $\frac{(210 \text{ mg/kg}) (50 \text{ mg/day}) (0.68) (1 \text{ kg}/10^6 \text{ mg})}{70 \text{ kg}} = 1 \times 10^{-4}$ mg/kg/day

Chronic MRL = 3×10^{-4} mg/kg/day

If future redevelopment or changes in land use result in subsurface activities on the OCRR site, it is possible that construction and utility workers might have contact with onsite subsurface soils contaminated with arsenic, PCBs (less than 50 ppm), and PAHs during future construction activities. However, given the level of site characterization, such activities are likely to be undertaken using proper health and safety precautions to minimize potential exposure, or additional site cleanup could be undertaken.

Regarding adjacent residential properties, lead was detected above the action level at an undeveloped property and was not detected above the action level in soil samples collected from the other residential properties along Spring Street. Since lead exposure is of most concern for 0–6 year-olds, who probably did not trespass on the undeveloped property because they were very young, it is unlikely that children in this age group would have been exposed to lead above the action level.

For children at adjacent residences who might have inadvertently ingested the maximum concentration of arsenic detected in residential backyards for 7 days a week, for 50 weeks, over 18 years, no increased cancer risk would be expected[9]. The same exposure conditions would also be unlikely to result in adverse noncancer health effects[10].

Because contaminated soil on residential properties was replaced with clean soil during EPA removal activities, current and future exposure pathways of residents to soil were eliminated.

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[9] Cancer Effects Exposure Factor = $\frac{(7 \text{ days/week})\ (50 \text{ weeks/year})\ (18 \text{ years})}{(365 \text{ days/year})\ (70 \text{ years})} = 0.25$

Cancer Effects Exposure Dose = $\frac{(58 \text{ mg/kg})\ (200 \text{ mg/day})\ (0.25)\ (1 \text{ kg}/10^6 \text{ mg})}{35 \text{ kg}} = 8 \times 10^{-5}$ mg/kg/day

Cancer Risk = 8×10^{-5} mg/kg/day x 1.5 $(\text{mg/kg/day})^{-1} = 1 \times 10^{-4}$

[10] Noncancer Effects Exposure Factor = $\frac{(7 \text{ days/week})\ (50 \text{ weeks/year})\ (18 \text{ years})}{(18 \text{ years})\ (365 \text{ days/year})} = 0.96$

Noncancer Effects Exposure Dose = $\frac{(58 \text{ mg/kg})\ (200 \text{ mg/day})\ (0.96)\ (1 \text{ kg}/10^6 \text{ mg})}{35 \text{ kg}} = 3 \times 10^{-4}$ mg/kg/day

Chronic MRL = 3×10^{-4} mg/kg/day

B. Exposure to Groundwater

The groundwater wells sampled at the OCRR site are for monitoring purposes only, and no one drinks water from these wells. The surrounding neighborhood is supplied with municipal drinking water. There are no known private drinking water wells in the area (S. McCann, East Bridgewater Water Department, personal communication, 2004). Since groundwater in this area is not being used as a source of drinking water, ingestion of or dermal contact with groundwater is not a completed exposure pathway for residents.

The OCRR site is on the edge of a Zone II wellhead protection area for East Bridgewater Well #5. Under extreme drought conditions (i.e., 180 days with no precipitation to recharge aquifers) groundwater from the site might reach Well #5 and affect water quality. However, these conditions would occur infrequently, if at all. In addition, public water supplies are tested and treated on a routine basis in accordance with state and federal laws (S. McCann, East Bridgewater Water Department, person communication, 2005). Hence, exposure to contaminants in groundwater was eliminated as a possible route of exposure.

C. Exposure to Surface Water/Sediment

It is possible that incidental ingestion and dermal contact with surface water and sediment contaminants in a drainage ditch onsite and immediately adjacent to some Spring Street properties may have occurred in the past for children accessing the site. However, it is important to note that the comparison values used in this evaluation represent a daily drinking water exposure or residential soil exposure. Because a wooded, marshy area separates the drainage ditch from backyard lawns, a child accessing the ditch would have likely been exposed less frequently and to significantly less contaminated surface water and sediment through incidental ingestion and dermal contact.

EPA has withdrawn its cancer slope factor for TCE pending further evaluation and, therefore, the increased cancer risks from exposure to TCE detected in surface water cannot be quantified. However, assuming that a child had dermal contact with surface water for 1 hour/day, 2 days/week, 26 weeks/year, for 10 years, using their hands, arms, and legs, and they ingested

about a mouthful (50 mL) per day of surface water with the maximum concentration of TCE detected (59.7 ppb) for the same amount of time, the estimated exposure dose would be

3.2×10^{-5} mg/kg/day[11]. This estimated exposure would be 3×10^{7} times lower than the Lowest Observed Adverse Effect Level (LOAEL) for cancer observed in scientific studies of rats exposed to TCE (ATSDR 2003c)[12]. For noncancer effects related to inadvertent ingestion and dermal contact with surface water containing the maximum concentration of TCE under the same exposure conditions described above, the estimated exposure would be below ATSDR's MRL, and noncancer health effects in a child would not be expected[13].

Under the same assumptions as used to estimate exposure for children inadvertently ingesting surface soil when accessing the site, children ingesting drainage ditch sediment contaminants

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[11] Cancer Effects Exposure Dose (Ingestion) = $\frac{\text{(max. contaminant conc.) (water ingestion rate) (exposure factor)}}{\text{body weight}}$

$= \frac{\text{(0.0579 mg/L) (0.05 L/day) (0.02)}}{\text{35 kg}} = 1.7 \times 10^{-6}$ mg/kg/day

Cancer Effects Exposure Dose (Dermal) = $\frac{\text{(max. contam. conc.) (permeability constant) (exposed body surface area) (exposure time) (1L)}}{\text{(body weight) (1,000 cm}^3\text{)}}$

$= \frac{\text{(0.0597 mg/L) (0.21 cm/hr}^a\text{) (4,310 cm}^2\text{) (0.02 hr/day) (1 L)}}{\text{(35 kg) (1,000 cm}^3\text{)}} = 3 \times 10^{-5}$ mg/kg/day

[a] ATSDR 2003c

Cancer Effects Exposure Dose (Total) = Cancer Effects Exposure Dose (Ingestion) + Cancer Effects Exposure Dose (Dermal)

$= 1.7 \times 10^{-6}$ mg/kg/day + 3×10^{-5} mg/kg/day = 3.2×10^{-5} mg/kg/day

[12] Margin of Exposure = LOAEL/Cancer Effects Exposure Dose (Total)

$= \frac{\text{1000 mg/kg/day}}{3.2 \times 10^{-5} \text{ mg/kg/day}} = 3 \times 10^{7}$

[13] Noncancer Effects Exposure Dose (Ingestion) = $\frac{\text{(max. contaminant conc.) (water ingestion rate) (exposure factor)}}{\text{body weight}}$

$= \frac{\text{(0.0579 mg/L) (0.05 L/day) (0.14)}}{\text{35 kg}} = 1.2 \times 10^{-5}$ mg/kg/day

Total Noncancer Effects Exposure Dose = 1.2×10^{-5} mg/kg/day + 3×10^{-5} mg/kg/day = 4×10^{-5} mg/kg/day

Acute MRL = 0.2 mg/kg/day

that were detected at lower levels than surface soil contaminants would not likely experience adverse health effects or increased cancer risk.

Because the drainage ditch was remediated during site clean-up, present and future exposures to contaminated surface water and sediment were eliminated from further consideration.

D. Exposure to Indoor Air

The 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. The shallow groundwater flow direction at the OCRR site is to the south-southwest, in the general direction of some Spring Street homes. To evaluate the possibility of a potential vapor infiltration exposure pathway, the maximum concentrations of VOCs detected in shallow groundwater at the site were compared with GW-2 groundwater cleanup standards set by MDEP. GW-2 standards are intended for use at contaminated sites where shallow groundwater is considered to be a potential source of volatile oil and/or hazardous material to indoor air. They are applicable when contaminated groundwater is located within 30 feet of an existing occupied building and when the average annual depth to groundwater in that area is 15 feet or less. For houses and buildings in the vicinity of the OCRR site, application of the GW-2 standards represents a conservative approach since offsite downgradient concentrations are expected to be lower than those detected at the site.

Environmental data from shallow groundwater (i.e., 15 feet or less) included results from monitoring wells on the Precise Engineering property. Six wells were sampled in 1988, three of which were sampled again in 1996. Six new wells were sampled in 1997. In 1988, trans-DCE was detected at a maximum concentration of 1,020 ppb, which is below the GW-2 standard (20,000 ppb) for indoor air exposure. Also in 1988, vinyl chloride was detected in three of six wells above GW-2 standards (2 ppb) for indoor air exposure to vinyl chloride. The maximum concentration of vinyl chloride in 1988 was 1,170 ppb in monitoring well OW-3. OW-3 was located in a southern area of the OCRR site about 20 feet west of the southwest corner of the Precise Engineering building, just north of the drainage ditch. Vinyl chloride was not detected in OW-3 or in two other existing wells that were resampled in 1996. Vinyl chloride was not detected in any new monitoring well in 1997; however, none of the new 1997 wells was located

downgradient (to the south-southwest) of monitoring well OW-3 where vinyl chloride was detected at 1,170 ppb in 1988. Therefore, no data on vinyl chloride concentrations in groundwater downgradient from well OW-3 exist. Thus, it was not possible based on available data to assess potential exposure opportunities to vinyl chloride (through volatilization from shallow groundwater to indoor air) for residents living downgradient from the OCRR site. The MDEP reported that indoor air exposures to vinyl chloride for residents in downgradient Spring Street homes were unlikely (G. Martin, MDEP, personal communication, 2005). When surface water was present in the seasonal drainage ditch that is located between Precise Engineering and downgradient homes, onsite groundwater likely discharged to the ditch and possible contaminants, including vinyl chloride, would have volatilized and dispersed (G. Martin, MDEP, personal communication, 2005). Vinyl chloride was detected in drainage ditch surface water in 1988 at a level of 14.5 ppb and was not detected upstream, which suggests that contaminated onsite groundwater was likely discharging to the ditch (SEA Consultants 1998). Therefore, MDEP reported that vinyl chloride is unlikely to be present in shallow groundwater near homes at a concentration of concern for possible indoor air exposure. However, because there were insufficient environmental data available to evaluate this pathway, the pattern of liver cancer diagnoses, which is a cancer type associated with exposure to vinyl chloride, was evaluated for Spring Street residents.

VII. ANALYSIS OF CANCER INCIDENCE

A. Methods for Analyzing Cancer Incidence

1. Case Identification/Definition

Cancer incidence data, reports of new cancer diagnoses, for the years 1982–2000 were obtained for the town of East Bridgewater from the MCR, a division of the Center for Health Information, Statistics, Research, and Evaluation within MDPH. Eight cancers types were evaluated in this investigation and include leukemia, non-Hodgkin's lymphoma (NHL), and cancers of the bladder, brain and central nervous system (CNS), breast, kidney, liver, and lung and bronchus. [Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O system). See Appendix A for the incidence coding definitions used in this report for these cancer types.] These cancer types were selected for evaluation based on

potential associations with contaminants of concern identified at the OCRR site, as well as resident concerns over suspected elevations of some cancer types in the surrounding neighborhoods, including the Ashley Drive area. For example, because trespassers and workers could have been exposed to soil contamination (e.g. arsenic, lead, and PCBs) in the past, cancer types with known or possible associations with these contaminants of concern at the OCRR site were selected for evaluation.

Only cases reported to the MCR as a primary cancer for one of the eight cancer types and diagnosed among a resident of East Bridgewater 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. Each case was matched to the corresponding census tract.

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 6 months of the date of diagnosis (M.G.L. c.111s.111B). The 19-year period, 1982–2000, constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis[14].

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

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. Duplicate cases are additional reports of the same primary site cancer

[14] The data summarized in this report are drawn from data entered on MCR computer files before March 10, 2004. 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.

case. The data in this report have been controlled for duplicate cases by excluding them from the analyses. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital/diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in 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 East Bridgewater, one duplicate report was identified during the years 1982–2000 and excluded from the analyses.

2. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer cases occurred in East Bridgewater, cancer incidence data were tabulated by gender according to 18 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 1982–2000 for each of the eight primary cancer types for East Bridgewater as a whole and the census tracts that comprise the town. SIRs were also calculated for three smaller time periods, 1982–1987, 1988–1993, and 1994–2000, in order to evaluate patterns or trends in cancer incidence over time.

In order to calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 United States census data for East Bridgewater (U.S. DOC 1980, 1990, 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990, and 1997). 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[15].

[15] 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.

Because accurate age group and gender specific population data are required to calculate SIRs, the census tract (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 United States 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. DOC 1990).

According to the Unites States Census, the town of East Bridgewater is subdivided into three census tracts (i.e., CTs 5231, 5232.01, and 5232.02) (U.S. DOC 2000). As stated previously, the OCRR site and Alloy Castings Company are located in East Bridgewater CT 5231 (see Figure 1).

3. Interpretation of a Standardized Incidence Ratio (SIR)

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as a larger comparison 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.

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 population being evaluated 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 bladder, kidney, and lung and bronchus 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 East Bridgewater residents diagnosed with any of the eight cancer types included in this report. However, information about personal risk factors that might include 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 them 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 within a census tract (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cases in East Bridgewater. In instances where the address information from the MCR was incomplete, that is did not include specific streets or street numbers, all cases were able to be mapped using telephone books and town residential lists issued within 2 years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. [Note: MDPH is bound by law not to reveal the name or identifying information of an individual diagnosed with cancer whose case is reported to the MCR.]

B. Results of Cancer Incidence Analysis

The following section presents cancer incidence rates for the 19-year time period, 1982–2000, for East Bridgewater and the census tracts that divide the town, with a particular focus on CT 5231, where the OCRR site and Alloy Castings are located. To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–2000. Table 6a through Table 6d summarize cancer incidence data for the town of East Bridgewater as a whole. Table 7a through Table 9d summarize cancer incidence data for East Bridgewater CTs 5231, 5232.01 and 5232.02. 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 excess numbers of cancer cases were occurring.

1. Cancer Incidence in East Bridgewater

The eight cancer types evaluated in this report generally occurred approximately at or near expected rates in the town of East Bridgewater as a whole during the 19-year time period, 1982–2000, as well as smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–2000) (see Table 6a through Table 6d). One statistically significant elevation was observed (i.e., female leukemia during the middle time period, 1988–1993) and is discussed later in this report. No other statistically significant elevations were observed in the town of East Bridgewater as a whole.

Overall, cancers of the bladder, breast, kidney, and lung and bronchus all occurred at about the rates expected. For example, there were 29 diagnoses of bladder cancer during the time period, 1982–2000, whereas approximately 30 diagnoses were expected (SIR = 97). During the earliest time period, 1982–1987, there were 11 diagnoses of bladder cancer compared to 9.2 expected. During 1988–1993, bladder cancer occurred less often than expected (6 diagnoses observed vs. 9.2 expected), though the difference was not statistically significant. Finally, during the most recent time period, 1994–2000, bladder cancer occurred at approximately the expected rate (12 diagnoses observed vs. 11.8 expected). Residents of East Bridgewater experienced breast cancer at about the rate expected during 1982–2000 (139 diagnoses observed vs. 135.1 expected, SIR = 103) and during each of the three smaller time periods evaluated. Kidney cancer occurred at approximately the expected rate in East Bridgewater during the overall time period 1982–2000 (18 diagnoses observed vs. 19.2 expected, SIR = 94). The incidence of kidney cancer during the first time period, 1982–1987, was less than expected (1 diagnosis observed vs. 4.2 expected) and at about the rate expected during the two later time periods.

The incidences of lung and bronchus cancer among males and females combined (123 diagnoses observed vs. 115.6 expected, SIR = 106) and among males (75 diagnoses observed vs. 68.9 expected, SIR = 109) were slightly higher than expected from 1982–2000. However, the elevations were not statistically significant. Females were diagnosed with lung and bronchus cancer at about the rate expected during 1982–2000 (48 diagnoses observed vs. 46.7 expected; SIR = 103).

Brain and CNS cancer and liver cancer occurred less often than expected during the 1982–2000 time period. There were nine diagnoses of brain and CNS cancer during 1982–2000, whereas approximately 15 diagnoses were expected (SIR = 58). Fewer brain and CNS cancer diagnoses were also observed than were expected during each of the three smaller time periods evaluated. Three diagnoses of liver cancer were observed in East Bridgewater during 1982–2000 versus about five expected. When examined over time, liver cancer occurred about as expected in all three smaller time periods.

Leukemia occurred more often than expected in East Bridgewater during the 19-year time period 1982–2000 (22 diagnoses observed vs. 17.3 expected, SIR = 127). However, the elevation was

not statistically significant (95% CI = 80–193). This elevation was largely due to a statistically significant elevation in the incidence of leukemia among females during the middle time period 1988–1993 (6 diagnoses observed vs. 2.1 expected, SIR = 289, 95% CI = 105–630). Although this result was statistically significant, the wide 95% confidence interval indicates that the SIR is unstable. Leukemia has four main subtypes and several rare types and, for that reason, is generally evaluated as separate diseases because each subtype has different risk factors. An evaluation of the incidence of leukemia by subtype revealed that, among the six females diagnosed during 1988–1993, there were five leukemia subtypes represented. During both the earlier and later time periods, leukemia among females occurred about as expected. During all three time periods, the incidence of leukemia among males occurred at the expected rate.

NHL occurred more often than expected in East Bridgewater during 1982–2000 (36 diagnoses observed vs. 30.0 expected; SIR = 121). The elevation was largely attributed to NHL incidence among males (22 diagnoses observed vs. 16.0 expected, SIR = 138); however, neither elevation was statistically significant. NHL among males was also elevated during the two earlier time periods when the observed number of males diagnosed with NHL was about three more than expected for each time period. Again, these elevations are not statistically significant. The occurrence of NHL among males during the latest time period (1994–2000) was about as expected (7 diagnoses observed vs. 7.8 expected).

2. Cancer Incidence in East Bridgewater Census Tracts

Of the eight cancer types evaluated in this report, none were statistically significantly elevated in East Bridgewater CT 5231, where the OCRR site and Alloy Castings Company are located, for any of the time periods evaluated. Five cancer types (brain and CNS, breast, kidney, and liver cancer and leukemia) occurred approximately equal to or less often than expected during 1982–2000 among males and females combined (Table 7a). When examined by the three smaller time periods, these five cancers occurred about as or less than expected for each gender. Tables 7b-7d show the observed and expected rates for each of the eight cancer types.

In CT 5231, nine individuals were diagnosed with bladder cancer compared to 7.3 expected (SIR = 123). This elevation was not statistically significant. The incidence of lung cancer among males was elevated in East Bridgewater CT 5231 during 1982–2000 (23 diagnoses observed vs.

16.9 expected, SIR = 136), but again the elevation was not statistically significant. Lung cancer diagnosed among females during 1982–2000 occurred slightly less often than expected (9 diagnoses observed vs. 10.9 expected, SIR = 82).

NHL in East Bridgewater CT 5231 was elevated during 1982–2000 among females (6 diagnoses observed vs. 3.2 expected; SIR = 185); however, this elevation was not statistically significant. During 1982–2000, NHL occurred at about the rate expected among males in CT 5231 (4 diagnoses observed vs. 3.8 expected).

Of the eight cancer types evaluated in this report, none were statistically significantly elevated in East Bridgewater CT 5232.01 for any of the time periods examined (Tables 8a-8d). All eight cancer types occurred approximately equal to or less often than expected during 1982–2000 among males and females combined (Table 8a). For example, there were 30 diagnoses of breast cancer vs. 32.4 expected (SIR = 92). When examined by the three smaller time periods, seven of the cancer types (bladder, brain and CNS, breast, kidney, and liver cancer, leukemia, and NHL) occurred about as or less than expected for each gender. Any elevations noted were generally based on about one or two excess cases.

Lung and bronchus cancer was elevated for the time period 1982–1987 (12 diagnoses observed vs. 7.3 expected, SIR = 166). Among males in CT 5232.01 there were nine diagnoses observed versus 4.7 expected (SIR = 191). During this time period, lung and bronchus cancer occurred at about the rate expected among females in CT 5232.01 (3 diagnoses vs. 2.5 expected). During the latter two time periods, lung and bronchus cancer occurred less than expected among males (i.e., 3 diagnoses observed vs. 5.1 expected during 1988–1993).

In East Bridgewater CT 5232.02, four cancer types (bladder, brain and CNS, liver, lung and bronchus cancer) occurred approximately equal to or less often than expected during 1982–2000 among males and females combined (Table 9a). When examined by the three smaller time periods, these four cancers occurred about as or less than expected for each gender. A slight elevation in lung and bronchus cancer diagnoses among males (20 observed vs. 15.7 expected, SIR = 127) occurred in the time period 1994–2000; however, the elevation was not statistically significant.

The incidence of breast cancer was slightly elevated in East Bridgewater CT 5232.02 during 1982–2000 (80 diagnoses observed vs. 71.8 expected, SIR = 111), but the elevation was not statistically significant. Breast cancer occurred as expected among females during 1982–1987.

There were 14 diagnoses of kidney cancer compared to 10.0 expected (SIR = 139) in CT 5232.02 during 1982–2000. This elevation was based on approximately two additional diagnoses over the expected number for males and females when evaluated separately and was not statistically significant. When examined by smaller time periods, kidney cancer occurred about as expected during the two earlier time periods and was elevated during 1994–2000 (9 diagnoses observed vs. 5.1 expected), but the elevation was not statistically significant.

The incidence of leukemia was elevated in East Bridgewater CT 5232.02 during 1982–2000 (13 diagnoses observed vs. 9.0 expected, SIR = 144), but the elevation was not statistically significant. Seven females were diagnosed, while approximately four would be expected. There were five different leukemia subtypes reported among these females. Leukemia diagnosed among males during 1982–2000 occurred about as expected (6 diagnoses observed vs. 5.1 expected, SIR = 118).

NHL in East Bridgewater CT 5232.02 was elevated among males during 1982–2000 (15 diagnoses observed vs. 8.4 expected; SIR = 179). The elevation was nearly statistically significant (95% CI = 100–296) and largely due to a statistically significant elevation in the incidence of NHL among males in the middle time period, 1988–1993 (7 diagnoses observed vs. 2.6 expected, SIR = 272, 95% CI = 109–560). Although NHL incidence among males in 1988–1993 was statistically significant, the wide 95% confidence interval indicates that the SIR is unstable. During the middle time period, 1988–1993, NHL occurred less often than expected among females in CT 5232.02 (1 diagnosis observed vs. 2.2 expected). NHL diagnoses occurred at about the expected rate for both males and females during the other two time periods, 1982–1987 and 1994–2000.

C. 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 East Bridgewater. Information for each of the eight cancer types 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, and therefore, it was not possible to evaluate the role these types of risk factors may have played in the incidence of cancer in East Bridgewater. For detailed information regarding risk factors associated with the cancer types evaluated in this report, please refer to Appendix B.

Age and gender are risk factors in many types of cancers, including all eight types evaluated in this report. Tobacco use is also a known or suggested causal risk factor in several types of cancer, including bladder cancer, kidney cancer, and lung and bronchus 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 East Bridgewater. 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 exposures specific to certain occupations and an increase in the incidence of bladder cancer, brain and CNS cancer, kidney cancer, leukemia, liver cancer, lung and bronchus cancer, and NHL. 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 East Bridgewater. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. Further, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

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

1. *Bladder Cancer*

The American Cancer Society estimates that bladder cancer will affect 61,420 people in the Unites States in 2006, accounting for 6% of all cancers diagnosed in the United States among men and 2% among women (ACS 2006). White males have the highest prevalence of bladder cancer across all racial groups. A male to female ratio of four to one has been observed among whites, while a slightly lower male to female ratio of three to one has been observed among most other racial groups. Further, the occurrence of bladder cancer rises with increasing age. The mean age at diagnosis in Massachusetts for the years 1982–2000 was 70 years.

Bladder cancer is strongly associated with a history of cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS 2000a). Tobacco use is associated with approximately 25-60% of all bladder cancers (Johansson and Cohen 1997).

Studies have 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 United States (Johansson and Cohen 1997). Occupational exposure to aromatic amines, such as benzidine and 2-naphthylamine, increases the risk of bladder cancer (ACS 2000a). 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 2000a, 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 2000a).

a) Age and Gender

A review of individuals diagnosed with bladder cancer in East Bridgewater from 1982–2000 revealed that the majority of diagnoses in the town were male (69%, n = 20). Males comprised 72% of bladder cancers statewide for this time period. The mean age at diagnosis in East Bridgewater was 66 years, which is consistent with statewide bladder cancer incidence.

b) Tobacco Use

Of the 29 individuals in East Bridgewater who were diagnosed with bladder cancer during the years 1982–2000, two-thirds of those with known smoking history were current/former smokers (n = 12), which is nearly identical to the 67% of individuals diagnosed with bladder cancer in Massachusetts during 1982–2000 with known smoking history who were current/former smokers.

c) Occupation

Review of occupation for individuals diagnosed with bladder cancer in East Bridgewater revealed that at least two individuals might have worked at a job in which occupational exposures potentially related to the development of bladder cancer 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, occupation was reported as retired or unknown for many of these individuals (38%, n = 11).

2. Brain and Central Nervous System Cancer

The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with brain and CNS cancer in 2006 (ACS 2006). According to epidemiological literature, brain tumor incidence (cancerous and non-cancerous) declines after a peak in childhood (under 10 years of age), increases from age 25 to 75, and levels off after age 75 (Preston-Martin and Mack 1996). Certain types of brain tumors are more likely to develop in children and others are more typically seen in adults (Black 1991, NCI 1996). Brain and spinal

cord cancers account for over 20% of all cancer types diagnosed among children 0-14 years of age (ACS 2004).

Various studies on worker exposure to vinyl chloride and chemicals in the petrochemical industry have had conflicting results as to the association between these chemicals and the development of brain tumors. Studies investigating the possible association between parental occupational exposures (e.g., paper or pulp mill, aircraft, rubber, and electric workers) and the onset of brain tumors (cancerous and non-cancerous) in their children have also provided inconsistent results (Preston-Martin and Mack 1996).

a) Age and Gender

From 1982 to 2000, males and females statewide were diagnosed with brain and CNS cancer about equally. A review of gender patterns among brain and CNS cancer cases in East Bridgewater revealed that more males than females were diagnosed with brain and CNS cancer during that time period (6 males and 3 females). However, because of the relatively small number of individuals diagnosed with brain and CNS cancer in East Bridgewater (nine individuals during the entire time period of 1982–2000), the difference in gender distribution is likely due to chance. The age at diagnosis for individuals with brain and CNS cancer was consistent with the pattern expected based on the scientific literature for this cancer type. All but one of the individuals diagnosed with brain or CNS cancer were diagnosed after age 25. There was one child diagnosed with brain or CNS cancer. This child lived in a different census tract and more than 1 mile away from both the OCRR site and Alloy Castings Company.

b) Occupation

Among the eight adults in East Bridgewater diagnosed with brain or CNS cancer, an occupation was reported for four individuals. None of these four individuals reported occupations where exposures to the chemicals listed above were likely to have occurred, based on the available information.

3. *Breast Cancer*

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, approximately 77% of new cases of breast cancer occur in women over age 50 (ACS 1999). Breast cancer incidence and age have been shown to be related where the incidence increases from age 35 to 45, increasing 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

Breast cancer occurred at about the rate expected in the town of East Bridgewater from 1982-2000 (139 diagnoses observed vs. 135.1 expected, SIR=103). The majority (99%, n = 137) of individuals diagnosed with breast cancer in the town were women. Two men were diagnosed with breast cancer in East Bridgewater during this time. This gender pattern is consistent with statewide trends. The pattern of increasing breast cancer incidence with increasing age was observed among women diagnosed with breast cancer in East Bridgewater. Approximately 70% of women diagnosed with breast cancer during the time period evaluated were over age 50, which is consistent with national trends.

4. *Kidney and Renal Pelvis 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). Epidemiological studies have shown that incidence rates of kidney cancer rise with increasing age before reaching a plateau at approximately age 70 (McLaughlin et al. 1996). 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 those 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 TCE, may increase the risk of this cancer (ACS 2001a). More recently, renal cell carcinoma, 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).

a) Age and Gender

The incidence of kidney cancer in East Bridgewater generally increased with increasing age. The average age of individuals diagnosed with kidney cancer in East Bridgewater during 1982–2000 was 51 years, while the state mean was 64 years. Fifty-six percent (n = 10) of individuals diagnosed were over the age of 50 at the time of diagnosis, which is consistent with the

literature. More males (n = 10) than females (n = 8) were diagnosed with kidney cancer in East Bridgewater, which is consistent with state and national trends.

b) Tobacco Use

Of the 18 individuals diagnosed with kidney cancer in East Bridgewater during 1982–2000, 73% of those with known smoking history were current/former smokers (n = 11). Of individuals diagnosed with kidney cancer in Massachusetts during 1982–2000 with known smoking history, 57% were current/former smokers.

c) Occupation

Review of occupation for individuals diagnosed with kidney cancer in East Bridgewater revealed that one individual might have worked a job in which occupational exposures potentially related to the development of kidney cancer may have been possible. However, information regarding specific job duties that could help to further define exposure potential for this individual was not available. Occupations reported for the remaining individuals are not likely to be related to an increased risk of this cancer type. However, occupation was reported as retired, unknown, or housewife for 33% of these individuals (n = 6).

5. Leukemia

In 2006, leukemia is expected to affect approximately 35,070 individuals (20,000 males and 15,070 females) in the United States, resulting in 22,280 deaths (ACS 2006). In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, representing more than 2% of all cancer diagnoses (ACS 2006). 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 2003a).

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 1982–2000, 34% of all leukemia cases were AML, 26% were CLL, 13% were ALL, 11% were CML, and 16% 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. Exposures to ionizing radiation, 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.

a) Age and Gender

The average age of individuals diagnosed with leukemia in East Bridgewater was 59 years, which mirrors the statewide experience. Seventy-three percent (n = 16) were age 50 or older at the time of diagnosis. One diagnosis occurred in a child, which is less than the number expected (2.5 cases expected). A town-wide elevation in the incidence of leukemia in females during the middle time period, 1988–1993, was observed. Leukemia occurred about as expected among females during the other two time periods, 1982–1987 and 1994–2000. The incidence of leukemia among males was about as expected for all three smaller time periods.

b) Histology

The four main leukemia subtypes have different risk factors suspected to be associated with their development and generally occur with different frequency among adults and children. Of the 22 individuals diagnosed with leukemia in East Bridgewater during 1982–2000, 23% were

diagnosed with AML subtype, 27% were diagnosed with CLL, 14% were diagnosed with ALL, 9% were diagnosed with CML, and 27% were not specified or were diagnosed with other types of leukemia. This distribution is somewhat similar to that seen statewide. The one child diagnosed with leukemia in East Bridgewater was diagnosed with the ALL subtype, the most common subtype among children.

Of the six females diagnosed with leukemia during 1988–1993, there were two cases of ALL, one case of CML, one case of hairy cell leukemia, one case of adult T-cell leukemia, and one case of an unspecified myeloid leukemia. As previously mentioned, these leukemia cell types are different and have varied risk factors.

c) Occupation

Review of occupation for individuals diagnosed with leukemia in East Bridgewater revealed that at least one individual may have worked a job 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, occupation was reported as retired or unknown for many of these individuals (32%, n = 7).

6. Liver Cancer

An estimated 18,510 people in the United States (12,600 men and 5,910 women) will be diagnosed with liver 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, 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) or hepatitis C virus (HCV) is the most significant risk factor for developing liver cancer (ACS 2001b), epidemiological 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 2001).

a) Age and Gender

The three individuals diagnosed with liver cancer during 1982–2000 had a mean age of 52 years. For the most part, this observation was consistent with trends for this cancer type in the general population. Two of the three diagnoses were males.

b) Occupation

The one individual diagnosed with liver cancer in East Bridgewater that reported an occupation was employed in an occupation not likely to be related to an increased risk of developing liver cancer. Occupation was unknown or reported as "at home" for two individuals.

7. Lung and Bronchus Cancer

The American Cancer Society estimates that lung cancer will be diagnosed in 174,470 people in the United States in 2006, accounting for about 12% of all cancers (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 2000b). According to epidemiological literature, the incidence of lung cancer increases sharply with age and peaks around approximately age 60 to 70. Only two percent 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 sub-divided into three types: adenocarcinoma, squamous cell carcinoma, and large-cell undifferentiated carcinoma. The different types of lung cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung cancers are adenocarcinomas, 25-30% are

squamous cell carcinomas, 20% are small cell cancers, and 10-15% of cases are large cell carcinomas (ACS 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 second hand 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 one's smoking habits (e.g., duration of habit, amount smoked, tar yield of cigarette, and filter type). Smoking cessation decreases the elevated risk by about 50%; however, former smokers still carry a greater risk of developing lung cancer than those who have never smoked.

Several occupational exposures have been identified as playing a role in the development of lung cancer. For example, workplace exposure to asbestos is an established risk factor for this disease (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).

a) Age and Gender

Among the 123 individuals diagnosed with lung and bronchus cancer in East Bridgewater during 1982–2000, the average age at diagnosis was 67 years. None of these individuals were under the age of 40 at the time of diagnosis. The incidence of lung and bronchus cancer occurred about as expected for both males and females.

b) Histology

Of the 100 lung and bronchus cancer diagnoses in East Bridgewater with a specific histology classification, 41% were diagnosed as adenocarcinomas, 27% were squamous cell carcinomas, 27% 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.

c) Tobacco Use

Of the 123 individuals diagnosed with lung and bronchus cancer in East Bridgewater during 1982–2000, 95% of those with known smoking history were current/former smokers (n = 106). Of individuals diagnosed with lung and bronchus cancer in Massachusetts during 1982–2000 with known smoking history, 92% were current/former smokers.

d) Occupation

Many individuals diagnosed with lung and bronchus cancer in East Bridgewater did not indicate working in jobs likely to have played a role in their cancer diagnosis. However, 39 individuals (32%) reported either a history of asbestos exposure or occupations where exposures to asbestos or other chemical compounds thought to be associated with lung and bronchus cancer may have been possible. Occupation was reported as retired, unknown, or at home for nearly half (43%, n = 53) of the individuals.

8. Non-Hodgkin's Lymphoma

NHL can occur at all ages, however, the average age at diagnosis is in the early 60s and the incidence of this disease generally increases with age. This disease is more common in men than in women (ACS 2003a). The American Cancer Society estimates that approximately 58,870 Americans will be diagnosed with NHL in 2006, making it the sixth most common cancer in the United States among both men and women, excluding non-melanoma skin cancers (ACS 2006). Although the primary factors related to the development of NHL include conditions that suppress the immune system and viral infections, certain occupational exposures 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 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 2003b), 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

The average age at diagnosis for individuals diagnosed with NHL in East Bridgewater during 1982–2000 was 59 years, which is generally consistent with that seen in the general population. Sixty-one percent (n = 22) of diagnoses of NHL were males, which is similar to trends in the general population. The elevation in NHL incidence among males in CT 5232.02 during the middle time period 1988–1993 was influenced by about four excess cases. NHL occurred about as expected among males during the other two time periods, 1982–1987 and 1994–2000, in CT 5232.02. The incidence of NHL among females in CT 5232.02 was less than or about as expected for all three smaller time periods.

b) Occupation

Review of occupational information for individuals diagnosed with NHL in East Bridgewater revealed that at least one individual might have worked at a job 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 39% of individuals (n = 14). Therefore, it is difficult to assess the role that occupation may have played in the incidence of NHL among residents of East Bridgewater.

D. 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. 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 the OCRR site,

Alloy Castings Company, the Ashley Drive area, or other potential locations of environmental concern (i.e., MDEP 21E hazardous material and oil releases) located in the town of East Bridgewater. 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 the cancer types evaluated in this report, no apparent concentrations of cancer diagnoses (of any type) were observed in any one area of East Bridgewater. There was no geographic pattern observed among the six females diagnosed with leukemia during the middle time period 1988–1993, when a statistically significant elevation in leukemia incidence among females in East Bridgewater was observed. There was no geographic pattern observed among the seven males diagnosed with NHL in CT 5232.02 during the middle time period 1988–1993, when a statistically significant elevation in NHL incidence among males was observed. No apparent geographic concentrations of cancer diagnoses were noted in neighborhood areas surrounding the OCRR site, in the Ashley Drive area, and/or in relation to Alloy Castings.

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 was apparent for any of the eight cancer types evaluated. Any patterns that were observed appeared to be consistent with what would be expected based on the population distribution and areas of higher population density. For example, in East Bridgewater, 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 East Bridgewater 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 town.

VIII. DISCUSSION

At the request of Representative Kathleen Teahan, the East Bridgewater Board of Health, and concerned residents, the MDPH conducted an evaluation of cancer and possible environmental exposures in East Bridgewater. The OCRR site consists of three distinct properties: Precise Engineering, Eastern States Steel, and an old MBTA railroad bed. Historically, various manufacturing operations occupied the Precise Engineering and the Eastern States Steel properties. The inactive MBTA railroad bed was originally part of the Old Colony Railroad. The EPA performed removal activities at the OCRR site in 2001; however, buildings and equipment in disrepair remain onsite, and an 800-foot section of the fencing has fallen down. Community concerns also focused on cancer in the Ashley Drive neighborhood and possible environmental exposures related to Alloy Castings Company, an active aluminum foundry.

As part of this Public Health Assessment, the MDPH evaluated both cancer incidence data for East Bridgewater as a whole and for the census tracts that divide the town and reviewed available environmental information for the OCRR site to determine possible pathways of exposure for nearby residents. In addition, the pattern of cancer was evaluated in neighborhoods within East Bridgewater census tracts to identify any unusual concentrations of cases and to evaluate community concerns about cancer and other locations of community environmental concern, including Alloy Castings Company near the Ashley Drive area.

There are some potential exposure pathways that may have occurred in the past related to the OCRR site. Past exposure to lead, arsenic, and PCBs in onsite surface soil and contaminants in surface water and sediment in the drainage ditch immediately adjacent to some Spring Street residences could have been possible for trespassers and residents accessing the site prior to 2001, when the EPA performed removal activities. In addition, workers could have been exposed to contaminants such as PCBs and arsenic identified in onsite surface soil in the past. However, upon considering conservative exposure doses for trespassers and workers and blood lead level for children, adverse health effects or increased cancer risk due to exposure to past contamination in onsite surface soil, surface water, and sediment were determined to be unlikely.

It is also possible that residents on adjacent Spring Street properties and one West Union residence may have had contact with soil contamination identified in backyard surface soil of

residences abutting the site. However, based on a review of available environmental data, lead was detected above the comparison value on an undeveloped property only. Adverse health effects or increased cancer risk in children due to exposure to past arsenic contamination in residential surface soil was determined to be unlikely after considering conservative exposure doses. Therefore, past exposure for abutting residents to both lead and arsenic is not likely to have resulted in adverse health effects.

The OCRR site is located in a Zone II water supply protection area whose aquifer deposits could be pumped for public drinking water in an extreme drought situation. However, municipal water supplies are tested and treated on a routine basis according to federal and state laws. Therefore, it is not expected that groundwater contaminated with chemicals originating from the OCRR site would be consumed as drinking water.

A potential data gap exists with respect to the detection of vinyl chloride at 1,170 ppb in an onsite groundwater monitoring well in 1988. Although the MDEP reported that it is unlikely that residents downgradient from the OCRR site were or are impacted by vinyl chloride coming from the site, no groundwater data from any downgradient location were available for more recent years. The elevated concentration of vinyl chloride in 1988 occurred in a shallow monitoring well (approximately 15 feet deep). At the OCRR site, the depth to groundwater is about 2 feet below the surface. The direction of groundwater flow in the shallow portion of the aquifer is generally to the south-southwest and appears to flow at a rate of 2 to 5 feet per year (SEA Consultants 1998). Available soil data for this area suggest that subsurface soils are fine-grained silts and clay (SEA Consultants 1998). The nearest residence that is downgradient of the monitoring well where vinyl chloride was detected is approximately 250 feet away on Spring Street. If vinyl chloride is present in groundwater beneath downgradient homes at concentrations that are similar to the concentration detected in 1988, the potential exists for the volatilization of vinyl chloride into indoor air. Because offsite groundwater conditions are unknown, it would be beneficial to address this data gap with additional investigation, for example, hydrogeological modeling or groundwater sampling at downgradient locations.

Vinyl chloride exposure is associated with liver cancer; hence, the MDPH/CEH examined the pattern of liver cancer diagnoses in the Spring Street area, which is downgradient (to the south-

southwest) of the OCRR site. There were no liver cancer diagnoses from 1982 to the present among residents of Spring Street or among anyone in CT 5231 where the OCRR site is located.

Assuming current site conditions, other present and future pathways of exposure were eliminated because the EPA performed removal actions at the OCRR site and nearby residences. Contaminants in surface soil, surface water, and sediment have been removed or are covered by clean soil and are no longer accessible to trespassers or nearby residents. Future exposure of construction or utility workers to contamination in subsurface soil is considered unlikely if construction activities are undertaken using proper health and safety precautions. Also, additional site remediation may be required in order for development to proceed.

The cancers evaluated in this report were selected based on their potential association with contaminants of concern identified at the OCRR site and residents' concerns about cancer in surrounding neighborhoods and the Ashley Drive area. Although conservative estimates of exposure were determined to be unlikely to result in adverse health effects or increased cancer risk, certain cancer types (e.g., bladder, kidney, liver, lung and bronchus) were evaluated because of known or suspected links to contaminants identified at the site. None of the eight cancer types evaluated were statistically significantly elevated in the census tract that contains the OCRR site (CT 5231), and the majority occurred approximately as expected. For example, CT 5231 had no diagnoses of liver cancer, which has been associated with PCB and arsenic exposure. Kidney cancer, which has been linked to arsenic and lead exposure, occurred less often than expected. Bladder cancer and lung and bronchus cancer, which have been associated with arsenic, were slightly elevated; however, the risk factor analysis suggested that tobacco use likely played a major role in diagnoses of these cancer types for some individuals.

In the town of East Bridgewater as a whole, cancer incidence rates for the eight cancer types evaluated during the 19-year time period, 1982–2000, and the three smaller time periods were near expected rates based on cancer incidence in the state of Massachusetts. No cancer type was statistically significantly elevated in CT 5231, where the OCRR site and Alloy Castings are located, for any of the time periods. For East Bridgewater CT 5232.01, there were elevations in some cancer types, but none were statistically significant. Most cancer types occurred approximately at or near expected rates in East Bridgewater CT 5232.02, however, a statistically

significant elevation was observed among males diagnosed with NHL during the middle time period, 1988–1993. The geographic pattern of NHL did not indicate a concentration or an atypical distribution of males diagnosed in this census tract. Also, there was no indication of a temporal trend in elevated NHL among males in this area of East Bridgewater.

Leukemia among females occurred at a rate that was statistically significantly elevated in East Bridgewater as a whole during the middle time period 1988–1993. The geographic pattern of leukemia did not indicate a concentration or an atypical distribution of females diagnosed in the area surrounding the OCRR site or Alloy Castings Company or in any other area of town. Also, as previously discussed, leukemia is classified into four main types of disease that have different risk factors. From 1988 to 1993 in East Bridgewater, a variety of leukemia types were diagnosed, and no pattern of any one type occurred, nor did the evaluation of geographic distribution of cases suggest that environmental factors played a primary role. In addition, there was no trend over time in elevated leukemia among females. For these reasons, it does not appear that the increased occurrence of leukemia observed among females in East Bridgewater from 1988 to 1993 is related to a common factor.

In addition to an evaluation of cancer incidence rates, available risk factor information for those diagnosed with cancer was compared to known or established trends to assess whether any unexpected patterns existed in East Bridgewater for the time period evaluated. In general, cancer trends observed in East Bridgewater were similar to those seen in the general population and in Massachusetts. Data reviewed suggest that smoking likely played some role in the diagnosis of certain cancers (bladder, kidney, and lung and bronchus cancers) among some individuals in East Bridgewater. Also, occupational exposures may have played a role for some individuals in the development of certain cancers, such as leukemia, NHL, and cancers of the bladder, brain and CNS, kidney, and lung and bronchus. However, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in East Bridgewater due to incomplete information for some risk factors (e.g., occupation).

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 related to the incidence of cancer in East Bridgewater as a whole or in the three census tracts that

comprise the town. That is, no unusual concentrations of individuals diagnosed with the eight cancer types evaluated were observed in the vicinity of the OCRR site, Alloy Castings Company, the Ashley Drive neighborhood, or any other area in East Bridgewater. Based on 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 where the OCRR site and Alloy Castings Company are located, in the census tracts that divide the town, or in the town of East Bridgewater as a whole during the 19-year time period, 1982–2000.

IX. CHILD HEALTH CONSIDERATIONS

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

The incidence and patterns of cancer among children in East Bridgewater is discussed in Section VII ("Cancer Incidence in East Bridgewater") of this report. As discussed before, past exposure to onsite and offsite lead, arsenic, and PCB-contaminated surface soil could have been possible for children before the EPA completed removal activities in 2001. In addition, past exposure to contaminants in surface water and sediment in the drainage ditch immediately adjacent to some Spring Street properties may have been possible for children. However, it is unlikely that anyone would have had contact with soil, surface water, or sediment at the OCRR site for a sufficient frequency and duration of time to result in health effects. Present and future exposures are not of concern because contaminated surface soils, surface water, and sediments at the site and adjacent residential properties were removed by the EPA. No other exposures were

identified that would indicate that children are more likely than adults to be impacted by either the OCRR site or Alloy Castings Company.

X. LIMITATIONS

There are several limitations encountered when analyzing environmental data. 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. That is, due to historical and analytical data gaps in the environmental data, this type of evaluation cannot conclude what may have caused any one individual's cancer or other illness, whether the cause is environmental, behavioral, viral, genetic, or a combination of these factors.

This public health assessment is 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 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 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 any one individual's cancer. 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 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 East Bridgewater.

XI. CONCLUSIONS

- Although some of the soil, surface water, and sediment at the OCRR site were contaminated in the past, the remediation activities conducted to date make it unlikely that individuals visiting or residing in neighborhoods adjacent to the site would be exposed presently or in the future.

- Past intermittent exposures to onsite surface soil, surface water, and sediment and offsite surface soil may have been possible for trespassers, former workers, or residents living on adjacent properties. However, based on the contaminant levels detected and the frequency and duration of contact assumed, it is unlikely that potential exposures related to the OCRR site would have resulted in adverse health effects.

- Potential exposure to vinyl chloride volatilized to indoor air may be possible for downgradient Spring Street residents if the contaminant is present in shallow groundwater beneath homes at concentrations similar to those detected in onsite monitoring wells in 1988. The MDEP reported that an investigation of the presence of VOCs in groundwater south of the site is being conducted (G. Martin, MDEP, personal communication, 2007). These data should help to address this potential exposure pathway.

- Trespassers could incur physical harm on the OCRR site because an 800-foot section of the fencing surrounding the property has fallen down and buildings and equipment in disrepair remain onsite.

- If future redevelopment or changes in land use, such as a recreational rail trail, result in subsurface activities on the OCRR site, it is possible that construction and utility workers might have contact with onsite subsurface soil contaminants, such as arsenic, lead, PCBs, and PAHs.

- In general, the eight cancer types evaluated in relation to the OCRR site or due to community concerns occurred near expected rates for the town of East Bridgewater as a whole and for the census tracts that comprise the town during the 19-year time period,

1982–2000. In addition, none of the cancer types that are associated in the scientific literature with contaminants of concern identified at the OCRR site were statistically significantly elevated in CT 5231.

- Leukemia was statistically significantly elevated among females in East Bridgewater as a whole during the middle time period, 1988–1993. However, no apparent geographic concentrations of cases were noted and a variety of different subtypes of leukemia were represented, indicating the occurrence of different diseases.

- Review of the geographic distribution of each of the cancer types in East Bridgewater revealed no apparent spatial patterns at the neighborhood level. Further, no unusual concentrations of individuals diagnosed with cancer were observed in the vicinity of the OCRR site, Alloy Castings Company, in the Ashley Drive area, or in any other area of East Bridgewater.

- Based on the information reviewed in this evaluation, including available environmental data for the OCRR site 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 containing the OCRR site and Alloy Castings Company or in the town of East Bridgewater as a whole during the 19-year time period, 1982–2000.

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. Therefore, based on MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to the cancer types evaluated in this analysis, ATSDR would classify the OCRR site, in the past, present, and future, as posing an Indeterminate Public Health Hazard. This conclusion category was chosen based on a data

gap related to the potential for the presence of vinyl chloride in groundwater to the south of the OCRR site.

XII. RECOMMENDATIONS

- Because there are buildings and equipment in disrepair on the OCRR site, the fencing that surrounds the site should be properly maintained in order to prevent physical harm to potential trespassers.

- The MDEP reported that the Town of East Bridgewater is conducting an assessment of the presence of VOCs in groundwater to the south of the OCRR site (G. Martin, MDEP, personal communication, 2007). Upon request, the MDPH will review new groundwater data related to VOCs as they become available in order to assess potential exposure opportunities to vinyl chloride in indoor air for residents of downgradient Spring Street homes.

- Any future development or changes in land use that result in subsurface activities at the OCRR site should be undertaken using proper health and safety precautions in order to minimize potential exposure to subsurface soil contaminants, or additional site cleanup may be required.

XIII. PUBLIC HEALTH ACTION PLAN

The Public Health Action Plan for East Bridgewater, Massachusetts, contains recommendations for actions to be taken at and in the vicinity of the OCRR site and Alloy Castings Company. The purpose of the Public Health Action Plan is to ensure that this public 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 the ATSDR/MDPH to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by ATSDR/MDPH are as follows:

- Should new groundwater data be generated for VOCs in groundwater to the south of the OCRR site, upon request, the MDPH will assess potential exposure opportunities to vinyl chloride in indoor air upon request.

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

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PREPARER

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

CERTIFICATION

The Public Health Assessment *Evaluation of Cancer, 1982–2000, and Environmental Concerns Related to the Old Colony Railroad Site and Alloy Castings Company in East Bridgewater, Plymouth County, Massachusetts* 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

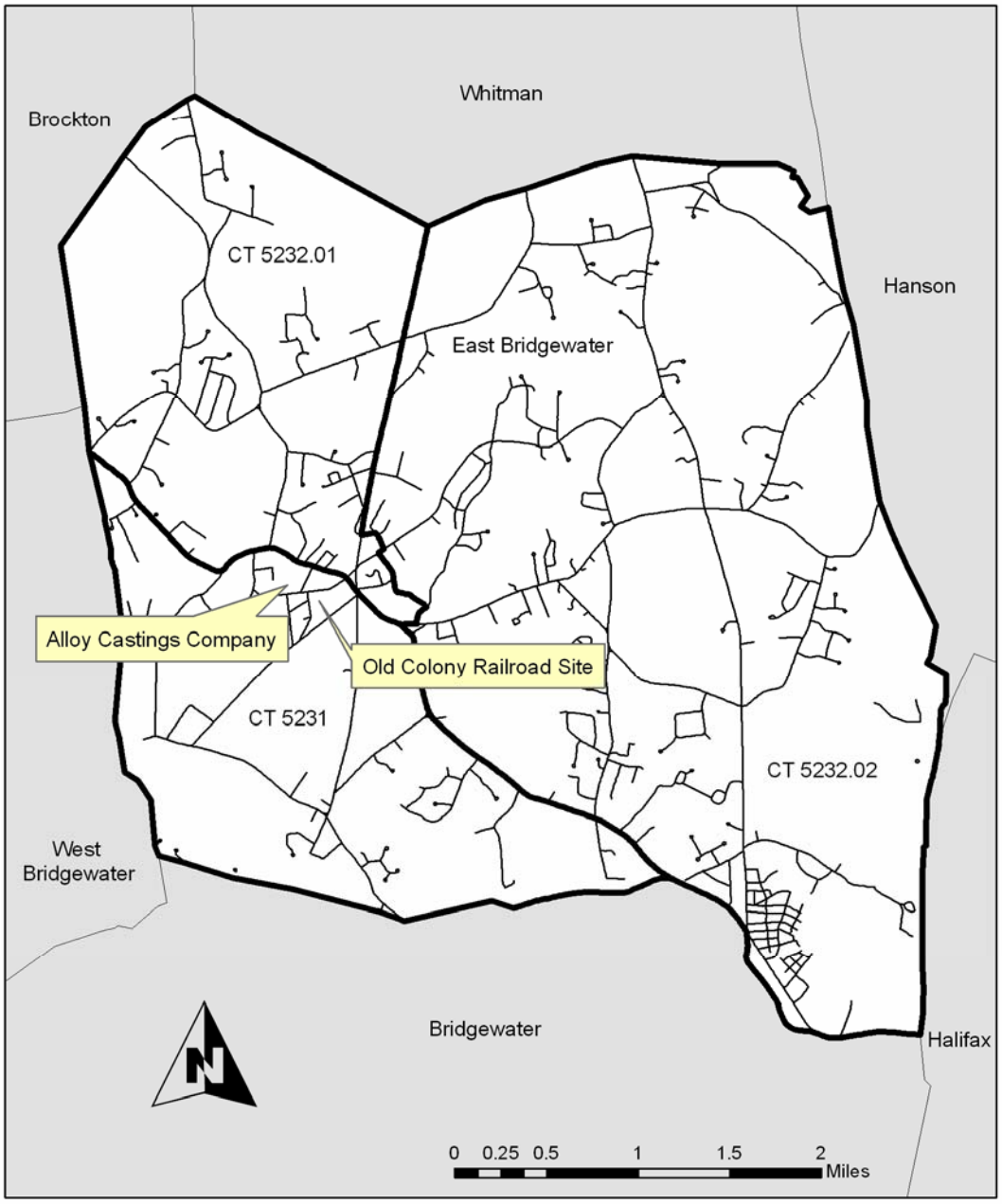
The Division of Health Assessment and Consultation, ATSDR, has reviewed this Health Consultation and concurs with its findings.

_____

Team Lead, CAT SPAB, DHAC

FIGURES

Figure 1
Location of Census Tracts
East Bridgewater, Massachusetts



Center for CEH Environmental Health

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

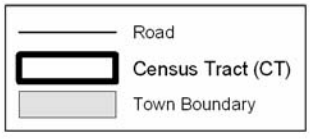


Figure 2
Old Colony Railroad Site and Alloy Castings Company
East Bridgewater, Massachusetts

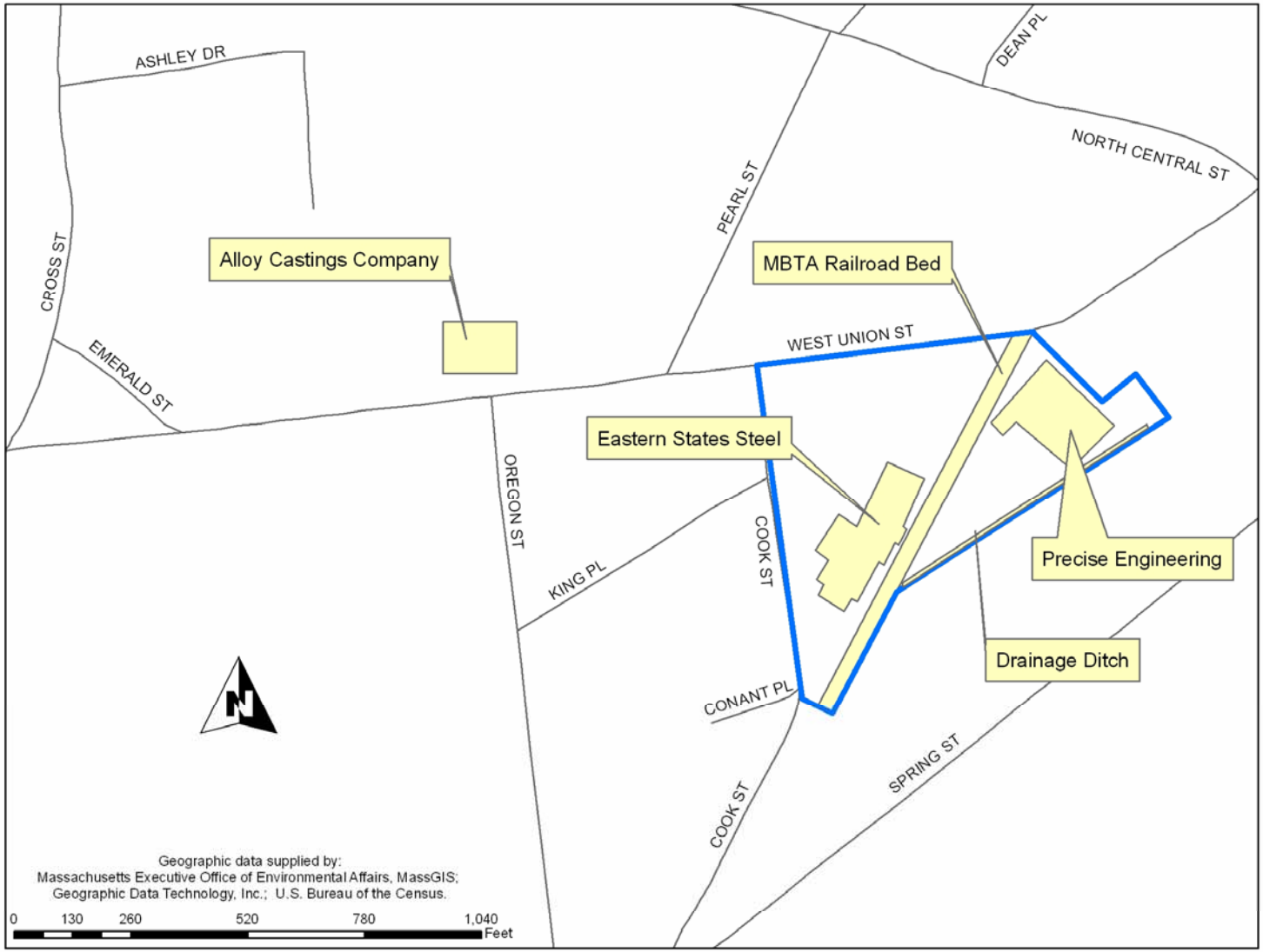
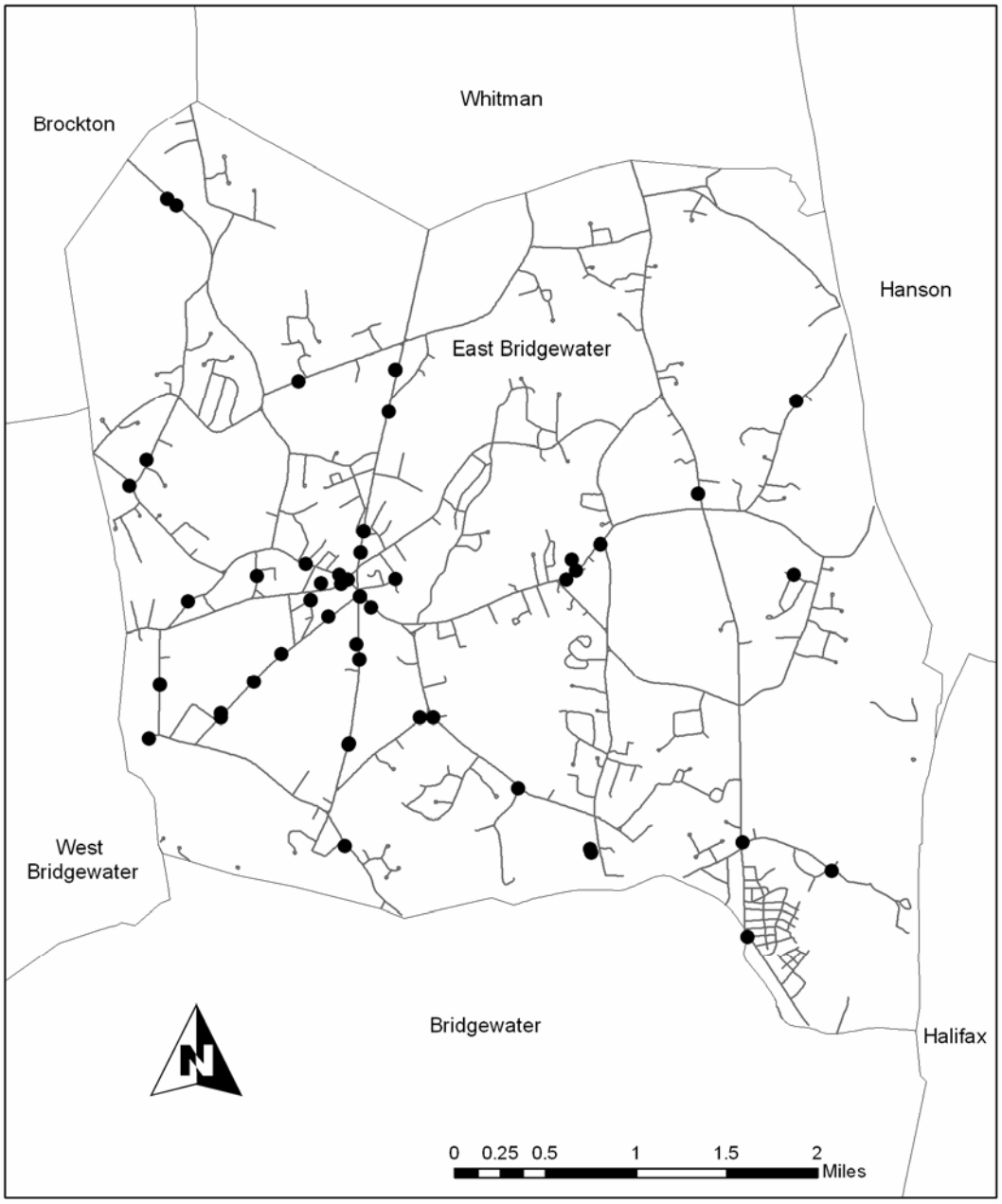


Figure 3
Massachusetts Department of Environmental Protection 21E Hazardous Material and Oil Releases
East Bridgewater, Massachusetts



Center for CEH Environmental Health

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

Road
Town Boundary
MDEP 21E Site

Formatted: Left: 1", Right: 1"

TABLES

Table 1
Massachusetts Department of Environmental Protection 21E Hazardous Material and Oil Releases
East Bridgewater, Massachusetts

MAPPED/NOT MAPPED	SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES	STATUS
MAPPED	S88-0069	NA	RESIDENCE	82 CROSS STREET	02/10/1988	#2 FUEL OIL	AST	UNKNOWN
MAPPED	S88-0810	4-0751	APARTMENT & RETAIL STORES	55 NORTH BEDFORD STREET	UNKNOWN	METALS	UNKNOWN	UNKNOWN
MAPPED	S89-0122	4-0860	CONRAD'S SERV STATION	49 CENTRAL ST	03/01/1989	GASOLINE	UNKNOWN	UNKNOWN
MAPPED	S89-0761	NA	ROADWAY	234 THATCHER ST	10/27/1989	DIESEL FUEL	OTHER SOURCE: FUEL LINE	UNKNOWN
MAPPED	S90-0022	NA	PROPERTY	582 WEST ST	07/15/1989	#2 FUEL OIL	AST	UNKNOWN
MAPPED	S90-0113	NA	E BRIDGEWATER LANDFILL	E BRIDGEWATER LANDFILL	02/17/1990	UNKNOWN: UNKNOWN	OTHER SOURCE: LEACHATE VENTS	UNKNOWN
MAPPED	S90-0152	NA	FIRE STATION	268 BEDFORD ST	UNKNOWN	#2 FUEL OIL	UNKNOWN	UNKNOWN
MAPPED	S90-0304	NA	PROPERTY/RESIDENCE	90 HOBART ST	04/26/1990	#2 FUEL OIL (51-100 GALLONS)	AST	UNKNOWN
MAPPED	S90-0410	NA	BFI LANDFILL	THATCHER ST	UNKNOWN	OTHER MATERIAL: FLUFF	OTHER SOURCE: TRANSPORTING	UNKNOWN
MAPPED	S90-0429	NA	PROPERTY	475 POND ST	UNKNOWN	OTHER MATERIAL: REFUSE/DRUM STORAGE	OTHER SOURCE: REFUSE/DRUMS	UNKNOWN
MAPPED	S90-0526	NA	PROPERTY/RESIDENCE	115 SPRING ST	UNKNOWN	UNKNOWN: UNKNOWN SUBSTANCE	OTHER SOURCE: RUST COLOR SUB	UNKNOWN
MAPPED	S90-0535	NA	PROPERTY	396 HIGHLAND ST	07/19/1990	UNKNOWN: UNKNOWN MATERIAL	OTHER SOURCE: TREES DYING	UNKNOWN
MAPPED	S90-0731	NA	ROADWAY	768 CENTRAL ST	10/03/1990	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
MAPPED	S90-0783	NA	ROADWAY	709 CENTRAL ST	10/19/1990	WASTE OIL	OTHER SOURCE: CONTAINERS	UNKNOWN
MAPPED	S91-0015	NA	MUELLER CORP	530 SPRING ST	UNKNOWN	MISCELLANEOUS OIL	PIPE/HOSE/LINE	UNKNOWN
MAPPED	S91-0304	NA	WATERWAY	SATUCKET RIVER/RTE 106	05/31/1991	OTHER MATERIAL: OIL SHEEN	OTHER SOURCE: UNKNOWN	UNKNOWN
MAPPED	S91-0549	4-1311	PROPERTY	RT 18, 117 N. BEDFORD ST	UNKNOWN	#2 FUEL OIL	UST	UNKNOWN
MAPPED	S92-0006	NA	TRANSFORMER	635 PLYMOUTH ST	01/04/1992	TRANSFORMER OIL (11-50 GALLONS)	TRANSFORMER	UNKNOWN
MAPPED	S92-0228	NA	FOXBORO COMPANY	600 N BEDFORD ST	UNKNOWN	#6 FUEL OIL	UST	UNKNOWN
MAPPED	S92-0293	NA	PROPERTY	103 EAST ST	UNKNOWN	LUBRICATING OIL	OTHER SOURCE: DUMPING	UNKNOWN
MAPPED	S92-0478	NA	BATTI'S AUTO BODY	26 NORTH CENTRAL ST	UNKNOWN	GASOLINE	UNKNOWN	UNKNOWN

Table 1 (Continued)

MAPPED/NOT MAPPED	SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES	STATUS
MAPPED	S92-0525	NA	GENERAL MAIL FACILITY	W UNION ST (E OF PEARL ST)	UNKNOWN	OTHER MATERIAL: PETROLEUM HYDROCARBONS (251-500)	OTHER SOURCE: FOUNDRY FILL	UNKNOWN
MAPPED	S92-0693	NA	SHAW'S SUPERMARKET	140 LAUREL ST	09/28/1992	DIESEL FUEL	UST	UNKNOWN
MAPPED	S93-0090	NA	FOXBORO CORP	600 N BEDFORD ST	11/09/1992	#4 FUEL OIL	AST	UNKNOWN
MAPPED	S93-0110	NA	ROADWAY	N CENTRAL @ WILLOW AVE	02/21/1993	HYDRAULIC FLUID	DRUM	UNKNOWN
MAPPED	S93-0539	9-9999	FLINT & SON AUTO	NORTH CENTAL ST	07/29/1993	GASOLINE	VEH. FUEL TANK	UNKNOWN
MAPPED	NA	4-0000060	MURRAY CARVER LANDFILL	15 WHITMAN ST	01/15/87	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	ADQREG
MAPPED	NA	4-0000061	E BRIDGEWATER LANDFILL	BRIDGE ST	01/15/87	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	ADQREG
MAPPED	NA	4-0000526	COUNTRY CONVENIENCE STORE (COMMERCIAL)	210 POND ST	03/23/88	PETROLEUM BASED OIL	UST	TIER1C
MAPPED	NA	4-0000594	PRECISE ENGINEERING (COMMERCIAL)	24 WEST UNION ST	01/15/89	UNKNOWN CHEMICAL OF TYPE - OIL; UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	UNKNOWN	TIER1A
MAPPED	NA	4-0000751	APARTMENTS & RETAIL STORES (COMMERCIAL; FORMER; FOUNDRY; RESIDNTIAL; WETLANDS)	55 NORTH BEDFORD ST	07/15/89	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	UNCONTAIN; UNKNOWN	RAO
MAPPED	NA	4-0000860	CONRAD'S CITGO STATION (GASSTATION)	49 CENTRAL ST	03/23/89	GASOLINE	UST	TIER1C
MAPPED	NA	4-0001311	SKIPS GAS & DIESEL (GASSTATION)	117 NORTH BEDFORD ST	07/15/93	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	NA	4-0010354	CUMBERLAND FARMS (COMMERCIAL)	1055 WASHINGTON ST	03/28/94	GASOLINE (135 PPM); LEAD (.59 PPM)	UST	RAO
MAPPED	NA	4-0010458	CARRIAGE CROSSING PLAZA (COMMERCIAL)	225 BEDFORD ST	05/06/94	ETHENE, TETRACHLORO- (10 GAL); ETHENE, TETRACHLORO- (10 GAL)	PIPE	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES	STATUS
MAPPED	NA	4-0010463	SELF SERVE GAS STATION (COMMERCIAL; RESIDNTIAL)	475 NORTH BEDFORD ST	04/04/94	BENZENE (3300 PPB); BENZENE, METHYL- (16000 PPB); BENZENE, ETHYL- (3500 PPB); BENZENE, DIMETHYL (16000 PPB); PROPANE, 2-METHOXY-2-METHYL- (8500 PPB); UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (8500 PPB)	UST	RAO
MAPPED	NA	4-0010473	OFF PLEASANT ST (ROADWAY)	100 SUMMER ST	05/10/94	UNKNOWN CHEMICAL OF UNKNOWN TYPE (50 GAL)	PIPE	RAO
MAPPED	NA	4-0010517	BLDG 5 (INDUSTRIAL)	600 NORTH BEDFORD ST	05/26/94	FUEL OIL #2 (40 GAL); FUEL OIL #2 (15 GAL)	PIPE	RAO
MAPPED	NA	4-0010811	NO LOCATION AID (RESIDNTIAL)	855 CENTRAL ST	10/01/94	GASOLINE (10 GAL)	VEHICLE	RAO
MAPPED	NA	4-0010832	EAST BRIDGEWATER FIRE STATION (MUNICIPAL)	268 BEDFORD ST RTE 18	10/11/94	TOTAL PETROLEUM HYDROCARBONS (TPH) (50 PPMV); FUEL OIL #2	PIPE	RAO
MAPPED	NA	4-0010943	AUGUST INTERIORS INC (INDUSTRIAL)	523 SPRING ST	11/22/94	FUEL OIL #2 (130 PPM); PHENOL, PENTACHLORO- (33 PPM)	8K GALCAP; UST	RAO
MAPPED	NA	4-0010944	NO LOCATION AID (RESIDNTIAL)	4 LYNN LEE TER	11/22/94	FUEL OIL #2 (25 GAL)	AST	RAO
MAPPED	NA	4-0012061	HOPPYS SERVICE STA FMR (COMMERCIAL)	1325 PLYMOUTH ST	04/05/96	OIL	AST; DRUMS	RAO
MAPPED	NA	4-0012116	NEXT TO EASTERN STATES STEEL (ABANSITE; INDUSTRIAL)	24 WEST UNION ST	04/24/96	1,1'-BIPHENYL, CHLORO-DERIVS. (110 MG/KG); ARSENIC (43 MG/KG); LEAD (1100 MG/KG)	UNKNOWN	RAONR
MAPPED	NA	4-0012315	AUGUST INTERIORS	523 SPRING ST	05/30/96	PHENOL, PENTACHLORO- (33 PPM)	UNKNOWN	RAO
MAPPED	NA	4-0012369	COMMERCIAL PROPERTY (COMMERCIAL)	4-6 WEST UNION ST	07/24/96	FUEL OIL #2	UST	TIERII
MAPPED	NA	4-0012450	EASTERN STATES STEEL (INDUSTRIAL)	36 COOK ST	08/23/96	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	DRUMS; NUMEROUS; UNKNOWN	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES	STATUS
MAPPED	NA	4-0012862	MARTELL RESIDENCE (RESIDNTIAL)	494 BEDFORD ST RTE 18	02/25/97	FUEL OIL #2 (150 GAL)	AST	RAO
MAPPED	NA	4-0012940	EASTERN STATES STEEL	36 COOK ST	04/04/97	BENZ[A]ANTHRACENE (23 PPM); BENZ[E]ACEPHENANTHRYLENE (58 PPM); BENZO[A]PYRENE (29 PPM); CHRYSENE (24 PPM); INDENO(1,2,3-CD)PYRENE (29 PPM); DIBENZ[A,H]ANTHRACENE (6.5 PPM)	UNKNOWN	TIER1D
MAPPED	NA	4-0013087	EASTERN STATES STEEL (COMMERCIAL)	36 COOK ST	06/11/97	1,1'-BIPHENYL, CHLORO-DERIVS. (1000 PPM); ARSENIC (110 PPM)	UNKNOWN	TIER1D
MAPPED	NA	4-0013479	NO LOCATION AID (COMMERCIAL)	378 PLEASANT ST	11/12/97	WASTE OIL (173 PPMV)	UST	RAO
MAPPED	NA	4-0013797	CUMBERLAND FARMS (COMMERCIAL)	1055 WASHINGTON ST	04/13/98	GASOLINE (100 PPM)	UST	REMOPS
MAPPED	NA	4-0014224	NO LOCATION AID (INDUSTRIAL)	394 SPRING ST	10/02/98	WASTE OIL (50 GAL)	DRUMS	RAO
MAPPED	NA	4-0014476	D&J TRUCKING (COMMERCIAL)	498 BEDFORD ST	01/17/99	FUEL OIL #2 (200 GAL)	AST	RAO
MAPPED	NA	4-0015302	NO LOCATION AID (ROADWAY)	1668 CENTRAL ST	02/15/00	DIESEL FUEL (70 GAL)	SADDLETANK	RAO
MAPPED	NA	4-0015508	PRIME ENERGY (COMMERCIAL)	49 CENTRAL ST	05/28/00	GASOLINE (20 GAL)	UST	RAO
MAPPED	NA	4-0015548	NO LOCATION AID (RESIDNTIAL)	281 SPRING ST	06/16/00	FUEL OIL #2 (25 GAL)	AST; PIPE	RAO
MAPPED	NA	4-0016093	CENTRAL SCHOOL	107 CENTRAL ST	03/01/01	FUEL OIL #2 (75 GAL)	AST	RAO
MAPPED	NA	4-0016318	NO LOCATION AID	600 NORTH BEDFORD ST	06/18/01	ETHENE, 1,1-DICHLORO- (3.4 PPB); BENZO(K)FLUORANTHENE (5.4 PPM)		RAO
MAPPED	NA	4-0016805	BFI LANDFILL	234 THATCHER ST	12/19/01	FUEL OIL #2 (100 PPM)	PIPE; UST	RAO
MAPPED	NA	4-0016937	NO LOCATION AID (COMMERCIAL)	49 CENTRAL ST	03/08/02	GASOLINE (70.2 GAL)	UNKNOWN	TCLASS
MAPPED	NA	4-0017187	SHAW'S DISTRIBUTION CENTER	140 LAUREL ST	06/27/02	CADMIUM (7.4 PPB); NICKEL (170 PPB); ZINC (2400 PPB)	UNKNOWN	TIER1C

Table 1 (Continued)

MAPPED/NOT MAPPED	SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES	STATUS
MAPPED	NA	4-0017840	IN FRONT OF # 75 LEAF LN (ROADWAY)	LEAF LN	06/02/03	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (20 GAL)	PIPE	UNCLSS
NOT MAPPED	S88-0054	4-0526	UNKNOWN	210 FORD ST	02/02/1988	GASOLINE	UST	UNKNOWN
NOT MAPPED	S89-0067	NA	UNKNOWN	OFF HUDSON STREET NEAR HANSON	UNKNOWN	UNKNOWN	DRUM	UNKNOWN
NOT MAPPED	S89-0828	NA	ROADWAY	WASHINGTON ST	11/26/1989	#2 FUEL OIL (SHEEN)	OTHER SOURCE: UNKNOWN	UNKNOWN
NOT MAPPED	S90-0453	NA	PROPERTY	OFF POND ST	UNKNOWN	OTHER MATERIAL: SUSPECTED WASTE OIL	DRUM	UNKNOWN
NOT MAPPED	S91-0284	NA	TRANSFORMER	BRIDGE ST - POLE 23	05/22/1991	TRANSFORMER OIL	TRANSFORMER	UNKNOWN
NOT MAPPED	NA	4-0012198	POLE #26 (ROADWAY)	ELM ST	05/22/96	UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL)	TRANSFORM	RAO
NOT MAPPED	NA	4-0012727	POLE #9 (RESIDNTIAL; ROADWAY)	ELM ST	12/23/96	UNKNOWN CHEMICAL OF TYPE - OIL (22 GAL)	TRANSFORM	RAO
NOT MAPPED	NA	4-0012933	POLE #2 (RESIDNTIAL)	KINGMAN CIR	04/02/97	UNKNOWN CHEMICAL OF TYPE - OIL (21 GAL)	TRANSFORM	RAO

Source: MDEP Bureau of Waste Site Cleanup. 2004. Downloadable Site Lists. http://www.state.ma.us/dep/bwsc/sites/sdown.htm

Notes:
Spill ID - Spill Identification Number (applicable for releases reported prior to October 1993). Definition: NA Not Applicable
RTN - Release Tracking Number. Unique ID number assigned to releases not remediated by October 1993 and to those occuring October 1993-present. Definition: NA Not Applicable.
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)
Materials - Chemical(s) in release
Sources - Origin(s) of release contamination. Definitions: AST Aboveground Storage Tank; UST Underground Storage Tank.
Status - 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.

Table 2
Maximum concentrations of contaminants detected in onsite soil
samples at the Old Colony Railroad site that exceeded comparison values (samples taken from 1985-2000)

Contaminant	Sample depth (feet)	Date of sample	Descriptive location of sample	Maximum concentration (ppm)	Comparison value (ppm)	Background soil levels (ppm)
Antimony	0-1	11/18/1997	SP-2 (soil pile on PE property)	26.3	RMEG (child) = 20 RMEG (adult) = 300	<1 - 8.8*
Arochlor 1248	0.5	6/3/1997	S-3 (soil pile on ESS property)	690	Total PCBs: CREG = 0.4	-
Arochlor 1254	0.5	6/3/1997	S-3 (soil pile on ESS property)	310	Chronic EMEG (child) = 1 Chronic EMEG (adult) = 10	-
Arochlor 1260	0.5	6/3/1997	S-9 (soil pile on ESS property)	2.7	Total PCBs: CREG = 0.4	-
Arsenic	4	8/1997	ESS property near former underground storage tanks	18,100	CREG = 0.5 Chronic EMEG (child), RMEG (child) = 20 Chronic EMEG (adult), RMEG (adult) = 200	<0.1 - 73*
Benzo(a)anthracene	4-8	8/1997	TP-D (south of ESS main building)	33	EPA RBC (residential) = 0.87	0.005 - 0.02 (rural soil) 0.169 - 59 (urban soil)
Benzo(a)pyrene	4-8	8/1997	TP-D (south of ESS main building)	42	CREG = 0.1	0.002 - 1.3 (rural soil) 0.165 - 0.22 (urban soil)
Benzo(b)fluoranthene	4-8	8/1997	TP-D (south of ESS main building)	65	EPA RBC (residential) = 0.87	0.02-0.03 (rural soil) 15 - 62 (urban soil)
Benzo(k)fluoranthene	4-8	8/1997	TP-D (south of ESS main building)	27	EPA RBC (residential) = 0.87	0.01 -0.11 (rural soil) 0.3-26 (urban soil)
Cadmium	0-1	11/18/1997	SP-4 (soil pile on PE property)	20.7	Chronic EMEG (child) = 10 Chronic EMEG (adult) = 100	0.01 - 1
Chromium (total)	0-1	11/18/1997	SP-4 (soil pile on PE property)	428	Hexavalent Chromium: RMEG (child) = 200 Hexavlent Chromium: RMEG (adult) = 2,000	1 - 1000*[†]
Dibenzo(a,h)anthracene	4-8	8/1997	TP-D (south of ESS main building)	11	EPA RBC (residential) = 0.087	-
Indeno(1,2,3-c,d)pyrene	0-7	12/19/1985	composite of Test Pits A,B,C,D (ESS property)	29	EPA RBC (residential) = 0.87	0.01 - 0.015 (rural soil) 8.0 - 61 (urban soil)
Lead	0-1	11/18/1997	SP-1 (soil pile on PE property)	18,100	S-1 & GW-1 MDEP standard = 300	<10 - 300*
PCBs (total)	0-0.25	9/1999	C 400 (northeast corner of ESS property)	22,500	CREG = 0.4	-

Table 2 (Continued)

*Observed range for the Eastern United States (east of 96th meridian). USGS. Shacklette HT, Boerngen JG. 1984. Element Concentrations in Soils and Other Surficial Materials of the conterminous United States. U.S. Geological Survey Professional Paper 1270. Washington: United States Government Printing Office, 1984.

†Background soil level for total chromium

ppm = parts per million

PE = Precise Engineering

< = less than

ESS = Eastern States Steel

PCBs = polychlorinated biphenyls

Table 3
Maximum concentrations of contaminants detected in onsite groundwater samples at the Old Colony Railroad site that exceeded comparison values (samples taken from 1985-1999)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Arsenic	2/22/1988	Composite of OW-1, OW-2, OW-3 (PE property)	10	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3 Chronic EMEG (adult); RMEG (adult) = 10 MDEP MMCL = 50
Benzene	2/22/1988	OW-2 (PE property near chlorinated solvent storage)	18.4	CREG = 0.6 MDEP MMCL = 5
Cadmium	3/19/1996	OW-5 (PE property)	60	Chronic EMEG (child) = 2 Chronic EMEG (adult) = 7 MDEP MMCL = 5
Chromium	3/19/1996	OW-5 (PE property)	90	Hexavalent Chromium: RMEG (child) = 30 Hexavalent Chromium: RMEG (adult) = 300 MDEP MMCL = 100
1,2-Dichloroethylene, cis-	3/19/1996	OW-3 (PE property near former UST)	65	Intermediate EMEG (child) = 3,000 Intermediate EMEG (adult) = 10,000 EPA RBC (tap water) = 61 MDEP MMCL = 70
1,2-Dichloroethylene, trans-	2/22/1988	OW-3 (PE property near former UST)	1,020	Intermediate EMEG (child) = 2,000 Intermediate EMEG (adult) = 7,000 RMEG (child) = 200 RMEG (adult) = 700 MDEP MMCL = 100
Lead	3/19/1996	OW-5 (PE property)	100	MCLG = 0 MDEP MMCL = 15
Mercury	2/22/1988	Composite of OW-1, OW-2, OW-3 (PE property)	140	MDEP MMCL = 2 Methylmercury: Chronic EMEG (child) = 3 Methylmercury: Chronic EMEG (adult) = 10
Tetrachlorethylene	12/11/1997	SEA-2S (PE property)	186	RMEG (child) = 100 RMEG (adult) = 400 EPA RBC (tap water) = 0.1 MDEP MMCL = 5

Comparison values (source organization, reference):

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)

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)

EPA RBC = EPA Region 3 Risk Based Concentration for soil (U.S. EPA, U.S. EPA 2004)

S-1 & GW-1 MDEP standard = Massachusetts Contingency Plan Method 1 soil category S-1 standards [310 CMR 40.0975(6)(a)] [Massachusetts Department of Environmental Protection (MDEP), MDEP 2003]

Data sources:

Cambridge Analytical Associates. 1986. Draft: Environmental Site Assessment, Eastern States Steel, 36 Cook Street, East Bridgewater, Massachusetts.

Cambridge Environmental, Inc. 1998. Method 3 Risk Characterization for the Precise Engineering Site, East Bridgewater, Massachusetts.

Horsley and Witten, Inc. 1998. Scope of Work: Environmental Site Assessment, Eastern States Steel (RTN 4-12940), 36 Cook Street, East Bridgewater, Massachusetts.

Kupferman and Weber, Inc. 1990. Environmental Site Assessment: Update, 36 Cook Street, E. Bridgewater, Massachusetts.

Roy F. Weston, Inc. 2001. Removal Program: Preliminary Assessment/Site Investigation Report for the Old Colony Railroad Site.

SEA Consultants, Inc. 1996. Phase I: Site Investigation, Precise Engineering, 24 West Union Street, East Bridgewater, Massachusetts.

SEA Consultants, Inc. 1997. Phase II: Scope of Work, Precise Engineering.

SEA Consultants, Inc. 1998. Phase II: Comprehensive Site Assessment, Precise Engineering.

Unless otherwise noted, soil background concentrations are from ATSDR Toxicological Profiles 2003 (on CD-ROM), ATSDR 2003.

Table 3 (Continued)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Trichloroethylene	2/22/1988	OW-3 (PE property near former UST)	313	EPA RBC (tap water) = 0.026 MCLG = 0 MDEP MMCL = 5
Vinyl Chloride	2/22/1988	OW-3 (PE property near former UST)	1,170	CREG = 0.03 Chronic EMEG (child) = 0.2 Chronic EMEG (adult) = 0.7 MCLG = 0 MDEP MMCL = 2

ppb = parts per billion

PE = Precise Engineering

UST = Underground Storage Tank

Data sources:

Cambridge Analytical Associates. 1986. Draft: Environmental Site Assessment, Eastern States Steel, 36 Cook Street, East Bridgewater, Massachusetts.

SEA Consultants, Inc. 1996. Phase I: Site Investigation, Precise Engineering, 24 West Union Street, East Bridgewater, Massachusetts.

SEA Consultants, Inc. 1997. Phase II: Scope of Work, Precise Engineering.

SEA Consultants, Inc. 1998. Phase II: Comprehensive Site Assessment, Precise Engineering.

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 mirroring greater than 1 year) (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 and considers vulnerabilities of children when it comes to environmental exposures). (ATSDR, ATSDR 2003a)

RMEG (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 (U.S. EPA, U.S. EPA 2004a)

MCLG = Maximum Contaminant Level Goal for drinking water (U.S. EPA, ATSDR 2003a)

MDEP MMCL = Massachusetts Department of Environmental Protection Massachusetts Maxiumum Contaminant Level for drinking water (MDEP, MDEP 2001)

Table 4
Maximum concentrations of contaminants detected in onsite surface water samples at the Old Colony Railroad site that exceeded comparison values (samples taken from 1988-1998)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
1,2-Dichloroethylene, trans-	5/27/1988	Downstream reach of drainage ditch	145	Intermediate EMEG (child) = 2,000 Intermediate EMEG (adult) = 7,000 RMEG (child) = 200 RMEG (adult) = 700 MDEP MMCL = 100
Tetrachlorethylene	5/27/1988	Downstream reach of drainage ditch	176	RMEG (child) = 100 RMEG (adult) = 400 EPA RBC = 1.1 MDEP MMCL = 5
Trichloroethylene	5/27/1988	Downstream reach of drainage ditch	59.7	EPA RBC = 1.6 MCLG = 0 MDEP MMCL = 5
Vinyl Chloride	5/27/1988	Downstream reach of drainage ditch	14.5	MCLG = 0 CREG = 0.03 Chronic EMEG (child) = 0.2 Chronic EMEG (adult) = 0.7 MDEP MMCL = 2

ppb = parts per billion

Data sources:

SEA Consultants, Inc. 1997. Phase II: Scope of Work, Precise Engineering.

SEA Consultants, Inc. 1998. Phase II: Comprehensive Site Assessment, Precise Engineering.

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 mirroring greater than 1 year) (ATSDR, ATSDR 2003a)

RMEG (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, ATSDR 2003a)

EPA RBC = EPA Region 3 Risk Based Concentration for tap water U.S. EPA, U. S. EPA 2004)

MDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MDEP, MDEP 2001)

MCLG = Maximum Contaminant Level Goal for drinking water (U.S. 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 and considers vulnerabilities of children when it comes to environmental exposures).(ATSDR, ATSDR 2003a)

Table 5
Maximum concentrations of contaminants detected in onsite sediment samples at the Old Colony Railroad site that exceeded comparison values (samples taken from 1985-1999)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppm)	Comparison value for soil (ppm)	Background soil levels (ppm)
Arsenic	11/18/1997	SED-3 (downstream drainage ditch)	42.6	CREG = 0.5 Chronic EMEG (child), RMEG (child) = 20 Chronic EMEG (adult), RMEG (adult) = 200	<0.1 - 73
Benzo(a)anthracene	11/18/1997	SED-2 (midstream drainage ditch)	8.72	EPA RBC (residential) = 0.87	0.169 - 59 (urban soil)
Benzo(a)pyrene	11/18/1997	SED-2 (midstream drainage ditch)	19.7	CREG = 0.1 EPA RBC (residential) = 0.087	0.165 - 0.22 (urban soil)
Benzo(b)fluoranthene	11/18/1997	SED-2 (midstream drainage ditch)	30.9	EPA RBC (residential) = 0.87	15 - 62 (urban soil)
Dibenzo(a,h)anthracene	11/18/1997	SED-2 (midstream drainage ditch)	49.4	EPA RBC (residential) = 0.087	-
Lead	11/18/1997	SED-1 (upstream drainage ditch)	436	S-1 & GW-1 MDEP standard = 300	160 - 840 (urban soil)

ppm = parts per million

< = less than

Data source:

SEA Consultants, Inc. 1997. Phase II: Scope of Work, Precise Engineering.

Soil background concentrations are from ATSDR Toxicological Profiles 2003 (on CD-ROM), ATSDR 2003.

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 (U.S. EPA, U.S. EPA 2004)

S-1 & GW-1 MDEP standard = Massachusetts Contingency Plan Method 1 soil category S-1 standards [310 CMR 40.0975(6)(a)] [Massachusetts Department of Environmental Protection (MDEP), MDEP 2003]

TABLE 6a
Cancer Incidence
East Bridgewater, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	29	29.9	97	65 -- 139	20	21.7	92	56 -- 143	9	8.2	110	50 -- 208
Brain & CNS	9	15.4	58	27 -- 111	6	8.0	74	27 -- 163	3	7.4	NC	NC -- NC
Breast	139	135.1	103	86 -- 121	2	0.9	NC	NC -- NC	137	134.2	102	86 -- 121
Kidney & Renal Pelvis	18	19.2	94	56 -- 149	10	11.9	84	40 -- 155	8	7.3	110	47 -- 216
Leukemia	22	17.3	127	80 -- 193	10	9.7	103	49 -- 190	12	7.6	158	82 -- 276
Liver	3	4.7	NC	NC -- NC	2	3.4	NC	NC -- NC	1	1.2	NC	NC -- NC
Lung & Bronchus	123	115.6	106	88 -- 127	75	68.9	109	86 -- 136	48	46.7	103	76 -- 136
NHL	36	30.0	121	85 -- 167	22	16.0	138	86 -- 209	14	13.9	101	55 -- 169

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
CNS = Central Nervous System

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

TABLE 6b
Cancer Incidence
East Bridgewater, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	11	9.2	120	60 -- 214	6	6.7	90	33 -- 196	5	2.5	198	64 -- 462
Brain & CNS	3	4.4	NC	NC -- NC	2	2.2	NC	NC -- NC	1	2.2	NC	NC -- NC
Breast	35	33.6	104	73 -- 145	0	0.2	NC	NC -- NC	35	33.4	105	73 -- 146
Kidney& Renal Pelvis	1	4.2	NC	NC -- NC	0	2.5	NC	NC -- NC	1	1.7	NC	NC -- NC
Leukemia	5	4.5	111	36 -- 260	3	2.5	NC	NC -- NC	2	2.0	NC	NC -- NC
Liver	1	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.3	NC	NC -- NC
Lung & Bronchus	34	30.2	112	78 -- 157	24	19.6	122	78 -- 182	10	10.6	94	45 -- 173
NHL	9	6.8	133	61 -- 252	7	3.5	197	79 -- 407	2	3.2	NC	NC -- NC

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

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

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

TABLE 6c
Cancer Incidence
East Bridgewater, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	9.2	66	24 -- 143	5	6.7	75	24 -- 176	1	2.5	NC	NC -- NC
Brain & CNS	1	5.2	NC	NC -- NC	0	2.6	NC	NC -- NC	1	2.6	NC	NC -- NC
Breast	43	42.2	102	74 -- 137	1	0.3	NC	NC -- NC	42	42.0	100	72 -- 135
Kidney & Renal Pelvis	7	6.2	114	46 -- 234	3	3.9	NC	NC -- NC	4	2.3	NC	NC -- NC
Leukemia	9	4.8	178	81 -- 338	3	2.8	NC	NC -- NC	6	2.1	289*	105 -- 630
Liver	0	1.3	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	34	35.6	96	66 -- 134	18	21.5	84	50 -- 132	16	14.1	114	65 -- 185
NHL	12	9.2	131	68 -- 229	8	5.0	163	70 -- 320	4	4.2	NC	NC -- NC

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

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

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

TABLE 6d
Cancer Incidence
East Bridgewater, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	12	11.8	102	52 -- 178	9	8.6	105	48 -- 199	3	3.2	NC	NC -- NC
Brain & CNS	5	5.9	85	28 -- 199	4	3.3	NC	NC -- NC	1	2.6	NC	NC -- NC
Breast	61	60.9	100	77 -- 129	1	0.5	NC	NC -- NC	60	60.5	99	76 -- 128
Kidney & Renal Pelvis	10	9.2	109	52 -- 200	7	5.7	122	49 -- 251	3	3.5	NC	NC -- NC
Leukemia	8	8.3	97	42 -- 190	4	4.6	NC	NC -- NC	4	3.7	NC	NC -- NC
Liver	2	2.6	NC	NC -- NC	2	2.0	NC	NC -- NC	0	0.7	NC	NC -- NC
Lung & Bronchus	55	51.7	106	80 -- 138	33	28.6	115	79 -- 162	22	23.1	95	60 -- 144
NHL	15	14.4	104	58 -- 172	7	7.8	90	36 -- 186	8	6.7	120	52 -- 237

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
CNS = Central Nervous System

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

TABLE 7a
Cancer Incidence
Census Tract 5231, East Bridgewater, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	7.3	123	56 -- 234	6	5.3	112	41 -- 244	3	1.9	NC	NC -- NC
Brain & CNS	1	3.6	NC	NC -- NC	1	1.9	NC	NC -- NC	0	1.7	NC	NC -- NC
Breast	29	30.9	94	63 -- 135	0	0.2	NC	NC -- NC	29	30.7	95	63 -- 136
Kidney & Renal Pelvis	3	4.6	NC	NC -- NC	1	2.9	NC	NC -- NC	2	1.7	NC	NC -- NC
Leukemia	4	4.1	NC	NC -- NC	1	2.3	NC	NC -- NC	3	1.8	NC	NC -- NC
Liver	0	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	32	27.9	115	78 -- 162	23	16.9	136	86 -- 204	9	10.9	82	38 -- 156
NHL	10	7.1	142	68 -- 261	4	3.8	NC	NC -- NC	6	3.2	185	68 -- 403

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
CNS = Central Nervous System

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

TABLE 7b
Cancer Incidence
Census Tract 5231, East Bridgewater, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	5	2.4	211	68 -- 492	3	1.7	NC	NC -- NC	2	0.6	NC	NC -- NC
Brain & CNS	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Breast	11	8.3	133	66 -- 237	0	0.0	NC	NC -- NC	11	8.2	133	66 -- 239
Kidney & Renal Pelvis	0	1.1	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.4	NC	NC -- NC
Leukemia	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Liver	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	8	7.9	101	44 -- 199	7	5.2	135	54 -- 278	1	2.7	NC	NC -- NC
NHL	4	1.7	NC	NC -- NC	3	0.9	NC	NC -- NC	1	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 7c
Cancer Incidence
Census Tract 5231, East Bridgewater, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	1	2.3	NC	NC -- NC	1	1.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Brain & CNS	0	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.6	NC	NC -- NC
Breast	5	9.8	51	17 -- 119	0	0.1	NC	NC -- NC	5	9.7	52	17 -- 120
Kidney & Renal Pelvis	2	1.5	NC	NC -- NC	0	1.0	NC	NC -- NC	2	0.5	NC	NC -- NC
Leukemia	3	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	3	0.5	NC	NC -- NC
Liver	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	13	8.8	148	79 -- 254	8	5.4	148	64 -- 291	5	3.3	149	48 -- 349
NHL	3	2.2	NC	NC -- NC	1	1.2	NC	NC -- NC	2	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 7d
Cancer Incidence
Census Tract 5231, East Bridgewater, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	2.5	NC	NC -- NC	2	1.8	NC	NC -- NC	1	0.7	NC	NC -- NC
Brain & CNS	1	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Breast	13	13.2	98	52 -- 168	0	0.1	NC	NC -- NC	13	13.1	99	53 -- 170
Kidney & Renal Pelvis	1	2.0	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC
Leukemia	1	1.8	NC	NC -- NC	1	1.0	NC	NC -- NC	0	0.8	NC	NC -- NC
Liver	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	11	11.1	99	50 -- 178	8	6.1	131	56 -- 257	3	4.9	NC	NC -- NC
NHL	3	3.1	NC	NC -- NC	0	1.7	NC	NC -- NC	3	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 8a
Cancer Incidence
Census Tract 5232.01, East Bridgewater, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	5	7.7	65	21 -- 152	4	5.4	NC	NC -- NC	1	2.3	NC	NC -- NC
Brain & CNS	3	3.5	NC	NC -- NC	3	1.8	NC	NC -- NC	0	1.7	NC	NC -- NC
Breast	30	32.4	92	62 -- 132	0	0.2	NC	NC -- NC	30	32.2	93	63 -- 133
Kidney & Renal Pelvis	1	4.5	NC	NC -- NC	1	2.8	NC	NC -- NC	0	1.8	NC	NC -- NC
Leukemia	5	4.2	118	38 -- 276	3	2.3	NC	NC -- NC	2	2.0	NC	NC -- NC
Liver	0	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	28	27.5	102	68 -- 147	17	16.4	104	60 -- 166	11	11.1	99	49 -- 177
NHL	6	7.3	83	30 -- 180	3	3.8	NC	NC -- NC	3	3.5	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 8b
Cancer Incidence
Census Tract 5232.01, East Bridgewater, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	0	2.4	NC	NC -- NC	0	1.7	NC	NC -- NC	0	0.7	NC	NC -- NC
Brain & CNS	1	1.0	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
Breast	7	8.4	84	34 -- 173	0	0.0	NC	NC -- NC	7	8.3	84	34 -- 174
Kidney & Renal Pelvis	0	1.0	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
Leukemia	1	1.1	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Liver	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	12	7.3	166	85 -- 289	9	4.7	191	87 -- 362	3	2.5	NC	NC -- NC
NHL	2	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	1	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 8c
Cancer Incidence
Census Tract 5232.01, East Bridgewater, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	2.3	NC	NC -- NC	2	1.7	NC	NC -- NC	1	0.7	NC	NC -- NC
Brain & CNS	0	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.6	NC	NC -- NC
Breast	12	10.2	118	61 -- 206	0	0.1	NC	NC -- NC	12	10.1	119	61 -- 208
Kidney & Renal Pelvis	1	1.5	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC
Leukemia	2	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC
Liver	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	5	8.4	60	19 -- 139	3	5.1	NC	NC -- NC	2	3.3	NC	NC -- NC
NHL	1	2.2	NC	NC -- NC	0	1.2	NC	NC -- NC	1	1.1	NC	NC -- NC

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

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

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

TABLE 8d
Cancer Incidence
Census Tract 5232.01, East Bridgewater, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	3.1	NC	NC -- NC	2	2.2	NC	NC -- NC	0	0.9	NC	NC -- NC
Brain & CNS	2	1.3	NC	NC -- NC	2	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Breast	11	14.4	76	38 -- 137	0	0.1	NC	NC -- NC	11	14.3	77	38 -- 138
Kidney & Renal Pelvis	0	2.2	NC	NC -- NC	0	1.3	NC	NC -- NC	0	0.8	NC	NC -- NC
Leukemia	2	2.0	NC	NC -- NC	1	1.1	NC	NC -- NC	1	1.0	NC	NC -- NC
Liver	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung & Bronchus	11	12.5	88	44 -- 158	5	6.8	73	24 -- 171	6	5.6	106	39 -- 232
NHL	3	3.5	NC	NC -- NC	2	1.8	NC	NC -- NC	1	1.7	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

TABLE 9a
Cancer Incidence
Census Tract 5232.02, East Bridgewater, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	15	14.9	101	56 -- 166	10	10.9	92	44 -- 169	5	4.0	126	40 -- 293
Brain & CNS	5	8.3	60	19 -- 140	2	4.3	NC	NC -- NC	3	4.0	NC	NC -- NC
Breast	80	71.8	111	88 -- 139	2	1.5	NC	NC -- NC	78	71.3	109	86 -- 137
Kidney & Renal Pelvis	14	10.0	139	76 -- 234	8	6.2	128	55 -- 253	6	3.8	157	57 -- 341
Leukemia	13	9.0	144	77 -- 247	6	5.1	118	43 -- 257	7	4.0	180	72 -- 370
Liver	3	2.4	NC	NC -- NC	2	1.8	NC	NC -- NC	1	0.6	NC	NC -- NC
Lung & Bronchus	63	60.2	105	80 -- 134	35	35.6	98	68 -- 137	28	24.6	114	76 -- 165
NHL	20	15.5	129	79 -- 200	15	8.4	179	100 296	5	7.1	71	23 -- 165

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
CNS = Central Nervous System

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

TABLE 9b
Cancer Incidence
Census Tract 5232.02, East Bridgewater, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	4.5	134	49 -- 291	3	3.3	NC	NC -- NC	3	1.2	NC	NC -- NC
Brain & CNS	2	2.3	NC	NC -- NC	1	1.2	NC	NC -- NC	1	1.1	NC	NC -- NC
Breast	17	16.9	100	59 -- 161	0	0.1	NC	NC -- NC	17	16.8	101	59 -- 162
Kidney & Renal Pelvis	1	2.1	NC	NC -- NC	0	1.3	NC	NC -- NC	1	0.8	NC	NC -- NC
Leukemia	4	2.3	NC	NC -- NC	2	1.3	NC	NC -- NC	2	1.0	NC	NC -- NC
Liver	1	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC
Lung & Bronchus	14	15.1	93	51 -- 156	8	9.7	82	35 -- 162	6	5.4	112	41 -- 243
NHL	3	3.4	NC	NC -- NC	3	1.8	NC	NC -- NC	0	1.6	NC	NC -- NC

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

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

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

TABLE 9c
Cancer Incidence
Census Tract 5232.02, East Bridgewater, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	4.5	NC	NC -- NC	2	3.3	NC	NC -- NC	0	1.2	NC	NC -- NC
Brain & CNS	1	2.8	NC	NC -- NC	0	1.4	NC	NC -- NC	1	1.4	NC	NC -- NC
Breast	26	22.3	117	76 -- 171	1	0.2	NC	NC -- NC	25	22.2	113	73 -- 167
Kidney & Renal Pelvis	4	3.2	NC	NC -- NC	2	2.0	NC	NC -- NC	2	1.2	NC	NC -- NC
Leukemia	4	2.5	NC	NC -- NC	2	1.5	NC	NC -- NC	2	1.1	NC	NC -- NC
Liver	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung & Bronchus	16	18.4	87	50 -- 141	7	11.0	64	26 -- 131	9	7.4	122	55 -- 231
NHL	8	4.7	169	73 -- 332	7	2.6	272*	109 -- 560	1	2.2	NC	NC -- NC

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

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

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

TABLE 9d
Cancer Incidence
Census Tract 5232.02, East Bridgewater, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	7	6.2	112	45 -- 231	5	4.6	108	35 -- 253	2	1.6	NC	NC -- NC
Brain & CNS	2	3.3	NC	NC -- NC	1	1.8	NC	NC -- NC	1	1.4	NC	NC -- NC
Breast	37	33.3	111	78 -- 153	1	0.3	NC	NC -- NC	36	33.1	109	76 -- 151
Kidney & Renal Pelvis	9	5.1	178	81 -- 338	6	3.2	189	69 -- 411	3	1.9	NC	NC -- NC
Leukemia	5	4.5	112	36 -- 262	2	2.5	NC	NC -- NC	3	1.9	NC	NC -- NC
Liver	2	1.4	NC	NC -- NC	2	1.1	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	33	28.2	117	81 -- 164	20	15.7	127	78 -- 197	13	12.5	104	55 -- 178
NHL	9	7.8	116	53 -- 220	5	4.3	117	38 -- 274	4	3.5	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
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance
CNS = Central Nervous System

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

APPENDICES

Appendix A: Coding Definitions of Cancer Site/Type*

Cancer Site / Type	*ICD-O-1 and Other Pre-ICD-O-2 Codes*		***ICD-O-2 Codes***		***ICD-O-3 Codes***	
	Site code	***Histology code***	***Site code***	***Histology code***	***Site code***	***Histology code***
Bladder	188.0-188.9	except 9590-9980	C67.0-C67.9	except 9590-9989	C67.0-C67.9	except 9590-9989
Brain & Central Nervous System (CNS)	191.0-192.9	†	C70.0-C72.9	†	C70.0-C72.9	‡
Breast	174.0-174.9, 175.9	except 9590-9980	C50.0-C50.9	except 9590-9989	C50.0-C50.9	except 9590-9989
Kidney & Renal Pelvis	189.0, 189.1	except 9590-9980	C64.9, C65.9	except 9590-9989	C64.9, C65.9	except 9590-9989
Leukemia	140.0-199.9	includes O9800-O9943, O9951, P9803-P9943, B9803-B9943	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9800-9822, 9824-9826, 9828-9941 2. includes 9823, 9827	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964 2. includes 9823, 9827
Liver	155.0	except 9590-9980	C22.0	except 9590-9989	C22.0	except 9590-9989
Lung & Bronchus	162.2-162.9	except 9050-9053, 9590-9980	C34.0-C34.9	except 9590-9989	C34.0-C34.9	except 9590-9989
Non-Hodgkin's Lymphoma (NHL)	140.0-199.9	includes O9590-O9642, O9670-O9710, O9750, P9593-P9643, P9693-P9713, P9753, B9593-B9643, B9703	1. C00.0-C80.9 AND 2. All sites except C42.0, C42.1, C42.4	1. includes 9590-9595, 9670-9717 2. includes 9823, 9827	1. C00.0-C80.9 AND 2. All sites except C42.0, C42.1, C42.4	1. includes 9590-9596, 9670-9729 2. includes 9823, 9827

*Includes invasive tumors only, selected by excluding in situ stages J0, S0, TTISNXM0, TTANXMX, TTANXM0, TTAN0MX, TTISN0M0, TTISNXMX, TTISN0MX, TTISN0M0, TTIN0M0, TTIN0MX, TTINXM0, and TTINXMX (1982-1994 data) or by specifying behavior code (1995-2000 data).

Appendix A (Continued)

†Histology codes for Brain and Central Nervous System (pre-ICD-O-3)

ICD-O	O	9370, 9380, 9381, 9382, 9390, 9391, 9392, 9400, 9401, 9403, 9410, 9411, 9420, 9421, 9422, 9423, 9424, 9430, 9440, 9441, 9442, 9443, 9450, 9451, 9460, 9470, 9471, 9472, 9473, 9480, 9481, 9490, 9500, 9501, 9502, 9503, 9530, 9539, 9540, 9560, 9561.
SNOP	P	9363, 9383, 9393, 9403, 9413, 9423, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9533, 9543, 9563.
HLTHSTT	B	9383, 9393, 9403, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9530, 9533, 9537, 9543, 9563.

‡Histology codes for Brain and Central Nervous System (ICD-O-3)

ICD-O	O	9370, 9371, 9372, 9380, 9381, 9382, 9390, 9391, 9392, 9400, 9401, 9410, 9411, 9420, 9421, 9423, 9424, 9430, 9440, 9441, 9442, 9450, 9451, 9460, 9470, 9471, 9472, 9473, 9474, 9480, 9490, 9500, 9501, 9502, 9503, 9530, 9539, 9540, 9560, 9561, 9571.

Appendix B: Risk Factor Information for Selected Cancer Types

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Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 63,210 people in the United States in 2005, accounting for 7% of all cancers diagnosed in the United States among men and 2% among women. In Massachusetts, bladder cancer accounts for approximately 6% of all cancers diagnosed among males and females combined (ACS, 2005). Males are three times more likely to develop bladder cancer than females and whites are two times more likely to develop this disease than blacks. The risk of bladder cancer increases with age and the mean age at diagnosis is 68-69 years (ACS, 2000).

The greatest risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS, 2000). The risk of developing bladder cancer increases with the number of packs smoked per day and with duration of smoking. Further, the risk of bladder cancer may be higher in women than in men who smoke comparable numbers of cigarettes (Castelao et al., 2001). Approximately 25-60% of all bladder cancers can be attributed to tobacco use (Johansson and Cohen, 1997). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30% to 60% (Silverman et al., 1996).

Studies have also revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the United States (Johansson and Cohen, 1997). Occupational exposure to aromatic amines, such as benzidine and 2-naphthylamine, increases the risk of bladder cancer (ACS, 2000). These chemicals were common in the dye industry in the past. A higher risk of bladder cancer has also been observed among aromatic amine manufacturing workers as well as among workers in the rubber, leather, textiles, printing, and paint products industries (ACS, 2000; Silverman et al., 1996). The development of new chemicals, changed worker exposures, and the elimination of many known bladder carcinogens in the workplace have caused shifts in those occupations considered to be high risk. For example, risks among dye, rubber, and leather workers have declined over time, while other occupations such as motor vehicle operation (e.g., drivers of trucks, buses, and taxis) and the aluminum industry have emerged as potential high-risk occupations (Silverman et al., 1996). However, specific occupational exposures in these occupations have not been confirmed and study findings are not consistent. Further, the risk of bladder cancer from occupational exposures may be increased among smokers (ACS, 2000).

Dietary factors such as consumption of fried foods as well as foods high in fat and cholesterol have been found to be associated with increased bladder cancer risk (Silverman et al., 1996). Use of the Chinese herb, *Aristocholia fangchi,* found in some dietary supplements, has also been linked with bladder cancer (ACS, 2000). Use of some anti-cancer drugs (e.g., cyclophosphamide and chlornaphazine), use of phenacetin, and infection with *Shistosoma haematobium* (a parasite found in Africa) are thought to be associated with the development of bladder cancer, however, not all epidemiological studies have produced convincing findings (Silverman et al., 1996).

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS, 2000; Silverman et al., 1996). Exposure to chlorinated by-products in drinking water has also been suggested to increase bladder cancer risk, however, a recent population-based study found that an association was present only among smokers (Cantor et al., 1998).

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Brain and CNS Cancer

Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 50% of all reported primary brain and spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. Therefore, approximately 7.5% of brain and CNS tumors are malignant meningiomas. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the primitive stem cells of the cerebellum and are most often seen in children. Also, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS, 1999a). The American Cancer Society estimates that 18,500 Americans (10,620 men and 7,880 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,760 people (7,280 men and 5,480 women) will die from this disease in 2005 (ACS, 2005).

Brain and spinal cord cancers account for over 20% of all cancer types diagnosed among children aged 0-14 (ACS, 2005). About half of all childhood brain tumors are astrocytomas and 25% are medulloblastomas (ACS, 1999b). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme). Incidence rates are higher in males than in females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack, 1996).

Despite numerous scientific and medical investigations, and analyses, the causes of brain cancer are still largely unknown. Among the possible risk factors investigated in relation to this type of cancer are ionizing radiation, electromagnetic fields, occupational exposures, exposure to N-nitroso compounds, head trauma, and genetic disorders.

The most established risk factor (and only established environmental risk factor) for brain tumors (either cancerous or non-cancerous) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain tumors are caused by radiation to the head from the treatment of other cancers (ACS, 1999a). Meningiomas are the most common type of tumors that occur from this type of exposure, but gliomas may also occur (Preston-Martin and Mack, 1996). Among adults, the risk of developing meningiomas has been associated with full-mouth dental x-rays taken decades ago when radiation doses were higher than today. Although the relationship between low-dose radiation exposure and increased risk of brain tumors has been debated in several studies, prenatal exposure from diagnostic x-rays has been related to an increase in childhood brain tumors (Preston-Martin and Mack, 1996).

In recent years, there has been increasing public concern and scientific interest regarding the relationship of electromagnetic fields (EMF) to brain cancer. However, results from recent epidemiological investigations provide little or no evidence of an association between residential EMF exposure (e.g., from power lines and home appliances) and brain tumors (Kheifets, 2001). Studies also suggest that the use of handheld cellular telephones is not associated with an increased risk of primary brain cancer

(Muscat et al., 2000). However, given the relatively recent use of cellular phones, evidence is preliminary and few studies have been conducted.

Other environmental factors such as exposure to vinyl chloride (used in the manufacturing of some plastics) and aspartame (a sugar substitute) have been suggested as possible risk factors for brain cancer but no conclusive evidence exists implicating these factors (ACS, 1999a). Although some occupational studies have suggested that electrical and electric utility workers may be at a slightly increased risk of brain cancer, these studies have important limitations, such as exposure misclassifications and a lack of dose-response relationships (Kheifets, 2001). Some researchers have also reported an increased risk of brain tumors in adults among veterinarians and farmers. Exposure to farm animals and pets have been considered as possible risk factors because of their association with bacteria, pesticides, solvents, and certain animal oncogenic (cancer-related) viruses (Yeni-Komshian and Holly, 2000). However, the relationship between farm life and brain cancer remains controversial.

Recent reports have proposed a link between occupational exposure to lead and brain cancer risk, but further analytic studies are warranted to test this hypothesis (Cocco et al., 1998). In a recent case-control study, the concentrations of metal and non-metal compounds in brain biopsies from patients with primary brain tumors were compared to results from an analysis of tumor-free brain tissue. Statistically significant associations were observed between the presence of brain tumors and the concentrations of silicon, magnesium, and calcium (Hadfield et al., 1998). However, further research using a larger sample size is needed to determine whether exposure to these elements plays a role in the development of brain cancer. Other occupations that may be associated with elevated risks include workers in certain health professions (e.g., pathologists and physicians), agricultural workers, workers in the nuclear industry, and workers in the rubber industry, although specific exposures have not been established (Preston-Martin and Mack, 1996). Studies investigating the possible association between occupational exposure of parents (in particular, paper or pulp-mill, aircraft, rubber, metal, construction, and electric workers) and the onset of brain tumors in their children have provided inconsistent results (Preston-Martin and Mack, 1996).

The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A recent study concluded that an increased risk of pediatric brain tumor may be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin, 2001). However, the role of nitrites and cured meats in the development of brain cancer remains controversial (Blot et al., 1999; Bunin, 2000). Because most people have continuous, low level exposure to N-nitroso compounds throughout their lives, further studies, especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin, 1996).

Injury to the head has been suggested as a possible risk factor for later development of brain tumors but most researchers agree that there is no conclusive evidence for an association (ACS, 1999b). Head trauma is most strongly associated with the development of meningiomas compared with other types of brain tumor. Several studies have found an increased risk in women with histories of head trauma; in men who boxed; and in men with a previous history of head injuries. Gliomas are the most common type of childhood brain tumor and have been positively associated with trauma at birth (e.g., Cesarean section, prolonged labor, and forceps delivery). However, other studies have found no association (Preston-Martin and Mack, 1996).

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-

Martin and Mack, 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS, 1999b). Of those afflicted with the disease, about 5-10% will develop a central nervous system tumor (Preston-Martin and Mack, 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS, 1999a). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack, 1996).

Other possible risk factors investigated for brain cancer have included alcohol consumption, use of barbiturates, smoking and exposure to second-hand smoke, pesticides, and infectious diseases (i.e., tuberculosis and chicken pox). To date, studies on these risk factors have yielded inconclusive results. Further, the majority of individuals diagnosed with brain cancer have no known risk factors (ACS, 1999a).

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Breast Cancer

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the North American Association of Central Cancer Registries, female breast cancer incidence in Massachusetts is the fifth highest among all states (Chen et al, 2000). Although during the 1980s breast cancer in the United States increased by about 4% per year, the incidence has leveled off to about 110.6 cases per 100,000 (ACS 2000). A similar trend occurred in Massachusetts and there was even a slight decrease in incidence (1%) between 1993 and 1997 (MCR 2000).

In the year 2005, approximately 211,240 women in the United States will be diagnosed with breast cancer (ACS 2005). Worldwide, female breast cancer incidence has increased, mainly among women in older age groups whose proportion of the population continues to increase as well (van Dijck, 1997). A woman's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon the well-established risk factors for breast cancer. These include increased age, an early age at menarche (menstruation) and/or late age at menopause, late age at first full-term pregnancy, family history of breast cancer, and high levels of estrogen. Other risk factors that may contribute to a woman's risk include benign breast disease and lifestyle factors such as diet, body weight, lack of physical activity, consumption of alcohol, and exposure to cigarette smoke. Data on whether one's risk may be affected by exposure to environmental chemicals or radiation remains inconclusive. However, studies are continuing to investigate these factors and their relationship to breast cancer.

Family history of breast cancer does affect one's risk for developing the disease. Epidemiological studies have found that females who have a first-degree relative with premenopausal breast cancer experience a 3-fold greater risk. However, no increase in risk has been found for females with a first degree relative with postmenopausal breast cancer. If women have a first-degree relative with bilateral breast cancer (cancer in both breasts) at any age then their risk increases five-fold. Moreover, if a woman has a mother, sister or daughter with bilateral premenopausal breast cancer, their risk increases nine fold. (Broeders and Verbeek, 1997). In addition, twins have a higher risk of breast cancer compared to non-twins (Weiss et al, 1997).

A personal history of benign breast disease is also associated with development of invasive breast cancer. Chronic cystic or fibrocystic disease is the most commonly diagnosed benign breast disease. Women with cystic breast disease experience a 2-3 fold increase in risk for breast cancer (Henderson et al, 1996).

According to recent studies, approximately 10% of breast cancers can be attributed to inherited mutations in breast cancer related genes. Most of these mutations occur in the BRCA1 and BRCA2 genes. Approximately 50% to 60% of women who inherit BRCA1 or BRCA2 gene mutations will develop breast cancer by the age of 70 (ACS 2001).

Cumulative exposure of the breast tissue to estrogen and progesterone hormones may be one of the greatest contributors to risk for breast cancer (Henderson et al, 1996). Researchers suspect that early exposures to a high level of estrogen, even during fetal development, may add to one's risk of developing breast cancer later in life. Other studies have found that factors associated with increased levels of estrogen (i.e., neonatal jaundice, severe prematurity, and being a fraternal twin) may contribute to an elevated risk of developing breast cancer (Ekbom et al, 1997). Conversely, studies have revealed that women whose mothers experienced toxemia during pregnancy (a condition associated with low levels of estrogen) had a significantly reduced risk of developing breast cancer. Use of estrogen replacement therapy is another factor associated with increased hormone levels and it has been found to confer a modest (less than two-fold) elevation in risk when used for 10-15 years or longer (Kelsey, 1993).

Similarly, more recent use of oral contraceptives or use for 12 years or longer seems to confer a modest increase in risk for bilateral breast cancer in premenopausal women (Ursin et al, 1998).

Cumulative lifetime exposure to estrogen may also be increased by certain reproductive events during one's life. Women who experience menarche at an early age (before age 12) have a 20% increase in risk compared to women who experience menarche at 14 years of age or older (Broeders and Verbeek, 1997; Harris et al, 1992). Women who experience menopause at a later age (after the age of 50) have a slightly elevated risk for developing the disease (ACS 2001). Furthermore, the increased cumulative exposure from the combined effect of early menarche and late menopause has been associated with elevated risk (Lipworth, 1995). In fact, women who have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than women with 30 years or less of menstrual activity (Henderson et al, 1996). Other reproductive events have also shown a linear association with risk for breast cancer (Wohlfahrt, 2001). Specifically, women who gave birth for the first time before age 18 experience one-third the risk of women who have carried their first full-term pregnancy after age 30 (Boyle et al, 1988). The protective effect of earlier first full-term pregnancy appears to result from the reduced effect of circulating hormones on breast tissue after pregnancy (Kelsey, 1993).

Diet, and particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, a hypothesis exists that the type of fat in a woman's diet may be more important than her total fat intake (ACS 1998; Wynder et al, 1997). Monounsaturated fats (olive oil and canola oil) are associated with lower risk while polyunsaturated (corn oil, tub margarine) and saturated fats (from animal sources) are linked to an elevated risk. However, when factoring in a woman's weight with her dietary intake, the effect on risk becomes less clear (ACS 1998). Many studies indicate that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey, 1993), probably due to fat tissue as the principal source of estrogen after menopause (McTiernan, 1997). Therefore, regular physical activity and a reduced body weight may decrease one's exposure to the hormones believed to play an important role in increasing breast cancer risk (Thune et al, 1997).

Aside from diet, regular alcohol consumption has also been associated with increased risk for breast cancer (Swanson et al, 1996; ACS 2001). Women who consumed one alcoholic beverage per day experienced a slight increase in risk (approximately 10%) compared to non-drinkers, however those who consumed 2 to 5 drinks per day experienced a 1.5 times increased risk (Ellison et al., 2001; ACS 2001). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al, 1996).

To date, no specific environmental factor, other than ionizing radiation, has been identified as a cause of breast cancer. The role of cigarette smoking in the development of breast cancer is unclear. Some studies suggest a relationship between passive smoking and increased risk for breast cancer; however, confirming this relationship has been difficult due to the lack of consistent results from studies investigating first-hand smoke exposure (Laden and Hunter, 1998).

Studies on exposure to high doses of ionizing radiation demonstrate a strong association with breast cancer risk. These studies have been conducted in atomic bomb survivors from Japan as well as patients that have been subjected to radiotherapy in treatments for other conditions (i.e., Hodgkin's Disease, non-Hodgkin's Lymphoma, tuberculosis, post-partum mastitis, and cervical cancer) (ACS 2001). However, it has not been shown that radiation exposures experienced by the general public or people living in areas of high radiation levels, from industrial accidents or nuclear activities, are related to an increase in breast cancer risk (Laden and Hunter, 1998). Investigations of electromagnetic field exposures in relation to breast cancer have been inconclusive as well.

Occupational exposures associated with increased risk for breast cancer have not been clearly identified. Experimental data suggests that exposure to certain organic solvents and other chemicals (e.g., benzene, trichloropropane, vinyl chloride, polycyclic aromatic hydrocarbons (PAHs)) causes the formation of breast tumors in animals and thus may contribute to such tumors in humans (Goldberg and Labreche, 1996). Particularly, a significantly elevated risk for breast cancer was found for young women employed in solvent-using industries (Hansen, 1999). Although risk for premenopausal breast cancer may be elevated in studies on the occupational exposure to a combination of chemicals, including benzene and PAHs, other studies on cigarette smoke (a source of both chemicals) and breast cancer have not shown an associated risk (Petralia et al, 1999). Hence, although study findings have yielded conflicting results, evidence does exist to warrant further investigation into the associations.

Other occupational and environmental exposures have been suggested to confer an increased risk for breast cancer in women, such as exposure to polychlorinated biphenyls (PCBs), chlorinated hydrocarbon pesticides (DDT and DDE), and other endocrine-disrupting chemicals. Because these compounds affect the body's estrogen production and metabolism, they can contribute to the development and growth of breast tumors (Davis et al, 1997; Holford et al, 2000; Laden and Hunter, 1998). However, studies on this association have yielded inconsistent results and follow-up studies are ongoing to further investigate any causal relationship (Safe, 2000).

When considering a possible relationship between any exposure and the development of cancer, it is important to consider the latency period. Latency refers to the time between exposure to a causative factor and the development of the disease outcome, in this case breast cancer. It has been reported that there is an 8 to 15 year latency period for breast cancer (Petralia 1999; Aschengrau 1998; Lewis-Michl 1996). That means that if an environmental exposure were related to breast cancer, it may take 8 to 15 years after exposure to a causative factor for breast cancer to develop.

Socioeconomic differences in breast cancer incidence may be a result of current screening participation rates. Currently, women of higher socioeconomic status (SES) have higher screening rates, which may result in more of the cases being detected in these women. However, women of higher SES may also have an increased risk for developing the disease due to different reproductive patterns (i.e., parity, age at first full-term birth, and age at menarche). Although women of lower SES show lower incidence rates of breast cancer in number, their cancers tend to be diagnosed at a later stage (Segnan, 1997). Hence, rates for their cancers may appear lower due to the lack of screening participation rather than a decreased risk for the disease. Moreover, it is likely that SES is not in itself the associated risk factor for breast cancer. Rather, SES probably represents different patterns of reproductive choices, occupational backgrounds, environmental exposures, and lifestyle factors (i.e., diet, physical activity, cultural practices) (Henderson et al, 1996).

Despite the vast number of studies on the causation of breast cancer, known factors are estimated to account for less than half of breast cancers in the general population (Madigan et al, 1995). Researchers are continuing to examine potential risks for developing breast cancer, especially environmental factors.

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Kidney cancer

Kidney cancer involves a number of tumor types located in various areas of the kidney and renal system. Renal cell cancer (which affects the main area of the kidney) accounts for over 90% of all malignant kidney tumors (ACS, 2001). The American Cancer Society estimates that there will be approximately 36,160 cases of kidney and upper urinary tract cancer, resulting in more than 12,660 deaths in 2005 (ACS, 2004). The incidence and mortality from kidney cancer is higher in urban areas, which may be due to increased access to diagnostic services and other factors such as smoking. Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in the fifth and sixth decades of life (50-70 year age group) (ACS, 2001). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

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

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

Virtually every study that has examined body weight and renal cell cancer has observed a positive association. Some studies suggest that obesity is a factor in 20% of people who develop kidney cancer (ACS, 2001). This is especially true among women and researchers suspect that this may be related to changes in certain hormones, such as estrogen in women (ACS, 2001; McLaughlin et al., 1996). A diet high in protein (meat, animal fats, milk products, margarine and oils) has been implicated in epidemiological studies as a risk factor for renal cell carcinoma (ACS, 2001; McLaughlin et al., 1996). Consumption of adequate amounts of fruits and vegetables lowers the risk of renal cell cancer. In addition, use of diuretics and antihypertensive medications are associated with increased risk of renal cell carcinoma. However, hypertension has also been linked to kidney cancer and it is not clear whether the disease or the medications used to treat them is the cause (ACS, 2001). Long-term use of pain relievers such as phenacetin (and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (ACS, 2001).

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al., 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS, 2001; McLaughlin et al., 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers,

and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS, 2001; Linehan et al,. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS, 2001). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al., 1997; McLaughlin et al., 1996).

Wilms' tumor is the most common type of kidney cancer affecting children and accounts for approximately 5% to 6% of all kidney cancers and about 6% of all childhood cancers. This cancer is more common among African Americans than other races and among females than males. Wilms' tumor most often occurs in children under the age of 5 years. The causes of Wilms' tumor are not known, but certain birth defect syndromes and other genetic risk factors (such as family history or genetic mutations) are connected with this cancer. However, most children who develop Wilms' tumor do not have any known birth defects or inherited gene changes. No environmental risk factors, either before or after a child's birth, have been shown to be associated with the development of Wilms' tumor (ACS, 1999).

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Leukemia

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

In 2005, leukemia is expected to affect approximately 34,810 individuals (19,640 males and 15,420 females) in the United States, resulting in 22,570 deaths. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2005, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML and CLL. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority of these cases are of the ALL type (ACS, 2005).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals. The increase in incidence among older individuals begins at approximately 40-50 years of age, peaking at about age 85 (Linet and Cartwright, 1996). ALL is more common among whites than African Americans and among males than females (Weinstein and Tarbell, 1997). Exposure to high-dose radiation (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents) is a known environmental risk factor associated with the development of ALL (Scheinberg et al., 1997). Significant radiation exposure (e.g., diagnostic x-rays) before birth may carry up to a 5-fold increased risk of developing ALL (ACS 2000b). However, few studies report an increased risk of leukemia associated with residing in proximity to nuclear plants or occupational exposure to low-dose radiation (Linet and Cartwright, 1996; Scheinberg et al., 1997). It is unclear whether exposure to electromagnetic fields (EMF) plays a role in the development of ALL, however, most studies to date have found little or no risk (ACS 2000b).

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

Although AML can occur in children (usually during the first two years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS, 2000a and 2000b). This type of leukemia is more common among males than among females but affects African Americans and whites at similar rates (Scheinberg et al., 1997). High-dose radiation exposure (e.g., by survivors of

atomic bomb blasts or nuclear reactor accidents), long-term occupational exposure to benzene, and exposure to certain chemotherapy drugs, especially alkylating agents (e.g., mechlorethamine, cyclophosphamide), have been associated with an increased risk of developing AML among both children and adults (ACS, 2000a and 2000b; Linet and Cartwright, 1996). The development of childhood AML is suspected to be related to parental exposure to pesticides and other chemicals, although findings are inconsistent (Linet and Cartwright, 1996). Recent studies have suggested a link between electromagnetic field (EMF) exposure (e.g., from power lines) and leukemia (Minder and Pfluger, 2001; Schuz et al., 2001). However, there is conflicting evidence regarding EMF exposure and leukemia and it is clear that most cases are not related to EMF (ACS, 2000a; Kleinerman et al., 2000).

Other possible risk factors related to the development of AML include cigarette smoking and genetic disorders. It is estimated that approximately one-fifth of cases of AML are caused by smoking (Scheinberg et al., 1997). Also, a small number of AML cases can be attributed to rare inherited disorders. These include Down's syndrome in children, Fanconi's anemia, Wiskott-Aldrich syndrome, Bloom's syndrome, Li-Fraumeni syndrome, and ataxia telangiectasia (ACS, 2000a and 2000b). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al., 2001).

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

Of all the leukemias, CML is among the least understood. While this disease can occur at any age, CML is extremely rare in children (about 2% of leukemias in children) and the average age of diagnosis is 40 to 50 years (ACS 1999). Incidence rates are higher in males than in females, but unlike the other leukemia types, rates are higher in blacks than in whites in the United States (Linet and Cartwright, 1996). High-dose radiation exposure may increase the risk of developing CML (ACS, 1999). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell, 1997).

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Liver Cancer

An estimated 17,550 people in the UNITED STATES (12,130 men and 5,420 women) will be diagnosed with liver cancer in 2005, accounting for approximately 1% of all new cancers (ACS, 2005). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Rarer forms of malignant liver cancer include cholangiocarcinomas, angiosarcomas, and hepatoblastomas in children. Although HCC is approximately ten times more common in developing countries in East and Southeast Asia and Africa, incidence is rapidly increasing in the United States (ACS, 2001). Rates of HCC in the UNITED STATES have increased by 70% over the past two decades (Yu et al., 2000). Similar trends have been observed in Canada and Western Europe. The primary reason for the higher rates observed in recent years is the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag, 2001; El-Serag and Mason, 2000). Men are at least two to three times more likely to develop liver cancer than women (Yu et al., 2000). Incidence rates are also higher among African Americans than whites. Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn, 1996).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS, 2001). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al., 2000). However, HBV accounts for only about a quarter of the cases in the UNITED STATES and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS, 2001).

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

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan, 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn, 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands, 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan, 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS, 2001; London and McGlynn, 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now

strictly regulated in the UNITED STATES (ACS, 2001). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS, 2001; ATSDR, 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS, 2001; London and McGlynn, 1996). Long-term anabolic steroid use may slightly increase the risk of HCC; however, a definitive relationship has not been established (ACS, 2001; London and McGlynn, 1996). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al., 2000; London and McGlynn, 1996).

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Lung Cancer

Lung cancer generally arises in the epithelial tissue of the lung. Several different histologic or cell types of lung cancer have been observed. The various types of lung cancer occur in different regions of the lung and each type is associated with slightly different risk factors (Blot and Fraumeni 1996). The most common type of lung cancer in the United States today is adenocarcinoma which accounts for about 40% of all lung cancers (ACS, 2000). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Lung cancer is very rare in people under the age of 40. The incidence is greater among men than women (probably because men are more likely to be smokers than women) and among blacks than whites (Blot and Fraumeni, 1996). The American Cancer Society estimates that lung cancer will be diagnosed in 172,570 people in the UNITED STATES in 2005, accounting for about 13% of all cancers (ACS, 2005). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS, 2000). In Massachusetts, incidence rates in 1997 were 76.7 per 100,000 and 49.2 per 100,000 for males and females, respectively (MCR, 2000). Nationwide, the incidence rate declined significantly in men during the 1990s, most likely as a result of decreased smoking rates over the past 30 years. Rates for women have continued to increase, but at a much slower pace and have begun to level off. This is because decreasing smoking patterns among women have lagged behind those of men (ACS, 2005). Trends in lung cancer incidence suggest that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals have higher rates of smoking than individuals of other groups (Blot and Fraumeni 1996).

More than 80% of all lung cancers are caused directly by smoking cigarettes and many of the rest are due to exposure to second hand smoke, or environmental tobacco smoke. The longer a person has been smoking and the higher the number of cigarettes smoked per day, the greater the risk of lung cancer. Smoking cessation decreases the elevated risk by about 50%, however, former smokers still carry a greater risk than those who have never smoked (ACS, 2000).

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

As noted above, exposure to radon (a naturally occurring radioactive gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer among miners. Recently, a number of studies have demonstrated that exposure to elevated levels of residential radon may also increase lung cancer risk (Lubin and Boice, 1997; Kreienbrock et al., 2001; Tomasek et al., 2001).

Epidemiological evidence suggests that radon may be the second leading cause of lung cancer after smoking (Samet and Eradze, 2000). However, actual lung cancer risk is determined by cumulative lifetime exposure to indoor radon. Therefore, normal patterns of residential mobility suggest that most people living in high-radon homes experience lifetime exposures equivalent to residing in homes with lower radon levels (Warner et al., 1996).

Tuberculosis and some types of pneumonia may increase the risk of lung cancer due to scarred lung tissue (ACS, 2000). In addition, people who have had lung cancer have a higher risk of developing another tumor. A family history of lung cancer may also slightly increase the risk, however, it is unclear whether this is due to inherited factors or environmental tobacco smoke (ACS, 2000).

Air pollution may increase the risk of developing lung cancer, however, this risk is much lower than that due to cigarette smoking (ACS, 2000).

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

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Non-Hodgkin's Lymphoma

Lymphomas are cancers involving the cells of the lymphatic system. The majority of lymphomas involve the lymph nodes and spleen but the disease may also affect other areas within the body. Non-Hodgkin's lymphoma (NHL) is a classification of all lymphomas except Hodgkin's disease. Thus NHL is a mixed group of diseases that is characterized by the malignant increase in specific cells of the immune system (B or T lymphocytes). B-cell lymphomas are more common than T-cell lymphomas, accounting for about 85% of all cases of NHL (ACS, 2003). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller, 1996). NHL can occur at all ages, however, the average age at diagnosis is in the early 60s and the incidence of this disease generally increases with age. This disease is more common in men than in women and affects whites more often than African Americans or Asian Americans (ACS, 2003). The American Cancer Society estimates that approximately 56,390 Americans will be diagnosed with NHL in 2005, making it the fifth most common cancer in the United States among women and the sixth most common cancer among men, excluding non-melanoma skin cancers (ACS, 2005).

Overall, between 1973 and 1997, the incidence of NHL in the UNITED STATES grew 81% (Garber, 2001), although during the 1990s, the rate of increase appears to have stabilized (ACS, 2005). In Massachusetts, the incidence of NHL increased 50% during 1982-1997 from 10.5 cases per 100,000 to 15.7 cases per 100,000 (MCR, 1997 and 2000). The increase in NHL incidence has been attributed to better diagnosis, greater exposure to causative agents, and, to a lesser extent, the increasing incidence of AIDS-related lymphomas (Devesa and Fears, 1992; Scherr and Mueller, 1996). Although the primary factors related to the development of NHL include conditions that suppress the immune system, viral infections, and certain occupational exposures, these factors are thought to account for only a portion of the increase observed in this cancer type (Scherr and Mueller, 1996). The observation that the rate of increase is declining for NHL may be attributed in part to increased use of antiretroviral therapy to slow HIV progression (Wingo et al., 1998).

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

Several viruses have been shown to play a role in the development of NHL. Among organ transplant recipients, suppression of the immune system required for acceptance of the transplant leads to a loss of control or the reactivation of viruses that have been dormant in the body (e.g., Epstein-Barr Virus [EBV] and herpesvirus infections). In addition, because cancer-causing viruses are known to cause lymphomas in various animals, it has been proposed that these types of viruses may also be associated with the development of NHL among humans without compromised immune systems. Infection with the human T-cell leukemia/lymphoma virus (HTLV-I) is known to cause T-cell lymphoma among adults. However, this is a relatively rare infection and most likely contributes only a small amount to the total incidence of NHL (Scherr and Mueller, 1996). EBV infection is common among the general population and has been shown to play a role in the development of most cases of transplant and AIDS related NHL. The combination of

immune system deficiencies and EBV infection may cause some people to develop NHL (ACS, 2003). Although viruses are causal factors for some subtypes of NHL, to date, studies have shown that the role of EBV in the development of NHL in the general population may not be large (Scherr and Mueller, 1996). Moreover, the high prevalence of EBV in the general population suggests that EBV may be only one of several factors in the development of this cancer.

Recent studies have found that a type of bacteria, *Helicobacter pylori*, a common cause of stomach ulcers, can also cause some lymphomas of the stomach (ACS, 2003). An important implication of this finding is that treatment with antibiotics could prevent some NHL of the stomach.

Some occupations have been associated with an increased risk of developing NHL, such as occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm, 1990 and 1993; Tatham et al., 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and organophosphate insecticides (Wigle et al., 1990; Zahm et al., 1990; Zahm et al., 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD alone have not demonstrated an increased risk of NHL (Scherr and Mueller, 1996). An elevated risk for NHL development has also been noted among fence workers, orchard workers, and meat workers. High-dose exposure to benzene has been associated with NHL (ACS, 2003), however, a recent international cohort study indicated that petroleum workers exposed to benzene were not at an increased risk of NHL (Wong and Raabe, 2000).

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

Although radiation (e.g., nuclear explosions or radioactive fallout from reactor accidents) has been implicated in the development of some cancers, including NHL (ACS, 2003), there is little evidence for an increased risk of lymphoma due to radiation (Scherr and Mueller, 1996).

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

Smoking has also been suggested to increase the risk of NHL. A study that evaluated the history of tobacco use and deaths from NHL determined that people who had ever smoked had a two-fold increase of dying from NHL as compared to those who never smoked. Further, a four-fold increase was found among the heaviest smokers (Linet et al., 1992). In addition, a more recent study that primarily examined occupation and NHL risk found a significant association with high levels of cigarette smoking and all NHL types (Tatham et al., 1997). However, a recent review of 5 cohort studies and 14 case-control studies concludes that results of epidemiological studies have been inconsistent and that smoking has not been determined to be a definitive risk factor in the development of NHL (Peach and Barnett, 2000).

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

Although NHL is associated with a number of risk factors, the causes of this disease remain unknown. Most patients with NHL do not have any known risk factors (ACS, 2003).

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Appendix C: ATSDR Glossary of 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 UNITED STATES Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

General Terms

Absorption
The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Anaerobic
Requiring the absence of oxygen [compare with aerobic].

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP [see Community Assistance Panel.]

Cancer
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

CERCLA [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure]

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)
A group of people from a community and from health and environmental agencies who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Delayed health effect
A disease or an injury that happens as a result of exposures that might have occurred in the past.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see route of exposure].

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease prevention
Measures used to prevent a disease or reduce its severity.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

DOD
United States Department of Defense.

DOE
United States Department of Energy.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction
A method of estimating the amount of people's past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Half-life (t½)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remain.

Hazard
A source of potential harm from past, current, or future exposures.

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to evaluate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.

Health statistics review
The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

mg/cm2
Milligram per square centimeter (of a surface).

mg/m3
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, a condition, or an injury) is stated.

Mutagen
A substance that causes mutations (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see National Priorities List for Uncontrolled Hazardous Waste Sites]

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevalence survey
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public availability session
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might

be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Public health statement
The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public health surveillance
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [see Resource Conservation and Recovery Act (1976, 1984)]

Receptor population
People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)
This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA
RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or an environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Stakeholder
A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Surveillance [see public health surveillance]

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen
A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)

National Center for Environmental Health (CDC)
(http://www.cdc.gov/nceh/dls/report/glossary.htm)

National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

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