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AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Evaluation of Environmental Concerns Related to the Barnes Aquifer and Cancer Incidence, 1982 - 2000

Easthampton & Southampton, Hampshire County, Massachusetts
Holyoke & Westfield, Hampden County, Massachusetts

MassDEP RTN 1-13737, 1-11888, 1-13735, 1-13736

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

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

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

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Prepared by:

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Bureau of Environmental Health
Community Assessment Program
Boston, Massachusetts
Under a Cooperative Agreement with:
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I. SUMMARY

At the request of the Southampton Board of Health and in response to a legislative directive, the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), Community Assessment Program (CAP) conducted an evaluation of possible environmental exposures and cancer incidence in the Barnes Aquifer region, which includes areas of Southampton, Easthampton, Holyoke, and Westfield in western Massachusetts. In the 1950s, trichloroethylene (TCE) wastes were released at two Holyoke residential properties and the former Southampton Sanitary Engineering in Southampton. Polychlorinated biphenyl (PCB) wastes were released at the two Holyoke residential properties. This evaluation was initiated in response to community concerns about possible environmental exposures in relation to TCE contamination in public and private drinking water wells whose source is the Barnes Aquifer and community concerns about cancer. Community concerns also included possible exposures to PCBs in soil, PCBs and benzene in private well drinking water, and dioxins in air.

This public health assessment provides a review of available environmental data for Barnes Aquifer drinking water and soil near the identified sources of TCE. It also considers potential ways people may have come in contact with the released contaminants and evaluates the pattern of cancer diagnoses in Easthampton, Holyoke, Southampton, and Westfield with a particular focus on neighborhoods where residents could have been exposed to TCE.

In the past, Easthampton residents and some western Holyoke residents were at risk of exposure to TCE in municipal drinking water from the Barnes Aquifer. Some residents of western Holyoke and eastern Southampton were at risk of exposure to TCE in drinking water from private wells that draw from the Barnes Aquifer. Based on the contaminant levels detected since 1980 in municipal wells and since 1997 in private wells, the frequency and duration of contact assumed, and a review of the scientific literature, it is unlikely that exposures to TCE in Barnes Aquifer drinking water resulted in adverse health effects.

Currently, residents are not at risk of exposure to TCE in municipal water. Holyoke and Southampton residents with TCE-contaminated private well water are no longer at risk of exposure if they properly maintain whole house charcoal filters or connected their households to municipal water not impacted by environmental contaminants. However, a potential exposure

pathway could remain if residents with private wells within the extent of groundwater contamination do not properly maintain their filters or if they use unfiltered water.

Children who lived and/or may have played in surface soil at the two Holyoke residences may have been at risk of exposure to PCBs; however, based on the levels detected and the frequency and duration of contact assumed, it is unlikely that potential exposures could have resulted in adverse health effects. The contaminated soils have since been removed. Potential exposures to PCBs in private well water were ruled out because PCBs have not been demonstrated to have migrated via groundwater from the release areas.

Although it is unlikely that exposures to TCE in Barnes Aquifer drinking water resulted in adverse health effects, a review of cancer incidence data was conducted to address community concerns. Using data from the Massachusetts Cancer Registry (MCR), incidence rates for eight cancer types were calculated for Easthampton, Holyoke, Southampton, and Westfield, as well as for specific census tracts (CTs) where some of the residents were at risk of exposure to TCE in drinking water. The eight cancer types (Hodgkin's disease, leukemia, non-Hodgkin's lymphoma, and cancers of the bladder, esophagus, kidney, liver, and pancreas) were selected based on potential associations with TCE and residents' concerns about particular types. Cancer incidence data were evaluated from 1982 to 2000, the most recent and complete data available at the time of the analysis, and three shorter time periods (1982–1987, 1988–1993, and 1994–2000). Available information about risk factors related to the development of cancer was also analyzed.

Although there were some statistically significant elevations observed during some time periods, no consistent trends were observed for any of the eight cancer types. A review of the geographic distribution of cancer diagnoses revealed no apparent spatial patterns at the neighborhood level. Further, no unusual concentrations of cancer diagnoses were observed in areas where residents were at risk of exposure to TCE or in any other area of the four communities.

In Easthampton CT 8223, non-Hodgkin's lymphoma (NHL) among males was statistically significantly elevated from 1994 to 2000. The histological types of NHL were consistent with the statewide distribution of NHL, and no unusual geographic concentrations of diagnoses were observed at the neighborhood level. There was a statistically significant elevation in pancreatic

cancer among males and females townwide and males in Easthampton CT 8224 from 1994 to 2000. However, based on a review of available risk factor information, smoking may have played a role in some individuals' diagnoses.

In Holyoke CT 8121, males were diagnosed with leukemia statistically significantly more often than expected from 1994 to 2000. Based on the location of their residences at the time of diagnosis, none of the 14 males were at risk of exposure to TCE in drinking water from the Barnes Aquifer. Pancreatic cancer was statistically significantly elevated for females from 1988 to 1993. Of the 12 females, 11 were not at risk of exposure to TCE from the Barnes Aquifer, based on their residences. It is unknown whether the remaining individual could have been exposed to TCE.

In Southampton, a statistically significant elevation in bladder cancer among males from 1982 to 2000 was attributed to elevations during two time periods, 1982–1987 and 1988–1993. Ten of the 14 males diagnosed from 1982 to 1993 were not at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences. While the residences of three of the 14 males were located within the extent of contaminated groundwater, it is unknown whether these males could have been exposed to TCE. The remaining individual could have been exposed to TCE; therefore, if exposure did occur, it could have played a role in the development of bladder cancer. Based on a review of available risk factor information, it is likely that smoking played a role in the development of bladder cancer among some of the males.

In Westfield CT 8125, bladder cancer was statistically significantly elevated for males and females from 1982 to 2000. This was largely due to elevations among males that were not statistically significant during the three smaller time periods. Of the 22 males diagnosed with bladder cancer, 21 were not at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences. It is unknown whether the remaining individual could have been exposed to TCE. Based on a review of available risk factor information, it is likely that smoking played a role in the individual's diagnosis. Based on their residences, none of the six females diagnosed with Hodgkin's disease, which was statistically significantly elevated from 1982 to 2000, were at risk of exposure to TCE from the Barnes Aquifer.

Residents living in the Dupuis Road neighborhood in Holyoke could have been at risk of exposure to air contaminants when PCB wastes were reportedly burned at one of the residential properties where PCBs were released. Because no air monitoring data were available for that time, it was not possible to quantitatively evaluate the potential for adverse health effects. However, a qualitative review of cancer diagnoses in the Dupuis Road neighborhood revealed no unusual pattern or concentration of diagnoses.

Based on the MDPH evaluation of available environmental data, exposure pathways, and available risk factor information related to the cancer types evaluated, the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) would classify the TCE-contaminated section of the Barnes Aquifer as posing an indeterminate public health hazard in the past due to incomplete historical sampling data for private wells prior to 1997. Most exposure opportunities have been eliminated through municipal water treatment and well closures, whole house filters, and connections to municipal water not impacted by contaminants; however, for some residents with private wells (i.e., residents of a few households that declined testing, residents who might not properly maintain their filters, and residents who use unfiltered water), the ATSDR would classify the contaminated section of the Barnes Aquifer as posing an indeterminate public health hazard presently or in the future.

In all, the information reviewed and analyzed for this public health assessment included available environmental data, cancer incidence data, available risk factor information for individuals diagnosed with cancer, residential history information, and a review of the relevant scientific literature. Based on this information, it does not appear that a common factor (environmental or non-environmental) played a major role in the overall incidence of cancer in the census tracts where some residents were at risk of exposure to TCE from the Barnes Aquifer or in the communities of Easthampton, Holyoke, Southampton, and Westfield as a whole during the 19-year time period, 1982–2000.

II. INTRODUCTION AND STATEMENT OF ISSUES

At the request of the Southampton Board of Health and in response to a legislative directive, the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), Community Assessment Program (CAP) conducted an evaluation of possible environmental exposures and cancer incidence in the Barnes Aquifer region, which includes areas of the towns of Southampton and Easthampton and the cities of Holyoke and Westfield in western Massachusetts. In the 1950s, trichloroethylene (TCE) wastes were released at two Holyoke residential properties and the former Southampton Sanitary Engineering in Southampton. Polychlorinated biphenyl (PCB) wastes were released at the two residential properties in Holyoke. The Massachusetts Department of Environmental Protection (MassDEP) believes that the wastes at two of the release locations originated from the former General Electric facility on Jackson Street in Holyoke. TCE contamination now extends approximately 4.5 miles through the Barnes Aquifer from the identified sources north to municipal wells in Easthampton. This evaluation was initiated in response to community concerns about cancer and possible environmental exposure in relation to TCE contamination in public and private drinking water whose source is the Barnes Aquifer. Community concerns also included possible exposures to polychlorinated biphenyls (PCBs) in soil, PCBs and benzene in private well drinking water, and dioxins in air from the burning of PCB wastes. This project was conducted under a cooperative agreement with the United States Agency for Toxic Substances and Disease Registry (ATSDR) for the MDPH to conduct public health assessments in Massachusetts. Refer to Figure 1 for the location of the four communities included in the evaluation.

This report provides a review of potential exposure pathways to contaminants, particularly TCE, in the Barnes Aquifer and a review of the pattern of cancer in Easthampton, Holyoke, Southampton, and Westfield through comparison of the incidence of eight cancer types with the incidence of these cancers in the state of Massachusetts as a whole. There was a particular focus on cancer incidence in census tracts and neighborhoods where some of the residents were at risk of exposure to TCE-contaminated drinking water from the Barnes Aquifer. 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 Barnes Aquifer drinking water, the MDPH contacted the MassDEP and

municipal water departments in the four communities to obtain and review available environmental data.

Cancer incidence rates were evaluated for the towns of Easthampton and Southampton and the cities of Holyoke and Westfield for 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 public health assessment. Cancer incidence rates were also evaluated for the census tracts that include residents who were at risk of exposure to TCE-contaminated drinking water from the Barnes Aquifer (some of the census tracts also include residents who were not at risk of exposure to TCE from the Barnes Aquifer). 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 incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated.

Easthampton is divided into three smaller geographic areas, or census tracts (CTs). The city of Holyoke comprises nine census tracts, the town of Southampton is one census tract, and the city of Westfield is divided into eight census tracts. The five census tracts that are the focus of this evaluation are Easthampton CT 8223 and CT 8224, Holyoke CT 8121, Southampton CT 8225, and Westfield CT 8125. The total population of the five census tracts combined is 42,410 (U.S. DOC 2000). The location and boundaries of the five census tracts, along with the estimated extent of TCE contamination in groundwater according to the MassDEP, are shown in Figure 2. It is important to note that the actual extent of TCE-contaminated groundwater may be larger than the extent depicted in Figure 2 (C. Chamberlain, MassDEP, personal communication, 2006).

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, to determine whether a common risk factor is possible, and can serve to identify areas where further public health investigations or other 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 non-environmental) and the development of cancer. The purpose of this evaluation is to report the findings on the patterns of cancer in the Barnes Aquifer region of Easthampton, Holyoke, Southampton, and Westfield and evaluate the findings 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) of residents to contaminants in drinking water from the Barnes Aquifer;

- To evaluate the incidence rates of eight cancer types (Hodgkin's disease, leukemia, non-Hodgkin's lymphoma and cancers of the bladder, esophagus, kidney, liver, and pancreas) in Easthampton, Holyoke, Southampton, and Westfield and in the census tracts where some residents were at risk of exposure to contaminated drinking water to determine if cancer occurred more or less often than expected;

- To evaluate the geographic distribution of the residences of individuals diagnosed with cancer in the four communities and see if there are any patterns in geographic areas within the communities, particularly in areas of potential environmental concern;

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

- To discuss possible exposure pathways related to the Barnes Aquifer and the results of the cancer incidence 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. BACKGROUND AND COMMUNITY ENVIRONMENTAL CONCERNS

Community environmental concerns focus largely on the historical presence of trichloroethylene (TCE) in drinking water from the Barnes Aquifer. TCE is a solvent that has wide industrial uses. It is most commonly used as a metal degreaser and has uses in metal finishing, textile manufacturing, rubber processing, paint and ink formulation, dry cleaning, and electronics manufacturing. TCE does not adsorb strongly to soil and is mildly soluble in water; therefore, TCE leaches quickly into groundwater upon release to soil. Because of these chemical properties and its many industrial uses, TCE is a common contaminant in groundwater that may impact drinking water sources. The ATSDR has estimated that 9% to 34% of drinking water sources in the United States contain some level of TCE (ATSDR 1997).

The Barnes Aquifer extends for 12 miles under parts of Easthampton, Holyoke, Southampton, and Westfield. It supplies drinking water to over 60,000 people in the area (EWW 2001). Because it supplies more than 50% of the drinking water for Easthampton, a community that has no viable alternative sources, the Broad Brook Basin of the Barnes Aquifer was designated a Sole Source Aquifer by the United States Environmental Protection Agency (U.S. EPA) in 1995.

In order to address community environmental concerns about TCE contamination in the Barnes Aquifer, the MDPH contacted the Western Regional Office of the MassDEP in Springfield, Massachusetts, Easthampton Water Works, Holyoke Water Works, the Southampton Water Department, and the Westfield Water Department to obtain and review available environmental information pertaining to groundwater in the aquifer and soil at the three properties where TCE was released. In addition, information regarding other potential environmental sources located in the area and listed with the MassDEP as Chapter 21E sites was reviewed (MassDEP 2005).

The public health assessment titled “Evaluation of Environmental Concerns Related to the Barnes Aquifer and Cancer Incidence, 1982–2000” was released on July 5, 2007, for a 30-day public comment period. The MDPH received comments from the MassDEP during the public comment period. Their comments, which were editorial in nature to clarify MassDEP’s source investigation efforts, were incorporated in this document.

A. Massachusetts Department of Environmental Protection Source Investigation

The MassDEP Site Discovery Program prioritized the task of identifying the source of TCE contamination in the Barnes Aquifer in 1994 (Pine and Swallow, Inc. 2000). Pine and Swallow installed 268 groundwater investigatory wells throughout the Barnes Aquifer region (C. Chamberlain, MassDEP, personal communication, 2004). Some of the investigatory wells were sampled at discrete depth intervals beginning at the water table and ending at bedrock (Pine and Swallow, Inc. 2000). Some of the wells were installed as water table wells only (C. Chamberlain, MassDEP, personal communication, 2004).

The investigation identified two source areas in western Holyoke and one source area in Southampton. TCE and PCBs, reportedly released once in 1955, were detected at a residential property on Apremont Highway in western Holyoke (Figure 2). A residential property on Dupuis Road in western Holyoke was also identified as a source area (Pine and Swallow, Inc. 2000). Disposal and burning of PCB wastes reportedly occurred about every other weekend on this property during the early to mid-1950s (C. Chamberlain, MassDEP, personal communication, 2005). The Potentially Responsible Party (PRP) for the Apremont Highway and Dupuis Road residential properties is General Electric. The MassDEP believes that the wastes originated at the former General Electric facility on Jackson Street in Holyoke. The Southampton Sanitary Engineering property on Pequot Road in Southampton, which once operated as a waste oil recycling facility and possibly received waste generated at the General Electric facility, was identified as the third TCE source area (Figure 2). The MassDEP also determined that a fourth occurrence of TCE in groundwater exists beneath a residential property on Mueller Road in western Holyoke; however, the exact release location associated with this TCE occurrence has not been identified (C. Chamberlain, MassDEP, personal communication, 2004). There is also the possibility that other unidentified sources in the Pequot Pond area might have contributed to the Barnes Aquifer contamination (C. Chamberlain, MassDEP, personal communication, 2005).

The maximum field screen TCE concentration detected at a source location was in an investigatory well [210 parts per billion (ppb)] on Mueller Road in Holyoke where TCE was retarded due to silty material beneath the surface (C. Chamberlain, MassDEP, personal

communication, 2005). The Dupuis Road residential property has low TCE levels remaining in the groundwater. TCE was also detected in the groundwater at Southampton Sanitary Engineering on Pequot Road. Groundwater at the Apremont Highway residential property was not able to be sampled with investigatory wells because the MassDEP investigation did not include boring into bedrock, where the water table is contained. TCE was not detected in the private well at this property; however, TCE was detected in soil, along with PCBs and other constituents of PCB transformer oil (C. Chamberlain, MassDEP, personal communication, 2005, 2007).

TCE and TCE breakdown products were detected in investigatory wells at Southampton Sanitary Engineering and in some investigatory wells downgradient to Southampton Sanitary Engineering, but were not detected in other investigatory wells (C. Chamberlain, MassDEP, personal communication, 2007). PCBs, which adsorb strongly to soil particles and are not highly mobile through soil, were detected in groundwater at only one of the source areas and have not been demonstrated to have migrated via groundwater from any source area (C. Chamberlain, MassDEP, personal communication, 2004). In 1997, benzene was detected above the MassDEP GW-1 standard (5 ppb) in two investigatory wells at Southampton Sanitary Engineering (C. Chamberlain, MassDEP, personal communication, 2006). The maximum concentration detected was 35 ppb.

Figure 2 shows the extent of the TCE contamination that the MassDEP delineated from the results of its investigation. [It is important to note that the actual extent of TCE-contaminated groundwater may be larger than the extent depicted in Figure 2.] Because groundwater in the Barnes Aquifer moves in a northern direction from the northern end of Pequot Pond, contamination from the source areas gradually migrated north to the Pequot Well in Holyoke and the Hendrick Street Wellfield and Pines Well in Easthampton (Figure 3) (Pine and Swallow, Inc. 2000). According to the MassDEP, TCE from the Apremont Highway and Dupuis Road residential properties in western Holyoke could have dispersed through bedrock fractures or sandy material west to Southampton and north to Easthampton (MassDEP 2004b). The Coronet Homes Well in Holyoke, which is south of the source areas, likely pulled the contamination slightly to the south when the wells were drawing water to serve nearby homes (C. Chamberlain, MassDEP, personal communication, 2005). TCE was detected at trace amounts in investigatory

wells south of Pequot Pond (MassDEP 2002). In all, the MassDEP determined that TCE contamination extends for about 4.5 miles within the Barnes Aquifer.

The Apremont Highway and Dupuis Road residential properties, for which General Electric is the PRP, have been remediated under Chapter 21E of the Massachusetts General Laws (C. Chamberlain, MassDEP, personal communication, 2005). PCB-contaminated soil was excavated up to 8 feet deep, until the remaining soil had less than the MassDEP S-1 soil standard of 2 ppm on average, and removed from the properties. Both properties have an Activity and Use Limitation (AUL), a deed restriction that limits the future uses of a property in order to be protective of public health, due to remaining PCB contamination below 8 feet. The Southampton Sanitary Engineering site on Pequot Road is undergoing assessment and response actions according to Chapter 21E and has not yet been remediated.

B. Municipal Water Supply

Easthampton, Holyoke, Southampton, and Westfield obtain their municipal drinking water from a variety of sources. Easthampton draws its municipal water solely from five wells located throughout the town that take water from the northern end of the Barnes Aquifer (Figure 3) (EWW 2001). The Hendrick Street Wellfield and Pines Well are adjacent municipal wells that came online in 1908 and 1962, respectively (MassDEP 2003). While most Easthampton residents received a portion of their drinking water from the Hendrick Street Wellfield and Pines Well, Easthampton residents living in the Plains area of southern Easthampton closest to the two wells received more water from them relative to Easthampton residents farther north (T. Newton, Easthampton Water Works, personal communication, 2005). [For the purposes of this evaluation, the Plains area was defined as the entire southern part of town that is south of the Brook Street Well (Figure 3).] This means that residents in the Plains area could have been at risk of exposure to a higher TCE concentration relative to the rest of Easthampton. Due to the presence of TCE in the Hendrick Street Wellfield and Pines Well (other municipal wells were unaffected), the town disconnected them from the water distribution system between 1987 and 1988 (T. Newton, Easthampton Water Works, personal communication, 2004). Between 1990 and 1991, both wells were blended with other sources in order to lower the TCE concentration and reconnected with the distribution system (T. Newton, Easthampton Water Works, personal

communication, 2004). From that time until 1996, the Pines Well was used continually and the Hendrick Street Wellfield was used infrequently. The town built a treatment plant in 1997 that treats water from both the Hendrick Street Wellfield and the Pines Well to remove TCE before it enters the distribution system. The town continues to use other more northern wells that draw from the Barnes Aquifer and are not contaminated with TCE.

Holyoke relies on surface water reservoirs, mainly the Tighe-Carmody Reservoir in Southampton, for municipal drinking water (HWW 2000). Water from the McLean Reservoir in Holyoke supplements the drinking water supply (HWW 2000). Historically, Holyoke drew some municipal water from the Barnes Aquifer via the Pequot Well and Coronet Homes Well in western Holyoke (Figure 3). In 1974, the Pequot Well was constructed near Winterberry Circle in western Holyoke (B. Seidel, Holyoke Water Works, personal communication, 2002). The well served about 600 Holyoke residents in the area near the well (B. Seidel; Holyoke Water Works; personal communication; 2002, 2005). The Coronet Homes Well was constructed in 1966 to supplement Holyoke's water supply and served about 400 residents in the Coronet Road area (C. Chamberlain, MassDEP, personal communication, 2002; B. Seidel, Holyoke Water Works, personal communication, 2002). In June of 1987, the distribution systems of the Pequot and Coronet Homes wells were connected (HWW 2002). Both wells were closed in December 1987 and are not maintained as emergency or future sources of municipal water (B. Seidel, Holyoke Water Works, personal communication, 2005).

Southampton uses surface water from the Tighe-Carmody Reservoir in Southampton as its primary municipal drinking water source and also draws from the College Highway Well in Southampton (Figure 3) (SWD 2000). The College Highway Well is separated from the main part of the Barnes Aquifer by a solid bedrock mountain range (Gary Swanson, Town of Southampton Moderator, personal communication, 2004).

Westfield obtains the majority of its municipal water from the Granville Reservoir in Granville, Massachusetts, which borders the city to the southwest (WWD 1999). The city also uses six groundwater wells, four of which (Well #1, Well #2, Well #7, and Well #8) draw from the Barnes Aquifer, but are separated by a groundwater divide from the contaminated area of the

aquifer (Figure 3) (C. Darling, Westfield Water Department, personal communication, 2004). The other two wells do not draw from the Barnes Aquifer.

C. Private Wells

The MassDEP investigation into the source of TCE in some Barnes Aquifer municipal wells revealed that TCE existed in some private wells in Southampton, Holyoke, Westfield, and Easthampton in 1997 (MassDEP 2002). In Southampton, TCE was detected in 51 private wells in the Pequot Pond area in the southeast corner of town. About 14 households subsequently connected to municipal water after the contamination was discovered (Gary Swanson, Southampton Water Department, personal communication, 2005). Approximately 200–300 Southampton households in this area continue to use private wells that draw from the Barnes Aquifer (Gary Swanson, Southampton Water Department, personal communication, 2004). In Holyoke, TCE was detected in 27 private wells in the Rock Valley area near Southampton. There are about 250–350 residences in this area that might have used private wells that draw from the Barnes Aquifer (MassDEP 2002). Some households connected to uncontaminated municipal water after TCE was identified in private wells in 1997 (D. Bresnahan, Holyoke Board of Health, personal communication, 2005). Some Holyoke residents on Rock Valley Road, Keyes Road, Mueller Road, and Southampton Road where TCE was detected were not able to connect to municipal water because there is no city water main in the area (C. Chamberlain; MassDEP; personal communication; 2004, 2005). In Westfield, TCE was identified in nine private wells in the Hampton Ponds area in the northeast corner of town. There are about 150–200 households in this general area that might have used private wells in the past. Approximately 20 households in the area of Westfield where TCE was detected connected to municipal water after 1997 (D. Reardon, Westfield Board of Health, personal communication, 2005). In Easthampton, TCE was detected in one private well on Fort Hill Road (MassDEP 2002). The private wells tested in Easthampton are located on Fort Hill Road, about 3 miles north of the approximate northern edge of TCE contamination (C. Chamberlain, MassDEP, personal communication, 2004).

The MassDEP supplied residents who had private wells above about 1 ppb TCE with bottled water as a temporary measure and installed and maintained carbon filters for 2 years (MassDEP

2002, 2004b). A daycare in Southampton with a trace TCE level was also supplied with bottled water. Currently, residents are responsible for maintaining water quality in their private wells.

D. 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 the Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000). Under this legislation, the MassDEP administers investigation and cleanup of hazardous material and oil release sites, known as "21E sites," in the Commonwealth. The MDPH reviewed available information regarding these releases to determine the possibility that environmental exposures could have played a role in the overall incidence of cancer in Easthampton, Holyoke, Southampton, and Westfield.

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. Depending on the relative severity of the release, the deadline for reporting a release to the MassDEP is 2 hours, 72 hours, or 120 days.

The MassDEP Bureau of Waste Site Cleanup has information on hazardous material and oil releases, including assessment and remedial response measures, for 1977 to the present; however, records prior to 1984 are known to contain significant data gaps (MassDEP 2004a). The MDPH obtained the most recent information regarding all hazardous material and/or oil releases (approximately 1,000 records) located in Easthampton, Holyoke, Southampton, and Westfield. The high number of releases in the study area precluded individual examination of each release in relation to patterns of cancer incidence. Therefore, the MDPH focused the analysis on only those releases categorized by 2-hour or 72-hour reporting categories. Releases categorized as 120-day reporting notifications and releases where reporting category information was unavailable were excluded. The 120-day reports are releases thought unlikely to result in human exposure to contaminants.

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

Using a geographic information system, the MDPH mapped the approximate location of 2-hour and 72-hour releases for which sufficient address information was available (ESRI 2005). According to the most current information, from 1985 to 2000, 44 releases were reported in the town of Easthampton; 124 releases were reported in Holyoke; five releases were reported in Southampton; and 125 releases were reported in Westfield (MassDEP 2005a). The majority of these releases could be mapped to an address in one of the four towns (see Figure 4); however, approximately 5% of the releases (n = 14) could not be mapped because sufficient address information was not available.

The majority of the 298 releases reported (62%) involved petroleum-based oil (e.g., gasoline, fuel oil, waste oil) or some combination of oil and another material (either known or unknown). Type of material was unknown for 96 (32%) of the releases. Information specific to each release is provided in Table 1.

The pattern of cancer in Easthampton, Holyoke, Southampton, and Westfield was reviewed in relation to these potential sources of environmental exposures and discussed in Section VII.

V. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with the Barnes Aquifer, the MDPH reviewed information from several reports on file with the MassDEP and municipal water departments in Easthampton, Holyoke, Southampton, and Westfield. 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 identified maximum concentrations of

contaminants detected in various types of environmental media (i.e., air, soil, water) and compared these concentrations to health-based comparison values established by the ATSDR (2005a, 2005c).

For compounds detected in groundwater, maximum concentrations were also compared with state or federal drinking water standards. All public water supplies in Massachusetts are sampled on a regular basis to monitor the quality and ensure the safety of drinking water. It is not unusual to detect some compounds in a drinking water supply. For this reason, the MassDEP has established standards known as Massachusetts Maximum Contaminant Levels (MMCLs) for public drinking water supplies (MassDEP 2004c). These standards dictate the maximum allowable concentration at which a chemical can be present in drinking water. These standards are protective of public health.

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.

A. Municipal Wells

Prior to 1980, volatile organic compound (VOC) testing in public wells occurred when a hazardous waste disposal problem was identified near a well (MDEQE 1980). In 1980, the joint MassDEP and U.S. EPA State Purgeable Organics Testing (SPOT) Program was implemented as

a more formal method of testing for VOCs. The SPOT Program identified the first indication of TCE contamination in the Barnes Aquifer in 1980 at the Pequot Well (Figure 3) in western Holyoke (HWW 2002). Of several VOCs tested for, TCE was detected in seven out of eight samples analyzed between 1980 and 1992, with six samples above the U.S. EPA Maximum Contaminant Level (MCL) and the MassDEP Massachusetts Maximum Contaminant Level (MMCL) of 5 ppb (MassDEP 1988, HWW 2002). The maximum TCE level detected in the Pequot Well was 15.0 ppb in 1984. Table 2 summarizes this sampling information. The wells were shut down in December 1987, and residents were supplied with drinking water from surface water sources in Holyoke (B. Seidel, Holyoke Water Works, personal communication, 2002).

Also in western Holyoke, the SPOT Program collected six groundwater samples from the Coronet Homes Well (Figure 3) between 1980 and 1988 (MassDEP 1988, HWW 2002). No VOCs were detected in 1980. There were no samples collected again until 1984, when TCE (1.7 ppb) was first detected in the well. This was the highest TCE concentration detected in the Coronet Homes Well and was below the MCL. Several other constituents were detected in other rounds of testing, but none were at levels that exceeded comparison values. The TCE sampling data are summarized in Table 3. The well was shut down in December 1987 (B. Seidel, Holyoke Water Works, personal communication, 2002).

In 1980, TCE was not detected in either the Hendrick Street Wellfield or the Pines Well (Figure 3), adjacent Easthampton municipal wells that draw from the Barnes Aquifer (C. Chamberlain, MassDEP, personal communication, 2005). In 1984, the next time the wells were tested by the SPOT Program, TCE was detected at the Hendrick Street Wellfield (3.2 ppb) and the Pines Well (<1 ppb) (EWW 2002). The wells were then monitored several times per year through 2003 for VOCs. At the Hendrick Street Wellfield, TCE was detected in 105 of 127 samples and exceeded the MCL (5 ppb) in 89 samples (Table 4) (EWW 2002, MassDEP 1988, MassDEP 2004a[1]). The maximum concentration of TCE detected at the Hendrick Street Wellfield was 12 ppb in 1991. Based on the available environmental data, the average TCE concentration detected in the wellfield from 1984 until the treatment plant was installed in 1997 was 7 ppb (EWW 2002,

MassDEP 1988, MassDEP 2004a). Of these years, the annual average concentration exceeded the MCL yearly from 1988 to 1996, with a maximum annual average of 10 ppb in 1992. At the Pines Well, TCE was detected in 70 of 72 samples and exceeded the MCL in 50 samples. Prior to treatment plant installation, the maximum TCE concentration measured at the Pines Well was 7.4 ppb in 1994. The average concentration detected from the time the well was tested multiple times per year for TCE until installation of the treatment plant (1980–1996) was 5 ppb. Of these years, the maximum annual average concentration of 6 ppb occurred yearly from 1990 to 1992.

Easthampton Water Works (2002) tested various points in the water distribution system across town before the water treatment plant began operating in 1997. In 1988, TCE was detected in 11 out of 11 samples, four of which exceeded the MCL. The highest TCE concentration detected was 8.4 ppb in drinking water at a property next door to Easthampton Water Works on Hendrick Street. In 1992, after the Hendrick Street Wellfield and Pines Well were blended with other sources to lower the TCE concentration and reconnected to the distribution system (T. Newton, Easthampton Water Works, personal communication, 2004), TCE was detected in four out of eight samples. Two of these samples had TCE concentrations greater than the MCL. The maximum TCE concentration detected (6.7 ppb) in 1992 was at Johnson Metal Products, which is about 1.5 miles from the Hendrick Street Wellfield.

The Hendrick Street Wellfield and the Pines Well were tested for other VOCs in addition to TCE. No VOCs other than TCE were detected at the Pines Well. Some other VOCs were detected in groundwater at the Hendrick Street Wellfield. Of 71 groundwater samples, methylene chloride was detected twice (5.1 ppb and 5.4 ppb) at levels slightly above the ATSDR Cancer Risk Evaluation Guide (CREG) and MCL of 5 ppb. Also, benzene was detected in Hendrick Street Wellfield groundwater once (0.6 ppb) at a level that equals the CREG, but is less than the MCL of 5 ppb.

Westfield operates four groundwater wells (Well #1, Well #2, Well #7, Well #8) that draw from a section of the Barnes Aquifer that is separate from the main aquifer (C. Darling, Westfield Water Department, personal communication, 2004). Groundwater from Well #8, located in the

[1]The MassDEP cautions that the Drinking Water Program makes every attempt to ensure that these data are accurate, complete and current. However, no guarantee is given that these data are error free. In addition, since updates and corrections are occurring at all times, these data are time sensitive (MassDEP 2004a).

northeast area of Westfield (Figure 3), was tested 47 times for VOCs from 1990 to 2004. TCE was detected once in 1993 (2.9 ppb) and once in 1996 (0.5 ppb) at levels below the MCL of 5 ppb (Table 5) (MassDEP 1988, 2004a). TCE was not detected in nine samples from Well #1 from 1986 to 2003, in 45 samples from Well #2 from 1986 to 2004, and in 50 groundwater samples from Well #7 from 1986 to 2004.

B. Private Wells

From 1997 to 2005, as part of the MassDEP investigation to determine the source of municipal water contamination in Easthampton, 541 groundwater samples from 452 private wells in the Barnes Aquifer region were analyzed for VOCs (Table 6) (MassDEP 2002, 2005b). Thirteen of these wells in the vicinity of the two Holyoke residential properties where PCBs were released were also tested for PCBs in 2000. Private well water was sampled at the tap and before the filter, if a drinking water filter was present. For most households with a filter where TCE was detected before the filter, a sample was also taken of the post-filter water. Four households declined testing.

TCE was detected in 51 of 240 Southampton, 27 of 146 Holyoke, nine of 64 Westfield, and one of two Easthampton private wells tested (MassDEP 2002, 2005b). Of the 88 wells where TCE was detected, 60 wells had a maximum concentration less than 1 ppb and 15 wells had a maximum concentration between 1 and 5 ppb. Thirteen wells had maximum TCE concentrations above the MCL of 5 ppb, with eight in southeastern Southampton near the Holyoke line and five in western Holyoke close to Southampton. The average of the maximum concentration detected in each of the 88 wells where TCE was detected was 3 ppb.

The maximum concentration of TCE detected in private well water was 34.2 ppb at a home on Camp Jahn Road in Southampton. The maximum TCE concentration detected in Holyoke was 19 ppb on Keyes Road. No private well in Westfield or Easthampton had TCE detected above 1 ppb.

Households with private wells where TCE levels were around 1 ppb and above, plus a daycare with a trace TCE level, were offered bottled water delivery from the MassDEP as a temporary measure (MassDEP 2002). Ten of the homes given bottled water were also supplied with whole

house granular-activated carbon filters, which remove TCE before drinking water enters the home.

Methyl-tert-butyl-ether (MTBE) was detected once at 74 ppb, which is above the MassDEP MMCL of 70 ppb, at the residential property on Dupuis Road in Holyoke where wastes were released. No other VOCs, including TCE breakdown products, were detected in private wells at levels above drinking water comparison values (Table 6). Benzene, which was expressed as a community concern, was not detected in private well water. PCBs were not detected in the 13 private wells closest to the two Holyoke residential properties where PCBs were released.

C. Surface Soil

Surface soil samples from a depth of 0 to 6 inches at the three residential properties on Apremont Highway, Dupuis Road, and Mueller Road in Holyoke were analyzed for PCBs during the MassDEP source investigation (MassDEP 2000; Pine and Swallow, Inc. 2000). The average PCB concentration of the 66 samples collected at the Apremont Highway residential property was 16 parts per million (ppm). The maximum PCB concentration (411 ppm) was identified at the Apremont Highway residential property and was greater than the CREG (0.4 ppm). The average concentration of the 66 samples from the Apremont Highway property was 16 ppm. PCBs were detected in surface soil at the Dupuis Road residential property at a maximum concentration of 68 ppm, which exceeds the CREG. The average concentration of the 26 samples from the Dupuis Road property was 4.1 ppm. PCBs were not identified at the Mueller Road residential property (Pine and Swallow, Inc. 2000).

VI. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS AND HEALTH CONCERNS

An evaluation of potential pathways of exposure was conducted to determine whether TCE contamination in the Barnes Aquifer has the potential to impact residents in the surrounding neighborhoods 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 2005).

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 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 or could occur in the present or future. An exposure pathway can be eliminated if at least one of the five elements is missing and will not likely be present in the future. Refer to Table 7 for a summary of the exposure pathways discussed in this section.

To evaluate the potential for health effects for the potential and completed exposure pathways listed in Table 7, exposure doses were estimated and compared to health guideline values. An exposure dose is an estimate of how much of a substance a person may contact based on their actions and habits. For noncancer health outcomes, the proposed U.S. EPA Reference Dose (RfD) for TCE was compared to exposure estimates for noncancer health effects[2] due to TCE (U.S. EPA 2001). The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population that is likely to be without an appreciable risk of deleterious health effects during a lifetime. To calculate potential cancer risk,

[2] Noncancer Health Effects Exposure Dose (Ingestion) =

$$\frac{\text{(Maximum Contaminant Concentration) (Water Ingestion Rate) (Noncancer Effects Exposure Factor*)}}{\text{Body Weight}}$$

*Noncancer Health Effects Exposure Factor = (F x ED) / AT
where,
F = Frequency of Exposure (days/year)
ED = Exposure Duration (years)
AT = Averaging Time (ED x 365 days/year)

exposure estimates for cancer effects[3] were multiplied by U.S. EPA cancer slope factors[4], which measure the relative potency of various carcinogens.

A. Exposure to Groundwater

Based on studies where TCE was shown to cause kidney and liver cancer in animals, TCE is currently classified as a probable human carcinogen by the International Agency for Research on Cancer (IARC) and is reasonably anticipated to be a carcinogen by the National Toxicology Program (NTP) of the United States Department of Health and Human Services. Previously, the U.S. EPA classified TCE as a probable human carcinogen based on inadequate human evidence and sufficient animal evidence. In 2001, the U.S. EPA proposed reclassifying the carcinogenicity (i.e., ability to cause cancer) of TCE to a probable human carcinogen based on sufficient animal evidence and limited human evidence (U.S. EPA 2001). The proposed changes are due to stronger epidemiologic evidence and new mechanistic information about what happens when TCE enters the human body.

The carcinogenicity of TCE in humans has been a matter of controversy within the scientific community. Occupational studies of workers exposed to unmeasured levels of TCE in air were often limited by multiple chemical exposures and small numbers of study participants (ATSDR 1997). While some studies have shown no association between inhalation exposure to TCE and cancer, others have found slight increases in a number of cancer types such as cancers of the kidney, liver, bladder, and NHL. However, problems with study design were often reported, and

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[3] Cancer Effects Exposure Dose =

$$\frac{(\text{Maximum Contaminant Concentration})\ (\text{Ingestion Rate})\ (\text{Cancer Effects Exposure Factor*})\ (2^{\dagger})}{\text{Body Weight}}$$

*Cancer Effects Exposure Factor = (F x ED) / AT
where,
F = Frequency of Exposure (days/year)
ED = Exposure Duration (years)
AT = Averaging Time (70 years x 365 days/year)

[†]Exposure doses received from inhalation and dermal exposures while showering were considered to be equal to the estimated ingestion exposure dose (U.S. EPA 2000).

[4] Cancer Risk = Cancer Effects Exposure Dose x Cancer Slope Factor

associations were often based on small numbers of individuals and complicated by confounding factors (ATSDR 1997).

For humans, the evidence of TCE as a human carcinogen is strongest for kidney and liver cancer in workers exposed through inhalation (Wartenberg et al. 2000). There is weak evidence of excess incidence of bladder cancer among dry cleaning and laundry workers. The strongest support in the scientific literature for elevations in bladder cancer due to TCE exposure is among dry cleaning workers (Wartenberg at al. 2000). Although an association between TCE exposure and dry cleaning workers who were employed before the 1950s and 1960s has been suggested, the elevation in bladder cancer incidence among these workers was probably due to exposure to tetrachloroethylene (PCE), instead of TCE (Wong 2004). Prior to the 1950s or 1960s, PCE was used often and in large amounts for dry cleaning and TCE was used less often and in smaller amounts for spot-cleaning. Studies of dry cleaners who were employed during that time period suggest that PCE was the likely carcinogen.

Several researchers have concluded that TCE is carcinogenic at very high concentrations, but that there is little evidence to support its carcinogenicity at levels typically measured in the environment (Bruning and Bolt 2000, Bull 2000, Green 2001, Clewell and Andersen 2004, Wong 2004). Wong (2004) estimated that an individual exposed daily to TCE in drinking water would have to ingest levels as high as 550,000 ppb TCE to have an exposure equivalent to a worker performing daily degreasing activities with TCE. This level is several orders of magnitude higher than a typical TCE concentration in drinking water. Considering the probable ways that TCE acts in the human body, Clewell and Andersen (2004) applied a conservative factor and concluded that a concentration of 265 ppb TCE in drinking water is unlikely to result in carcinogenicity in humans.

For communities exposed to TCE through the ingestion of drinking water, the strongest evidence in the scientific literature is for elevated leukemia incidence (Wartenberg et al. 2000). Five out of six regions where community studies were conducted showed an elevation in leukemia diagnoses in at least one gender. However, conclusions from these studies are limited because other contaminants were present in the drinking water and because researchers did not adjust for confounding factors such as smoking (ATSDR 1997). An additional community study

completed since Wartenberg's review found no excess leukemia incidence (Morgan and Cassady 2002).

Some studies of communities exposed to TCE in drinking water suggest a relationship between exposure and elevated incidence of leukemia, while some studies have not shown an association. For example, a study of the Woburn, Massachusetts, community exposed to a maximum of 267 ppb TCE reported a statistically significant elevation in leukemia incidence and a significant trend of increased risk with increased exposure opportunities (Costas et al. 2002). In New Jersey, increased leukemia incidence among females was correlated with contaminated drinking water of up to 72 ppb TCE and other VOCs (Fagliano et al. 1990, Cohn et al. 1994). In contrast, there was no difference between the observed and expected number of leukemia diagnoses in a California community that was exposed to up to 97 ppb TCE for a similar time frame as the Barnes Aquifer community (Morgan and Cassady 2002). Rates of leukemia were not elevated in two Finnish villages where residents consumed water with up to 220 ppb TCE and 180 ppb PCE (Vartiainen et al. 1993). In general, in most community studies, exposure to TCE was at a higher concentration than the levels detected in public and private wells drawing from the Barnes Aquifer.

1. Municipal Water Supply

In the past, the primary ways that Easthampton residents could have been exposed to TCE in municipal water from the Barnes Aquifer are via ingestion, inhalation (i.e., while showering), and/or dermal contact (i.e., washing hands or bathing with water containing TCE). Easthampton residents supplied with drinking water from the Hendrick Street Wellfield or Pines Well may have potentially been exposed as early as the 1960s, depending on how long it took for TCE in groundwater to travel 4 miles from the source properties to the wells. According to VOC sampling results, a completed exposure pathway is documented from 1984 to about 1988, when the wells were disconnected from the distribution system, and from about 1990, when the wells were reconnected, until the town water treatment plant came online in 1997. Using conservative assumptions that an adult ingested 2 liters of water and a child ingested 1 liter with the maximum concentration of TCE detected in Easthampton municipal water (i.e., 12 ppb at the Hendrick Street Wellfield) for 350 days per year for the maximum potential exposure duration (35 years),

the estimated noncancer effects exposure dose is 0.0003 milligrams per kilograms per day (mg/kg/day) for adults and children[5]. This estimated daily exposure dose is equal to the U.S. EPA draft RfD (0.0003 mg/kg/day), which represents an estimate of a daily oral exposure that is not expected to result in adverse noncancer health effects (U.S. EPA 2001). The draft RfD for TCE is based on adverse noncancer health effects observed in studies of mice and rats that were exposed to 1 mg/kg/day (U.S. EPA 2001). Because the estimated noncancer effects exposure dose for Easthampton residents in the above scenario is 3,000 times lower that the lowest exposure dose that resulted in adverse health effects in animal studies, noncancer health effects from past exposure to TCE in Easthampton municipal water were not expected.

In order to evaluate the potential for carcinogenic health effects, exposure doses were estimated and compared to health guideline values for cancer. TCE has been classified by the U.S. EPA in the past as a probable human carcinogen based on sufficient evidence in animals and inadequate or no evidence in humans. TCE is currently undergoing review by the U.S. EPA for its carcinogenicity and, thus, was quantitatively evaluated for its cancer-causing potential among residents in the Barnes Aquifer region using both the newly proposed range of cancer slope factors (0.02–0.4 $[mg/kg/day]^{-1}$), which are more conservative, and the old cancer slope factor (0.011 $[mg/kg/day]^{-1}$). For the purposes of evaluating cancer risk, the exposure dose received from inhalation and dermal exposures while showering were considered to be equal to the estimated ingestion exposure dose (U.S. EPA 2000). Under the same assumptions as for the above noncancer health effects and using the proposed cancer risk guidelines, which are more

[5] Noncancer Health Effects Exposure Factor = $\frac{(350 \text{ days/year})\ (35 \text{ years})}{(365 \text{ days/year})\ (35 \text{ years})} = 0.96$

Noncancer Health Effects Exposure Dose (Ingestion) for Adult = $\frac{(0.012 \text{ mg/L})\ (2 \text{ L/day})\ (0.96)}{70 \text{ kg}} = 0.0003 \text{ mg/kg/day}$

Noncancer Health Effects Exposure Dose (Ingestion) for Children = $\frac{(0.012 \text{ mg/L})\ (1 \text{ L/day})\ (0.96)}{35 \text{ kg}} = 0.0003 \text{ mg/kg/day}$

conservative, past opportunities for TCE exposure via municipal water were unlikely to result in unusual cancer risks for either adults or children[6].

Exposures to TCE in municipal water from the Hendrick Street Wellfield and Pines Well in the present and future were eliminated as exposure pathways because a water treatment plant has removed TCE from municipal water since 1997.

There is a completed exposure pathway for about 600 residents in western Holyoke who were supplied with drinking water from the Pequot Well. Samples from the Coronet Homes Well never exceeded the MCL for TCE in six samples from 1980 to 1988; hence, the well was not further evaluated here. Exposure to TCE in drinking water from the Pequot Well could have begun sometime between 1974, when the well was constructed, and 1980, when Pequot Well water was first analyzed for VOCs and TCE was detected. Exposure could have continued from 1980 until the Pequot Well was closed in 1987. If it is assumed that the maximum detected concentration of TCE in the Pequot Well (15 ppb) was continually consumed by residents, the estimated exposure dose for adults who consumed 2 liters of water per day and children who consumed 1 liter per day for the longest potential exposure period (14 years) was 0.0004

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[6] Note: The cancer slope factor used in the following cancer risk calculations [0.21 $(mg/kg/day)^{-1}$] is the midpoint of the U.S. EPA newly proposed range of cancer slope factors: 0.02–0.4 $(mg/kg/day)^{-1}$. The midpoint assesses cancer risk in the general population (U.S. EPA 2001).

$$\text{Cancer Effects Exposure Factor (Adult)} = \frac{(350 \text{ days/year})\ (35 \text{ years})}{(365 \text{ days/year})\ (70 \text{ years})} = 0.48$$

$$\text{Cancer Effects Exposure Dose (Adult)} = \frac{(0.012 \text{ mg/L})\ (2 \text{ L/day})\ (0.48)\ (2)}{70 \text{ kg}} = 0.00033 \text{ mg/kg/day}$$

$$\text{Cancer Risk (Adult)} = 0.00033 \text{ mg/kg/day} \times 0.21 \text{ (mg/kg/day)}^{-1} = 7 \times 10^{-5}$$

$$\text{Cancer Effects Exposure Factor (Child)} = \frac{(350 \text{ days/year})\ (18 \text{ years})}{(365 \text{ days/year})\ (70 \text{ years})} = 0.25$$

$$\text{Cancer Effects Exposure Dose (Child)} = \frac{(0.012 \text{ mg/L})\ (1 \text{ L/day})\ (0.25)\ (2)}{35 \text{ kg}} = 0.0002 \text{ mg/kg/day}$$

$$\text{Cancer Risk (Child)} = 0.0002 \text{ mg/kg/day} \times 0.21 \text{ (mg/kg/day)}^{-1} = 4 \times 10^{-5}$$

mg/kg/day[7]. Because the estimated exposure dose is higher than the U.S. EPA draft RfD (0.0003 mg/kg/day), this suggests that residents could have been exposed to a dose that could have resulted in adverse noncancer health effects. However, because the estimated exposure dose for residents in this scenario is 2,500 times lower than the lowest exposure dose that resulted in adverse health effects in animal studies, noncancer health effects from past exposure to TCE in Pequot Well water were determined to be unlikely.

Under the newly proposed cancer risk guidelines and the same assumptions, increased cancer risk from past exposure to TCE in drinking water from the Pequot Well was determined to be unlikely for adults and children[8].

Present and future exposures to TCE in drinking water from the Pequot Well and Coronet Homes Well were eliminated as exposure pathways because the wells closed in 1987 and are not maintained as emergency sources of water by Holyoke Water Works.

TCE was never detected in Westfield municipal water above the MCL, and the Southampton municipal supply did not draw from the contaminated section of the Barnes Aquifer.

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[7] Noncancer Health Effects Exposure Factor = $\frac{(350 \text{ days/year}) (14 \text{ years})}{(365 \text{ days/year}) (14 \text{ years})} = 0.96$

Noncancer Health Effects Exposure Dose (Ingestion) for Adults = $\frac{(0.015 \text{ mg/L}) (2 \text{ L/day}) (0.96)}{70 \text{ kg}} = 0.0004 \text{ mg/kg/day}$

Noncancer Health Effects Exposure Dose (Ingestion) for Children = $\frac{(0.015 \text{ mg/L}) (1 \text{ L/day}) (0.96)}{35 \text{ kg}} = 0.0004 \text{ mg/kg/day}$

[8] Cancer Effects Exposure Factor = $\frac{(350 \text{ days/year}) (14 \text{ years})}{(365 \text{ days/year}) (70 \text{ years})} = 0.19$

Cancer Effects Exposure Dose (Adult) = $\frac{(0.015 \text{ mg/L}) (2 \text{ L/day}) (0.19) (2)}{70 \text{ kg}} = 0.00016 \text{ mg/kg/day}$

Cancer Risk (Adult) = $0.00016 \text{ mg/kg/day} \times 0.21 (\text{mg/kg/day})^{-1} = 3 \times 10^{-5}$

Cancer Effects Exposure Dose (Child) = $\frac{(0.015 \text{ mg/L}) (1 \text{ L/day}) (0.19) (2)}{35 \text{ kg}} = 0.00016 \text{ mg/kg/day}$

Cancer Risk (Child) = $0.00016 \text{ mg/kg/day} \times 0.21 (\text{mg/kg/day})^{-1} = 3 \times 10^{-5}$

2. Private Wells

There is a completed exposure pathway prior to 1997 for some residents in Holyoke and Southampton who consumed private well water from the contaminated section of the Barnes Aquifer. The only Easthampton private wells are located about 3 miles north of the TCE contamination and had no TCE detections greater than 1 ppb. There have been no detections above 1 ppb in any Westfield private well.

In the past, the primary routes that residents could have been exposed to TCE in private well water are via ingestion, inhalation, and dermal contact. TCE could have entered private wells sometime between the mid-1950s, when TCE was released, and 1997, when the MassDEP first detected TCE in private wells. Assuming that an adult ingested 2 liters and a child ingested 1 liter of water with the maximum concentration of TCE (34.2 ppb) detected in private well water for 350 days per year for the maximum potential exposure duration (45 years), the estimated noncancer health effects exposure dose is 0.0009 mg/kg/day for adults and children[9]. Because the estimated exposure dose is higher than the U.S. EPA draft RfD (0.0003 mg/kg/day), this suggests that residents could have been exposed to a dose that could have resulted in adverse noncancer health effects. However, because the estimated exposure dose for residents in this scenario is 1,000 times lower that the lowest exposure dose that resulted in adverse health effects in animal studies, noncancer health effects from past exposure to TCE in private well water were determined to be unlikely.

In order to evaluate the potential for carcinogenic health effects, exposure doses were estimated and compared to both the newly proposed health guideline values, which are more conservative, and the old health guideline value. Under the newly proposed health guideline values and conservative assumptions, if an adult ingested 2 liters and a child ingested 1 liter of water with the maximum concentration of TCE (34.2 ppb) detected in private well water for 350 days per

[9] Noncancer Health Effects Exposure Factor = $\frac{(350 \text{ days/year}) (45 \text{ years})}{(365 \text{ days/year}) (45 \text{ years})} = 0.96$

Noncancer Health Effects Exposure Dose (Ingestion) for Adults = $\frac{(0.0342 \text{ mg/L}) (2 \text{ L/day}) (0.96)}{70 \text{ kg}} = 0.0009 \text{ mg/kg/day}$

Noncancer Health Effects Exposure Dose (Ingestion) for Children = $\frac{(0.0342 \text{ mg/L}) (1 \text{ L/day}) (0.96)}{35 \text{ kg}} = 0.0009 \text{ mg/kg/day}$

year for the maximum potential exposure duration (45 years), they could have been exposed to TCE at a level that could have presented a low increased cancer risk[10]. This scenario is considered a worst-case scenario. Under a more reasonable scenario that assumes exposure to the average of the maximum TCE concentration detected in each of the 88 private wells where TCE was detected (3 ppb), an increased cancer risk would not be expected[11]. Under the old health guideline value, residents who consumed the maximum TCE concentration (34.2 ppb) detected in private well water for 350 days per year over 45 years were not likely to have been exposed to TCE at a level that might present an increased cancer risk[12].

Because residents could have experienced a low increased risk of cancer under the conservative newly proposed guidelines, the above scenario was further evaluated by comparing the estimated

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[10] Cancer Effects Exposure Factor (Adult) = $\frac{(350 \text{ days/year}) (45 \text{ years})}{(365 \text{ days/year}) (70 \text{ years})} = 0.62$

Cancer Effects Exposure Dose (Adult) = $\frac{(0.0342 \text{ mg/L}) (2 \text{ L/day}) (0.62) (2)}{70 \text{ kg}} = 0.0012 \text{ mg/kg/day}$

Cancer Risk (Adult) = 0.0012 mg/kg/day x 0.21 $(\text{mg/kg/day})^{-1} = 3 \times 10^{-4}$

Cancer Effects Exposure Factor (Child) = $\frac{(350 \text{ days/year}) (18 \text{ years})}{(365 \text{ days/year}) (70 \text{ years})} = 0.25$

Cancer Effects Exposure Dose (Child) = $\frac{(0.0342 \text{ mg/L}) (1 \text{ L/day}) (0.25) (2)}{35 \text{ kg}} = 0.0005 \text{ mg/kg/day}$

Cancer Risk (Child) = 0.0005 mg/kg/day x 0.21 $(\text{mg/kg/day})^{-1} = 1 \times 10^{-4}$

[11] Cancer Effects Exposure Factor (Adult) = $\frac{(350 \text{ days/year}) (45 \text{ years})}{(365 \text{ days/year}) (70 \text{ years})} = 0.62$

Cancer Effects Exposure Dose (Adult) = $\frac{(0.003 \text{ mg/L}) (2 \text{ L/day}) (0.62) (2)}{70 \text{ kg}} = 0.0001 \text{ mg/kg/day}$

Cancer Risk (Adult) = 0.0001 mg/kg/day x 0.21 $(\text{mg/kg/day})^{-1} = 2 \times 10^{-5}$

Cancer Effects Exposure Factor (Child) = $\frac{(350 \text{ days/year}) (18 \text{ years})}{(365 \text{ days/year}) (70 \text{ years})} = 0.25$

Cancer Effects Exposure Dose (Child) = $\frac{(0.003 \text{ mg/L}) (1 \text{ L/day}) (0.25) (2)}{35 \text{ kg}} = 0.000042 \text{ mg/kg/day}$

Cancer Risk (Child) = 0.000042 mg/kg/day x 0.21 $(\text{mg/kg/day})^{-1} = 9 \times 10^{-6}$

[12] Cancer Risk (Adult) = 0.0012 mg/kg/day x 0.011 $(\text{mg/kg/day})^{-1} = 1 \times 10^{-5}$

Cancer Risk (Child) = 0.0005 mg/kg/day x 0.011 $(\text{mg/kg/day})^{-1} = 5 \times 10^{-7}$

exposure dose to the Cancer Effect Level (CEL) for TCE (ATSDR 2001a). The CEL is the lowest dose of a chemical that produces significant increases in cancer diagnoses in animal or human studies. The estimated exposure dose for residents exposed to the maximum TCE concentration detected in a private well for 350 days per year over 45 years would be 800,000 times lower for adults and 2,000,000 times lower for children than the CEL observed in scientific studies of mice exposed to high doses of TCE (1,000 mg/kg/day) that developed liver cancer and rats that developed kidney cancer (ATSDR 1997)[13]. The large margin of safety (800,000 for adults and 2,000,000 for children), which is the ATSDR CEL divided by the estimated exposure dose for residents exposed to the maximum TCE concentration detected in a private well, indicates that residents are unlikely to have an unusually increased risk of developing cancer as a result of their exposure.

Exposures to TCE in private well water in the present and future were eliminated as exposure pathways for the majority of residents because they accepted bottled water and carbon filters or connected to an uncontaminated municipal water supply after TCE was identified in their private wells in 1997. However, because a carbon filter needs to be replaced periodically (C. Chamberlain, MassDEP, personal communication, 2004), there is the potential for present or future exposure if residents do not properly maintain the filter or if they use unfiltered water. There also exists a potential exposure pathway for four households that declined to allow the MassDEP to test their wells in 1997.

Exposures to PCBs in private well water were eliminated as past, present, and future exposure pathways for residents. PCBs have not been demonstrated to have migrated from the source properties via groundwater and were not detected in drinking water samples in 2000 (MassDEP 2002).

[13] Margin of Safety (Adult) = $\frac{1{,}000 \text{ mg/kg/day}}{0.0012 \text{ mg/kg/day}} = 800{,}000$

Margin of Safety (Child) = $\frac{1{,}000 \text{ mg/kg/day}}{0.0005 \text{ mg/kg/day}} = 2{,}000{,}000$

B. Exposure to Soil

Past exposures to PCBs in surface soil may have been possible for children who lived and may have played in surface soil at two Holyoke residential properties (Apremont Highway and Dupuis Road) where PCB wastes were released. They may have been exposed through incidental ingestion to PCBs in surface soil. Assuming that a child resident ingested surface soil with the maximum PCB concentration detected in surface soil at either of the two properties (411 ppm at the Apremont Highway property) for 7 days a week and 50 weeks per year over 18 years, exposure could have presented a moderate increased risk of cancer[14]. However, these exposure assumptions are conservative, and it is very unlikely that a resident could have had consistent contact with soil containing the highest concentration of PCBs. It is more likely that soil with a range of contaminant concentrations could have been ingested over time. Based on readily available surface soil data from the Apremont Highway source property, the average concentration of PCBs was 16 ppm (MassDEP 2000)[15]. Under the more realistic assumption that a child resident at either property could have been exposed to the average concentration of PCBs, an increased cancer risk would be unlikely[16].

Since PCB-contaminated soil was removed from the Apremont Highway property and Dupuis Road property and replaced with clean soil, in addition to the placement of Activity and Use Limitation (AUL) deed restrictions on the properties, present and future ingestion of contaminants in surface soils by residents were eliminated as exposure pathways.

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

Cancer Effects Exposure Dose = $\frac{\text{(411 mg/kg) (200 mg/day) (0.25) (1 kg/}10^6\text{ mg)}}{\text{35 kg}} = 0.0006$ mg/kg/day

Cancer Risk = 0.0006 mg/kg/day x 2.0 $(\text{mg/kg/day})^{-1} = 1 \times 10^{-3}$

[15] Because the detection limit was not available, the average was calculated assuming that all samples where PCBs were not detected were equal to zero.

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

Cancer Effects Exposure Dose = $\frac{\text{(16 mg/kg) (200 mg/day) (0.25) (1 kg/}10^6\text{ mg)}}{\text{35 kg}} = 2.3 \times 10^{-5}$ mg/kg/day

Cancer Risk $= 2.3 \times 10^{-5}$ mg/kg/day x 2.0 $(\text{mg/kg/day})^{-1} = 5 \times 10^{-5}$

C. Exposure to Air

Some of the products of PCB combustion include chlorinated dibenzodioxins (commonly known as dioxins) and chlorodibenzofurans, as well as PCBs that might result from incomplete combustion (ATSDR 2000). The community in the Barnes Aquifer area expressed concern that residents living in the Dupuis Road neighborhood in west Holyoke could have been exposed through inhalation to contaminants when PCB wastes were reportedly burned at a residential property about every other weekend from the early to mid-1950s. Exposure to contaminants in smoke could have been possible in the past; however, air monitoring data were not available for that time. Also, surface soil data were not available for PCB combustion products that could have been deposited at neighboring properties, which could help to evaluate potential past exposure opportunities to contaminants in ambient air. Because no environmental data were available, it was not possible to quantitatively evaluate the potential for adverse health effects that could result from possible exposure. Since the extent of exposure opportunities is not known, the MDPH examined the geographic pattern of cancer among individuals living in the Dupuis Road neighborhood to assess whether any unusual patterns might be evident in relation to the property where burning occurred.

Present and future exposures at the Dupuis Road source property are not of concern because the soil contaminated with PCBs was removed and PCB wastes are no longer being burned.

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 Easthampton, Holyoke, Southampton, and Westfield from the MCR, a division of the Bureau of Health Information, Statistics, Research, and Evaluation within the MDPH. Eight cancers types were evaluated in this investigation: Hodgkin's disease, leukemia, non-Hodgkin's lymphoma, and cancers of the bladder, esophagus, kidney, liver, and pancreas. [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, such as TCE, and residents' concerns about suspected elevations in cancers of the esophagus and pancreas in the Barnes Aquifer region. Only cases reported to the MCR as a primary cancer for one of the eight cancer types and diagnosed among a resident of Easthampton, Holyoke, Southampton, or Westfield were included in the analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis. The address of each case was matched to its corresponding census tract. Due to incomplete information, the addresses of six cases (3%) in Easthampton, six cases (0.9%) in Holyoke, one case (2%) in Southampton, and two cases (0.4%) in Westfield could not be assigned to a census tract.

The MCR is a population-based surveillance system that has been monitoring cancer incidence in Massachusetts since 1982. All new diagnoses of cancer among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111s.111b). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment) and deletes duplicate case reports. Once these steps are finished, the data for that year are considered "complete." Due to the volume of information received by the MCR, the large number of reporting facilities, and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of 2 years prior to the current date. At the time of this analysis, the most recent and complete data records available from the MCR included diagnoses that occurred from 1/1/1982 to 12/31/2000. The cancer incidence statistics in Section VII, Part B, cover the 19-year period of 1982 through 2000. However, this surveillance system is ongoing and collects reports on a daily basis. Therefore, it is possible for CAP staff to review case reports for more recent years (i.e., 2001 to the present[17]), which can provide a qualitative review of cancer patterns in a given area. The geographic distribution of residences of individuals diagnosed in more recent years is evaluated in Section VII, Part D, and recent diagnoses in the Barnes Aquifer area are discussed along with diagnoses from 1982–2000 in Section VIII.

[17] Entered on MCR computer files before November 15, 2005.

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 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. Two duplicate reports in Easthampton, four duplicate reports in Holyoke, and four duplicate reports in Westfield were 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 Easthampton, Holyoke, Southampton, or Westfield, 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 each of the four

communities as a whole, as well as for the specific census tracts in the Barnes Aquifer region. 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. However, because statewide data for 2001 to the present were not complete at the time of analysis, as discussed above, incidence ratios cannot be calculated for recent years.

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 each census tract (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[18].

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

According to the United States Census Bureau, the four cities and towns encompassed by this evaluation are currently divided into 21 smaller census tracts. Easthampton is divided into three census tracts, Holyoke is comprised of nine census tracts, Southampton has one census tract, and Westfield is divided into eight census tracts. Census tracts can change over time. For instance, in 1990, Easthampton CT 8224 and Holyoke CT 8121 were both split into two census tracts each (CT 8224.01, 8224.02 and CT 8121.01, 8121.02, respectively) by the United States Census Bureau. Because this evaluation analyzes cancer incidence for a long time period (1982–2000), the census tracts that split in 1990 were combined and thus considered as the original 1980 census tracts throughout this entire evaluation.

[18] 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.

The focus areas of this evaluation were CT 8223 (eastern Easthampton) and CT 8224 (western Easthampton), CT 8121 (western Holyoke), CT 8225 (Southampton), and CT 8125 (eastern Westfield) (Figure 2). [Fifteen cases (0.1%) for which census tract designation was not possible were included in the city/town total for each community.] These particular census tracts were chosen for evaluation because they include residents who were at risk of exposure to TCE in Barnes Aquifer drinking water. Most residents of Easthampton CT 8223 and CT 8224 were at risk of exposure to TCE from the Barnes Aquifer because the majority of households were connected to municipal water when TCE was present in Easthampton drinking water. It is important to note that most residents of Holyoke CT 8121, Southampton CT 8225, and Westfield CT 8125, were not at risk of exposure to TCE from Barnes Aquifer drinking water. These three particular census tracts have a combined population of 26,416 (U.S. DOC 2000). Of those 26,416 residents, a conservative estimate is that at most about 7% (n = 1,900) were at risk of exposure to TCE in drinking water from the Barnes Aquifer.

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 applied 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 study population is significantly different from the comparison or "normal" population. "Significantly different" means there is less than a 5% chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer cases.

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

significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. 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, lung and bronchus, and pancreatic cancers. Other risk factors for various cancer types may include lack of crude fiber in the diet, high fat consumption, excessive alcohol consumption, 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 Easthampton, Holyoke, Southampton, and Westfield 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; therefore, it was not possible to consider their contributions to cancer 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 2005). This allowed assignment of census tract location for each case as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level within a census tract (i.e., neighborhoods). The geographic pattern was assessed by qualitatively evaluating the point pattern of cases in all areas of Easthampton, Holyoke, Southampton, and Westfield. In instances where the address information from the MCR was incomplete (that is did not include specific streets or street numbers) efforts were made to research those cases using telephone books and 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: The MDPH is bound by Massachusetts General 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 Easthampton, Holyoke, Southampton, Westfield, and selected census tracts in the communities: Easthampton CT 8223 and CT 8224, Holyoke CT 8121, and Westfield CT 8125. Because the town of Southampton has one census tract, only townwide cancer incidence rates were evaluated. To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–2000. Table 8a through Table 15d summarize cancer incidence data for the towns and the selected census tracts. Consistent with MDPH policy, 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 diagnoses of cancer were occurring.

1. *Cancer Incidence in Easthampton*

The eight cancer types evaluated in this report generally occurred approximately at or near expected rates in the town of Easthampton 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 8a through Table 8d). One exception was pancreatic cancer, which occurred more often than expected (34 observed versus 29.7 expected, SIR = 115, 95% CI = 79–160). This elevation was not statistically significant and was largely due to a statistically significant elevation in the incidence of pancreatic cancer during the most recent time period, 1994–2000 (21 diagnoses observed versus 12.6 expected, SIR = 167, 95% CI = 103–255). This elevation was due to non-statistically significant elevations among both males (11 diagnoses observed versus 5.9 expected, SIR = 186, 95% CI = 93–332) and females (10 diagnoses observed versus 6.6 expected, SIR = 151, 95% CI = 72–277). During the earlier two time periods, pancreatic cancer occurred about as or less than expected.

Bladder cancer, esophagus cancer, leukemia, liver cancer, and NHL all occurred approximately equal to or less often than expected during the 1982–2000 time period. There were 47 diagnoses of bladder cancer during 1982–2000, whereas approximately 53 diagnoses were expected (SIR = 89, 95% CI = 65–118). Fewer bladder cancer diagnoses were also observed than were expected during each of the three smaller time periods evaluated. Fifteen diagnoses of esophagus cancer were observed in Easthampton during 1982–2000 versus about 16 expected. When examined over time, esophagus cancer occurred about as expected in the three time periods. Residents of Easthampton experienced leukemia at a less than expected rate during 1982–2000 (23 diagnoses observed versus 27.8 expected, SIR = 83, 95% CI = 52–124) and during each of the three smaller time periods. There were seven diagnoses of liver cancer observed versus approximately eight diagnoses expected (SIR = 89, 95% CI = 36–183). Liver cancer occurred less often than expected during time periods 1982–1987 and 1994–2000 and slightly more often than expected during the middle time period, 1988–1993 (4 diagnoses observed versus 2.2 expected). NHL occurred less often than expected during the 1982–2000 time period. There were 45 diagnoses of NHL during 1982–2000, whereas approximately 49.3 diagnoses were expected (SIR = 91, 95% CI = 67–122). Fewer NHL diagnoses were observed than were expected during the middle

time period, 1988–1993, while NHL diagnoses occurred about as expected for the other two time periods.

Diagnoses of Hodgkin's disease, kidney cancer, and pancreatic cancer each occurred more often than expected in Easthampton during 1982–2000, but none of the elevations was statistically significant. There were 13 diagnoses of Hodgkin's disease during 1982–2000, whereas approximately 11 diagnoses were expected (SIR = 118, 95% CI = 62–201). Five individuals were diagnosed with Hodgkin's disease during 1982–1987, while 3.5 diagnoses were expected. Diagnoses of Hodgkin's disease occurred about as expected for the two subsequent time periods. Kidney cancer occurred more often than expected in Easthampton during 1982–2000 (36 diagnoses observed versus 32.1 expected, SIR = 112, 95% CI = 79–155). This elevation is largely attributed to kidney cancer incidence during the earliest time period, 1982–1987 (11 diagnoses observed versus 7.6 expected, SIR = 145, 95% CI = 72–260). Neither elevation was statistically significant. Kidney cancer occurred about as expected in the subsequent two time periods.

2. Cancer Incidence in Easthampton Census Tract 8223 and Census Tract 8224

The eight cancer types evaluated in this report generally occurred approximately near or below expected rates in Easthampton CT 8223 during the 19-year time period 1982–2000 (see Table 9a through Table 9d). More esophagus cancer diagnoses were observed during the overall time period (10 diagnoses observed versus 6.9 expected, SIR = 145, 95% CI = 69–266); however, the observed increase was not statistically significant.

In general, when cancer rates in CT 8223 were evaluated for smaller time periods, no consistent trends over time were observed. The overall elevation in esophagus cancer in CT 8223, which was not statistically significant, was primarily due to an elevation among males during the latest time period (i.e., 1994–2000), which also was not statistically significant (5 diagnoses versus 2.0 expected, SIR = 254, 95% CI = 82–592). There was one statistically significant elevation in NHL among males during the most recent time period (10 diagnoses observed versus 4.3 expected, SIR = 233, 95% CI = 111–428). NHL occurred about as or less than expected among males during the other two time periods and about as expected among females during all three time periods in this census tract. An elevation in pancreatic cancer diagnoses among females

during 1994–2000 was borderline statistically significant (7 diagnoses observed versus 2.8 expected, SIR = 249, 95% CI = 100–513). Pancreatic cancer in the two earlier time periods occurred less than expected for females and about as expected for males in all three smaller time periods. In general, bladder cancer, Hodgkin's disease, kidney cancer, leukemia, and liver cancer occurred about as expected in CT 8223 during each of the smaller time periods evaluated.

Of the eight cancer types evaluated in this report, six (bladder cancer, esophagus cancer, Hodgkin's disease, leukemia, liver cancer, and NHL) occurred about as or less often than expected in Easthampton CT 8224 during the 19-year time period, 1982–2000, among males and females combined. More kidney cancer (25 diagnoses observed versus 18.9 expected) and pancreatic cancer (22 diagnoses observed versus 16.7 expected) diagnoses were observed during the overall time period; however, the observed increases were not statistically significant.

For bladder cancer, esophagus cancer, Hodgkin's disease, leukemia, and NHL, most occurred less frequently or about as expected during each smaller time period in CT 8224. Any elevations observed were based on about one or two additional cases above the expected number. The overall elevation in kidney cancer in CT 8224, which was not statistically significant, was primarily due to elevations during the earliest and latest time periods (i.e., 1982–1987, 1994–2000). Neither elevation was statistically significant. There was one statistically significant elevation in pancreatic cancer among males during the most recent time period (9 diagnoses observed versus 3.7 expected, SIR = 246, 95% CI = 112–466). Pancreatic cancer occurred less than expected among males during the other two time periods and about as expected among females during all three time periods. Tables 10a through 10d provide additional details.

3. Cancer Incidence in Holyoke

Bladder cancer, Hodgkin's disease, kidney cancer, liver cancer, NHL, and pancreatic cancer all occurred approximately equal to or less often than expected during the 1982–2000 time period (Tables 11a–11d). There were 149 diagnoses of bladder cancer during 1982–2000, whereas approximately 164 diagnoses were expected (SIR = 91, 95% CI = 77–107). Diagnoses of bladder cancer occurred at about the expected rate during two of the smaller time periods, 1982–1987 and 1994–2000. Bladder cancer was diagnosed statistically significantly less often than expected during the middle time period, 1988–1993 (33 diagnoses observed versus 51.6

expected, SIR = 64, 95% CI = 44–90), for both males only and males and females combined. Diagnoses of esophagus cancer were elevated during 1982–2000, but the elevation was not statistically significant (58 diagnoses observed versus 49.0 expected, SIR = 118, 95% CI = 90–153). When examined over time, residents of Holyoke experienced esophagus cancer at about the expected rate during 1982–1987 and at elevated rates during 1988–1993 (18 diagnoses observed versus 15.1 expected, SIR = 119, 95% CI = 71–188) and 1994–2000 (25 diagnoses observed versus 18.6 expected, SIR = 134, 95% CI = 87–198). Neither of these elevations was statistically significant. Residents of Holyoke experienced Hodgkin's disease at a less than expected rate during 1982–2000 and during each of the three smaller time periods. Overall, 24 diagnoses of Hodgkin's disease were observed in the city of Holyoke during 1982–2000 versus about 29 expected. Kidney cancer also occurred at a less than expected rate overall and for each smaller time period. The incidence of leukemia was elevated from 1982–2000, although the elevation was not statistically significant (97 diagnoses observed versus 86.7 expected, SIR = 112, 95% CI = 91–136). During the first time period, 1982–1987, there were about two additional diagnoses above the expected number. In the middle time period, 1988–1993, there was a statistically significant elevation among males and females combined (36 diagnoses observed versus 24.8 expected, SIR = 145, 95% CI = 102–201) and among females alone (20 diagnoses observed versus 11.5 expected, SIR = 173, 95% CI = 106–268). Leukemia among males during this time period was slightly elevated, but not statistically significantly (16 diagnoses observed versus 13.3 expected, SIR = 121, 95% CI = 69–196). In 1982–1987, there was about one additional diagnosis of leukemia in females, and leukemia occurred less than expected in females from 1994–2000 (12 diagnoses observed versus 16.5 expected, SIR = 73, 95% CI = 38–127). Both liver cancer and NHL occurred about as or less than expected in the city of Holyoke during 1982–2000 and the three smaller time periods. There were 19 diagnoses of liver cancer, when about 23 were expected (SIR = 82, 95% CI = 49–128), and 140 diagnoses of NHL, when about 147 were expected for 1982–2000. Residents of Holyoke experienced pancreatic cancer at about the rate expected during 1982–2000 (99 diagnoses observed versus 95.1 expected, SIR = 104, 95% CI = 85–127). Pancreatic cancer was diagnosed at about the expected rate for the first two time periods, 1982–1987 and 1988–1993, and was slightly elevated during the most recent time period, 1994–2000 (39 diagnoses observed versus 35.1 expected, SIR = 111, 95% CI = 79–152). This elevation was not statistically significant.

4. *Cancer Incidence in Holyoke Census Tract 8121*

In general, when cancer rates in CT 8121 were evaluated for smaller time periods, no consistent trends over time were observed. Among males, there was about one more leukemia diagnosis than expected during the first time period, 1982–1987, and leukemia occurred less than the rate expected during the second time period, 1988–1993. In the most recent time period, 1994–2000, leukemia among males occurred statistically significantly more often than expected (14 diagnoses observed versus 7.4 expected, SIR = 190, 95% CI = 104–318). From 1982 to 1987, NHL diagnoses were elevated (21 diagnoses observed versus 14.2 expected, SIR = 148, 95% CI = 91–226). The elevation during this time period was not statistically significant. NHL diagnoses occurred less than expected during the next two time periods, 1988–1993 and 1994–2000. The overall elevation of pancreatic cancer among females in CT 8121 was primarily due to a statistically significant elevation during the middle time period, 1988–1993 (12 diagnoses observed versus 6.2 expected, SIR = 195, 95% CI = 101–304). There was about one diagnosis above the expected number in the first time period and about two diagnoses above the expected number in the most recent period for pancreatic cancer among females. Bladder cancer, Hodgkin's disease, and kidney cancer occurred less frequently or about as expected during each of the smaller time periods evaluated. Refer to Tables 12a through 12d for details.

5. *Cancer Incidence in Southampton*

The eight cancer types evaluated in this report generally occurred approximately near or below expected rates in the town of Southampton 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), with some exceptions (see Table 13a through Table 13d). The incidence of bladder cancer among males was statistically significantly elevated in Southampton during 1982–2000 (17 diagnoses observed versus 9.8 expected, SIR = 173, 95% CI = 101–277). Among females, bladder cancer occurred as expected during the overall period (3 diagnoses observed versus 3.0 expected). For males, bladder cancer was diagnosed more often than expected during the earliest time period, 1982–1987 (6 diagnosis versus 2.8 expected, SIR = 216, 95% CI = 79–470), but the elevation was not statistically significant. A statistically significant elevation did occur in the middle time period, 1988–1993 (8 diagnoses observed versus 3.0 expected, SIR = 266, 95% CI = 114–524).

Bladder cancer occurred slightly less than expected during the most recent time period, 1994–2000 (3 diagnoses observed versus 3.8 expected).

Kidney cancer occurred less often than expected in Southampton from 1982 to 2000, primarily due to a lower-than-expected rate among males in the town (1 diagnosis observed versus 5.4 expected). Among females, more cases occurred during 1982–2000 than expected (6 diagnoses observed versus 2.9 expected, SIR = 206, 95% CI = 75–448), but this elevation was not statistically significant. No females were diagnosed with kidney cancer from 1982 to 1987. There was one diagnosis above the expected number during the middle time period, 1988–1993. Females were diagnosed more often than expected during the most recent time period, 1994–2000 (4 diagnoses observed versus 1.5 expected).

6. Cancer Incidence in Westfield

From 1982 to 2000 in Westfield, cancer incidence rates were lower than expected for esophagus cancer, Hodgkin's disease, leukemia, NHL, and pancreatic cancer (see Table 14a through Table 14d). NHL occurred statistically significantly less often than expected among both males and females combined (94 diagnoses observed versus 120.2 expected, SIR = 78, 95% CI = 63–96) and females alone (43 diagnoses expected versus 58.6 expected, SIR = 73, 95% CI = 53–99). Incidence rates were about as expected for kidney cancer and were higher than expected for bladder cancer and liver cancer, although neither elevation was statistically significant. Overall, 147 individuals were diagnosed with bladder cancer compared to about 129 expected (SIR = 114, 95% CI = 96–134). Bladder cancer was elevated among males (110 diagnoses observed versus 92.5 expected, SIR = 119, 95% CI = 98–143) and occurred about as expected among females (37 diagnoses observed versus 36.3 expected, SIR = 102, 95% CI = 72–140). The elevations among both genders combined and among males separately were not statistically significant. Twenty-one individuals in Westfield were diagnosed with liver cancer compared to about 19 expected (SIR = 110, 95% CI = 68–168). Liver cancer occurred slightly higher than expected among males (16 diagnoses observed versus 13.7 expected, SIR = 117, 95% CI = 67–190) and was about as expected among females. Neither elevation was statistically significant. The lower-than-expected rate of pancreatic cancer among females was borderline statistically significant (27 diagnoses observed versus 39.1 expected, SIR = 69, 95% CI = 45–100).

In general, when cancer rates in Westfield were evaluated for smaller time periods, no consistent trends over time were observed. Diagnoses of bladder cancer among males and females combined and among males separately occurred at a higher than expected rate during each of the smaller time periods, although none of the elevations was statistically significant. When evaluated by smaller time periods, esophagus cancer, Hodgkin's disease, leukemia, NHL, and pancreatic cancer occurred less than expected or about as expected over time. Kidney cancer was slightly higher than expected in 1982–1987, lower than expected in the 1988–1993, and slightly higher than expected in 1994–2000. None of the elevations was statistically significant. Citywide rates of liver cancer were lower than expected during 1982–1987 and higher than expected during the later two time periods. Neither of the elevations was statistically significant.

7. Cancer Incidence in Westfield Census Tract 8125

In Westfield CT 8125, esophagus cancer, kidney cancer, liver cancer, NHL, and pancreatic cancer occurred approximately near or below expected rates during the 19-year time period, 1982–2000. More diagnoses of Hodgkin's disease in males and females combined (8 diagnoses observed versus 4.6 expected, SIR = 174, 95% CI = 75–342) and leukemia (15 diagnoses observed versus 10.3 expected, SIR = 146, 95% CI = 82–241) were observed during the overall time period; however, the observed increases were not statistically significant. Among females, Hodgkin's disease was statistically significantly elevated for the overall time period (6 diagnoses observed versus 2.1 expected, SIR = 287, 95% CI = 105–624). This elevation was due to small elevations in each of the three time periods (i.e., about one to two excess cases in each time period). Hodgkin's disease occurred less than expected among males from 1982 to 2000 (2 diagnoses observed versus 2.5 expected). Bladder cancer among males and females combined was statistically significantly elevated for 1982–2000 (28 diagnoses observed versus 18.1 expected, SIR = 155, 95% CI = 103–223). Bladder cancer among males was elevated and borderline statistically significant (22 diagnoses observed versus 13.8 expected, SIR = 160, 95% CI = 100–242). Among females, bladder cancer was slightly elevated (6 diagnoses observed versus 4.4 expected, SIR = 137, 95% CI = 50–299), but not statistically significantly.

In general, when cancer rates in CT 8125 were evaluated for smaller time periods, no consistent trends over time were observed. Esophagus cancer, kidney cancer, liver cancer, and NHL

occurred about as expected in CT 8125 during each of the smaller time periods evaluated. Bladder cancer was consistently elevated, although not statistically significantly, for each of the three time periods. This resulted in the statistically significant elevation during the overall 1982–2000 time period. For leukemia, there was about one diagnosis above the expected number during 1982–1987, about one fewer diagnosis than expected in the next time period, and about four additional diagnoses in the most recent time period. The occurrence of pancreatic cancer in the smaller time periods was also inconsistent. There were no diagnoses during 1982–1987, about two additional diagnoses during 1988–1993, and pancreatic cancer occurred about as expected during 1994–2000. Refer to Table 15a through Table 15d.

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 Easthampton, Holyoke, Southampton, and Westfield. 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 many of these cancer types. Unfortunately, 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 Easthampton, Holyoke, Southampton, and Westfield. 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 for 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 cancers of the bladder, esophagus, kidney, and pancreas. The smoking history of individuals diagnosed with these cancer types was reviewed to assess the possible role tobacco

smoking may have played in the development of these cancers among residents of the four communities.

In some studies, an association has been found with exposures specific to certain occupations and an increase in the incidence of bladder cancer, kidney cancer, leukemia, liver cancer, NHL, and pancreatic cancer. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types to determine the role that occupational factors may have played in the development of these cancers in Easthampton, Holyoke, Southampton, and Westfield. 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 frequently be reported as unknown, at home, or retired.

Finally, histologic (cell type) distribution was reviewed for diagnoses of leukemia in the four communities because the various subtypes of leukemia occur with different frequencies in a population. The frequencies of these subtypes in the four communities were compared to statewide incidence trends to assess whether any unusual patterns exist in the areas of evaluation.

1. Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 61,420 people in the United States in 2006 (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.

Because cigarette smoking is the most well-established risk factor for the development of bladder cancer, smoking history was reviewed for each individual diagnosed with this cancer type. 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 also 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, worker exposure reduction strategies, 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 Easthampton from 1982–2000 revealed that the majority of diagnoses in the town were male (74%, n = 35). Males comprised 72% of bladder cancers statewide for this time period. Both males and females in Easthampton were diagnosed at a rate slightly below the expected rate. The mean age at diagnosis was 72 years, which is consistent with statewide bladder cancer incidence.

In Holyoke, the majority of individuals diagnosed with bladder cancer were male (66%, n = 99). Males were diagnosed slightly less often than expected and females were diagnosed about as expected. The average age of individuals diagnosed with bladder cancer during 1982–2000 was 72 years, which is comparable to that observed in the general population. The majority of those diagnosed (97%, n = 144) were age 50 or older at the time of diagnosis.

The majority of bladder cancer diagnoses in Southampton were among males (85%, n = 17). Females experienced bladder cancer at about the rate expected, while males were diagnosed

statistically significantly more often than expected based on the state rate. The average age of individuals diagnosed with bladder cancer in Southampton during 1982–2000 was 71 years. All of the individuals diagnosed were age 50 or older at the time of diagnosis. This pattern is consistent with what would be expected in the general population. The statistically significant elevation in bladder cancer incidence among males in Southampton was the result of increased diagnoses among males aged 55 and older.

The majority of bladder cancer diagnoses in Westfield were male (75%, n = 110). The overall elevation in bladder cancer incidence among males, which was not statistically significant, was the result of increased diagnoses among males aged 55–84 years. Females experienced bladder cancer at approximately the rate expected. The average age of individuals diagnosed with bladder cancer in Westfield during 1982–2000 was 70 years, which is also the mean age at diagnosis statewide. Ninety-five percent (n = 139) were over the age of 50 at the time of diagnosis.

b) Tobacco Use

Of the 20,402 individuals diagnosed with bladder cancer from 1982 to 2000 in Massachusetts, 15,493 reported a smoking status. Of those individuals with a reported smoking status, 67% were current/former smokers and 33% were nonsmokers. Smoking history was unknown for 4,909 (24%) individuals.

Of the 47 individuals in Easthampton who were diagnosed with bladder cancer during the years 1982–2000, 30 reported a smoking status. Seventy percent (n = 21) of those with known smoking history were current/former smokers, which is slightly higher than the 67% of individuals diagnosed with bladder cancer in Massachusetts during 1982–2000 with known smoking history who were current/former smokers. Nine (30%) were nonsmokers. Smoking history was unknown for 17 (36%) individuals.

In Holyoke, 123 of 149 individuals diagnosed with bladder cancer reported a smoking status. Of those 123 individuals with a reported smoking status, 56% (n = 69) were current/former smokers and 44% (n = 54) were nonsmokers. Smoking history was unknown for 26 (17%) individuals.

In Southampton, where there was a statistically significant elevation in bladder cancer among males, 13 of 20 individuals reported a smoking status. Of those 13 individuals with a reported smoking status, 85% (n = 11) were current/former smokers and 15% (n = 2) were nonsmokers. Of the 17 males with bladder cancer, ten reported a smoking status. Of those 10 males with a reported smoking status, eight (80%) were current/former smokers and two (20%) were nonsmokers. Smoking history was unknown for seven (35%) individuals.

In Westfield, 110 out of 147 individuals with bladder cancer reported a smoking status. Of those 110 individuals with a report smoking status, 71% (n = 78) were current/former smokers and 29% (n = 32) were nonsmokers. Smoking history was unknown for 37 (25%) individuals. In Westfield CT 8125, where bladder cancer for males and females combined was statistically significantly elevated, 21 out of 28 individuals reported a smoking status. Of those 21 individuals with a reported smoking status, 86% (n = 18) were current/former smokers and 14% (n = 3) were nonsmokers. Smoking history was unknown for seven (25%) individuals.

In summary, it is likely that smoking played a role in the development of bladder cancer among some residents of Easthampton, Holyoke, Southampton, and Westfield.

c) Occupation

Review of occupation for individuals diagnosed with bladder cancer in Easthampton revealed that at least six individuals (13%) 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. Occupation was reported as retired or unknown for a number of these individuals (17%, n = 8).

In Holyoke, at least eleven individuals (7%) might have worked at a job in which an occupational exposure potentially related to the development of bladder cancer may have been possible. Occupation was reported as retired or unknown for 36% of individuals diagnosed with bladder cancer (n = 54).

In Southampton, at least two individuals (10%) might have worked at a job in which an occupational exposure potentially related to the development of bladder cancer may have been possible. Occupation was reported as retired or unknown for two individuals diagnosed with bladder cancer (10%).

In Westfield, at least 17 individuals (12%) might have worked at a job in which an occupational exposure potentially related to the development of bladder cancer may have been possible. Occupation was reported as retired or unknown for 48% of individuals diagnosed with bladder cancer (n = 70).

2. Esophagus Cancer

The American Cancer Society estimates that esophagus cancer will affect 14,550 people in the United States in 2005 (ACS 2006). Esophagus cancer is three times more common among men than women. It is also three times more common among African-Americans than among whites. The occurrence of esophagus cancer rises with increasing age. It is rarely diagnosed in individuals under 40. The mean age at diagnosis in Massachusetts for the years 1982–2000 was 68 years.

There are several risk factors associated with cancer of the esophagus (ACS 2005a). Esophagus cancer is strongly associated with a history of cigarette smoking, and the risk of developing this cancer type rises with length of tobacco use. In Massachusetts from 1982–2000, 80% of individuals diagnosed with esophagus cancer with known smoking history were current or former smokers. Long term heavy alcohol use, long term heartburn, a diet low in fruits and vegetables and certain vitamins and minerals, and ingestion of lye as a child are also associated with increased risk of esophagus cancer. Studies have revealed that dry cleaning workers have a greater risk of developing esophagus cancer (ACS 2005a). Inhalation of tetrachloroethene (PCE) in the workplace may be responsible for this increased risk.

a) Age and Gender

A review of individuals diagnosed with esophagus cancer in Easthampton from 1982–2000 revealed that most were male (87%, n = 13). Males were diagnosed at about the expected rate and females were diagnosed below the expected rate. The mean age at diagnosis was 70 years,

which is consistent with statewide esophagus cancer incidence. All of the individuals were over the age of 55 at the time of diagnosis.

In Holyoke, the majority of individuals diagnosed with esophagus cancer were also male (64%, n = 37). Both males and females were diagnosed more often than expected, although the elevations were not statistically significant. The average age of individuals diagnosed with esophagus cancer during 1982–2000 was 70 years, which is comparable to that observed in the general population.

In Southampton, males experienced esophagus cancer less than the rate expected and females were diagnosed at approximately the expected rate. The two individuals diagnosed with esophagus cancer in Southampton were both over age 65.

The majority of esophagus cancer diagnoses in Westfield were male (78%, n = 29). Esophagus cancer among males occurred at about the rate expected and less than expected for females. The average age of individuals diagnosed with esophagus cancer in Westfield was 67 years, which is nearly the mean age at diagnosis statewide. All of the individuals were over the age of 40 at the time of diagnosis.

b) Tobacco Use

Of the 6,234 individuals diagnosed with esophagus cancer from 1982 to 2000 in Massachusetts, 5,041 reported a smoking status. Of those individuals with a reported smoking status, 80% were current/former smokers and 20% were nonsmokers. Smoking history was unknown for 1,193 (19%) individuals.

In Easthampton, 12 of the 15 individuals diagnosed with esophagus cancer reported a smoking status. Of those 12 individuals with a reported smoking status, 83% (n = 10) were current/former smokers and 17% (n = 2) were nonsmokers. Smoking history was unknown for three (20%) individuals.

In Holyoke, 51 of the 58 individuals with esophagus cancer reported a smoking status. Of those 51 individuals with a reported smoking history, 75% (n = 38) were current/former smokers and 25% (n = 13) were nonsmokers. Smoking history was unknown for seven (12%) individuals.

Both of the individuals diagnosed with esophagus cancer in Southampton from 1982 to 2000 were current/former smokers.

In Westfield, 28 of the 37 individuals with esophagus cancer reported a smoking status. Of those 28 individuals with a reported smoking status, 86% (n = 24) were current/former smokers and 14% (n = 4) were nonsmokers. Smoking history was unknown for nine (24%).

In summary, it is likely that smoking played a role in the development of esophagus cancer among some residents of Easthampton, Holyoke, Southampton, and Westfield.

c) Occupation

Among the 15 individuals in Easthampton diagnosed with esophagus cancer, an occupation was reported for eight individuals. None of these eight individuals reported occupations where exposures to PCE or secondhand smoke were likely to have occurred, based on the available information.

In Holyoke, at least one individual might have worked at a job in which occupational exposures potentially related to the development of esophagus 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. Occupation was reported as retired or unknown for a number of these individuals (43%, n = 25).

One of the two individuals in Southampton diagnosed with esophagus cancer reported an occupation where exposures to PCE or secondhand smoke were unlikely to occur, based on the available information. The occupation for the other individual was reported as retired.

Among the 37 individuals in Westfield diagnosed with esophagus cancer, an occupation was reported for 25 individuals. None of these 25 individuals reported occupations where exposures to PCE or secondhand smoke were likely to have occurred, based on the available information.

3. *Hodgkin's Disease*

Hodgkin's disease (or Hodgkin's lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin's lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,800 new cases of this disease in the United States in 2006, accounting for less than 1% of all cancer types, and approximately 1,490 deaths (ACS 2006). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS 1999).

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women and more common among whites than blacks. People of Jewish descent appear to be at higher risk of Hodgkin's disease compared to people of non-Jewish descent (Mueller 1996). The disease is relatively rare among children. Two peaks in the age distribution have been observed for Hodgkin's disease. The first peak occurs in young adults usually between the ages of 15 to 40 (typically ages 25–30) and the second peak occurs in adults aged 55 years and above.

The clinical and cellular features of Hodgkin's disease suggest a chronic infectious process (Mueller 1996). The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. Researchers have proposed that among young adults, Hodgkin's disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is probably similar to those of other lymphomas (Mueller 1996). The virus that has been linked most specifically to this disease is the Epstein-Barr virus (EBV). EBV, a herpes virus, is common in the general population and causes mononucleosis or "mono." Approximately 40% to 50% of Hodgkin's disease cases are associated with EBV (Weiss 2000).

Hodgkin's disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin's disease is associated with an infectious agent (Mueller 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the

development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller 1996). Based on an examination of medical and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin's disease does not seem to be caused by genetic, lifestyle (e.g., dietary), or environmental factors (ACS 1999).

a) Age and Gender

Eleven of the 13 individuals diagnosed with Hodgkin's disease in Easthampton during 1982–2000 were between the ages of 15 and 40 or above age 55 at the time of diagnosis. Three individuals were aged 25–30, which is a common age for this cancer type, and there were no children under 16 years diagnosed with Hodgkin's disease. This is generally consistent with what is seen in the general population. Both males and females were diagnosed slightly above the expected rate.

Twenty-one of the 24 individuals diagnosed with Hodgkin's disease in Holyoke were between the ages of 15 and 40 or above age 55. Four individuals were aged 25–30, and there were no children under 16 years diagnosed with Hodgkin's disease. Both females and males were diagnosed at a less than expected rate.

The three individuals diagnosed with Hodgkin's disease in Southampton were between the ages of 30 and 60. Both males and females were diagnosed at a rate very near the expected rate.

In Westfield, 17 of the 21 individuals diagnosed with Hodgkin's disease were between the ages of 15 and 40 or above age 55. Four individuals were aged 25–30, and there were three children under age 15 diagnosed with Hodgkin's disease. Both males and females were diagnosed at a less than expected rate. In Westfield CT 8125, where there was a statistically significant elevation in Hodgkin's disease among females, the six females were between the ages of 12 and 33 when diagnosed.

b) Occupation

Of the 13 individuals in Easthampton diagnosed with Hodgkin's disease, an occupation was reported for 10 individuals. None of these 10 individuals reported occupations that are related to the chemical or woodworking industries, based on the available information.

Of the 24 individuals in Holyoke diagnosed with Hodgkin's disease, an occupation was reported for 17 individuals. None of these individuals reported occupations that are related to the chemical or woodworking industries, based on the available information.

The three individuals in Southampton diagnosed with Hodgkin's disease reported occupations that are unlikely to be related to the chemical or woodworking industries, based on the available information.

In Westfield, at least one adult might have worked at a job in which occupational exposures potentially related to the development of Hodgkin's disease 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. Occupation was reported as retired or unknown for some of these adults (33%, n = 6).

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. Also, 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 Easthampton generally increased with increasing age. The average age of individuals diagnosed with kidney cancer in the town during 1982–2000 was 63 years, while the state mean was 64 years. Eighty-six percent (n = 31) of individuals diagnosed were age 50 or older at the time of diagnosis, which is consistent with the literature. There was one diagnosis of kidney cancer in a child aged 0–19 years versus about 0.5 diagnoses expected for that age group. More males (n = 22) than females (n = 14) were diagnosed with kidney cancer in Easthampton, which is consistent with state and national trends.

In Holyoke, the average age of individuals diagnosed with kidney cancer was 65 years. Eighty-seven percent (n = 71) of individuals diagnosed were over the age of 50 at the time of diagnosis. There were two diagnoses of kidney cancer in children versus about two diagnoses expected in children aged 0–19 years. More males (n = 45) than females (n = 37) were diagnosed with kidney cancer in Holyoke.

In Southampton, the average age of individuals diagnosed with kidney cancer was 59 years. Five of the seven individuals were over the age of 50 at the time of diagnosis. There was one diagnosis of kidney cancer in a child aged 0–19 years versus about 0.2 diagnoses expected for that age group. Males were diagnosed at a rate that was less than expected and females were diagnosed more often than expected, though the elevation was not statistically significant.

In Westfield, the average age of individuals diagnosed with kidney cancer was 66 years. Eighty-eight percent (n = 68) of individuals diagnosed were age 50 or older at the time of diagnosis.

There was one diagnosis of kidney cancer in a child aged 0–19 years versus about 1.3 diagnoses expected for that age group. More males (n = 43) than females (n = 34) were diagnosed with kidney cancer in Westfield.

b) Tobacco Use

Of the 12,328 individuals diagnosed with kidney cancer from 1982 to 2000 in Massachusetts, 9,651 reported a smoking status. Of those individuals with a reported smoking status, 57% were current/former smokers and 43% were nonsmokers. Smoking history was unknown for 2,677 (22%) individuals.

Of the 36 individuals diagnosed with kidney cancer in Easthampton during 1982–2000, 29 individuals reported a smoking status. Of those 29 individuals with a reported smoking status, 59% (n = 17) were current/former smokers and 41% (n = 12) were nonsmokers. Smoking history was unknown for seven (19%) individuals.

In Holyoke, 67 of the 82 individuals diagnosed with kidney cancer reported a smoking status. Of those 67 individuals with a report smoking status, 54% (n = 36) were current/former smokers and 46% (n = 31) were nonsmokers. Smoking history was unknown for 15 (18%) individuals.

In Southampton, six of the seven individuals diagnosed with kidney cancer reported a smoking status. Of those six individuals with a reported smoking status, four were current/former smokers and two were nonsmokers. Smoking status was unknown for one individual.

In Westfield, 65 of 77 individuals with kidney cancer reported a smoking status. Of those 65 individuals with a reported smoking status, 55% (n = 36) were current/former smokers and 45% (n = 29) were nonsmokers. Smoking status was unknown for 12 (16%) individuals.

In summary, it is likely that smoking played a role in the development of kidney cancer among some residents of Easthampton, Holyoke, Southampton, and Westfield.

c) Occupation

Review of occupation for adults diagnosed with kidney cancer in Easthampton revealed that four individuals (11%) 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 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. It is important to note that occupation was reported as retired or unknown for 29% of these adults (n = 10).

In Holyoke, at least 12 adults (15%) might have worked at a job in which an occupational exposure potentially related to the development of kidney cancer may have been possible. Occupation was reported as retired or unknown for 29% of adults diagnosed with kidney cancer (n = 23).

In Southampton, at least one individual might have worked at a job in which an occupational exposure potentially related to the development of kidney cancer may have been possible. Occupation was reported as retired or unknown for two of the six adults diagnosed with kidney cancer.

In Westfield, at least 12 adults (16%) might have worked at a job in which an occupational exposure potentially related to the development of kidney cancer may have been possible. Occupation was reported as retired or unknown for 34% of adults diagnosed with kidney cancer (n = 26).

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 myeloid leukemia (AML), chronic lymphoid leukemia (CLL), chronic myeloid leukemia (CML), and acute lymphoid leukemia (ALL). 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. The average age at diagnosis is 65 years for AML and 70 years for CLL (ACS 2005b, 2005c). For CML, the average age at diagnosis is about 50 years (ACS 2005d). 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). 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, and peaks at about age 85 (Linet and Cartwright 1996). Statewide, the average age of all leukemia diagnoses is 59 years.

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 Easthampton was 50 years. The statewide average age for leukemia diagnoses was 59 years. Sixty-one percent (n = 14) were age

50 or older at the time of diagnosis. Leukemia generally occurred about as expected among males and less often than expected among females. However, five diagnoses occurred in children aged 0–19 years, while approximately three diagnoses would have been expected.

In Holyoke, the average age of individuals diagnosed with leukemia was 63 years, which is comparable to the statewide average age for leukemia diagnoses. Eighty-two percent (n = 80) were age 50 or older at the time of diagnosis. Nine diagnoses occurred in children aged 0–19 years, while approximately 11 would have been expected. From 1988 to 1993, when there was a statistically significant elevation for males and females combined and females alone, the average age for both sexes combined was 59. Six diagnoses occurred in children, while about three were expected during this time period. In CT 8121, where leukemia in males was statistically significantly elevated from 1994 to 2000, the average age among males was 67. One male aged 0–19 years was diagnosed in this census tract.

In Southampton, the average age of individuals diagnosed with leukemia was 61 years. Six of the seven individuals diagnosed were age 50 or older at the time of diagnosis. No diagnoses occurred in children aged 0–19 years, while about one diagnosis would have been expected. Leukemia occurred about as expected among both males and females.

In Westfield, the average age of individuals diagnosed with leukemia was 59 years, which is the same as the statewide average age. Seventy-eight percent (n = 51) were age 50 or older at the time of diagnosis. Eight diagnoses occurred in children aged 0–19 years, which is approximately the number expected (7.8 diagnoses). Leukemia occurred slightly more often among males and occurred less than expected among females.

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 23 individuals diagnosed with leukemia in Easthampton during 1982–2000, 39% were diagnosed with AML subtype, 4% were diagnosed with CLL, 26% were diagnosed with ALL, 13% were diagnosed with CML, and 17% were not specified or were diagnosed with other types of leukemia. This distribution is somewhat similar to that seen statewide, except that the relative

distribution of CLL was lower and the relative distribution of ALL was higher in Easthampton than in the state as a whole. This difference could have been due to the small number of leukemia diagnoses in Easthampton relative to the state. Three of the five children diagnosed with leukemia in Easthampton were diagnosed with the ALL subtype, the most common subtype among children. The remaining two children had two other histology types.

Of the 97 individuals diagnosed with leukemia in Holyoke, 31% were diagnosed with AML subtype, 26% were diagnosed with CLL, 21% were diagnosed with ALL, 7% were diagnosed with CML, and 15% were not specified or were diagnosed with other types of leukemia. Seven of the nine children diagnosed with leukemia in Holyoke were diagnosed with ALL, the most common subtype among children. The remaining two children had two other histology types. Of the 36 individuals diagnosed with leukemia during 1988–1993, when a statistically significant elevation occurred, there were eight cases of AML, six cases of CLL, 10 cases of ALL, two cases of CML, and 10 diagnoses of seven other leukemia types. As previously mentioned, these leukemia cell types are different and have varied risk factors. Four of the six children diagnosed during this time period had ALL, the most common subtype in children. Of the 14 males diagnosed with leukemia in CT 8121, when there was a statistically significant elevation among males from 1994 to 2000, seven were diagnosed with CLL, three with AML, and three were diagnosed with another or non-specified leukemia type. The child diagnosed in CT 8121 during this time period had ALL, the most common type in children.

Of the seven individuals diagnosed with leukemia in Southampton, two were diagnosed with CLL, two were diagnosed with ALL, two were diagnosed with CML, and one was diagnosed with another histology type. There were no children diagnosed with leukemia in Southampton.

Of the 65 individuals diagnosed with leukemia in Westfield, 31% were diagnosed with AML subtype, 22% were diagnosed with CLL, 14% were diagnosed with ALL, 11% were diagnosed with CML, and 23% were not specified or were diagnosed with other types of leukemia. Seven of the eight children diagnosed with leukemia in Holyoke were diagnosed with ALL, the most common subtype among children.

c) Occupation

Review of occupation for adults diagnosed with leukemia in Easthampton revealed that at least one adult 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 adults are not likely to be related to an increased risk of this cancer type. Occupation was reported as retired or unknown for a number of these individuals (33%, n = 6).

In Holyoke, at least one adult might have worked at a job in which occupational exposures potentially related to the development of leukemia may have been possible. Occupation was reported as retired or unknown for 15% of adults diagnosed with leukemia (n = 13).

Among the seven adults in Southampton diagnosed with leukemia, an occupation was reported for three individuals. None of these three individuals reported occupations where exposures to the chemicals listed above were likely to have occurred, based on the available information.

In Westfield, at least two adults might have worked at a job in which occupational exposures potentially related to the development of leukemia may have been possible. Occupation was reported as retired or unknown for 46% of adults diagnosed with leukemia (n = 26).

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 2001b).

a) Age and Gender

The seven individuals diagnosed with liver cancer in Easthampton during 1982–2000 had a mean age of 62 years. This is consistent with the statewide average age of 65. All of the diagnoses were among males.

In Holyoke, the average age of individuals diagnosed with liver cancer was 70 years. There was one diagnosis of an unspecified type of liver cancer in a child. Seventy-four percent of liver cancer diagnoses were among males.

There were no diagnoses of liver cancer in Southampton from 1982 to 2000.

In Westfield, the average age of individuals diagnosed with liver cancer was 65 years. There was one diagnosis of hepatoblastoma in a young child. Hepatoblastoma is a rare type of liver cancer that normally occurs in children under 4 years. Seventy-six percent of liver cancer diagnoses occurred in males.

b) Occupation

Of the five individuals who reported an occupation among the seven diagnosed with liver cancer in Easthampton, none were employed in an occupation that is likely to be related to an increased risk of developing liver cancer. Occupation was unknown for the remaining two individuals.

In Holyoke, at least one individual might have worked at a job in which occupational exposures potentially related to the development of liver cancer may have been possible. Occupation was reported as retired or unknown for 33% of adults diagnosed with liver cancer (n = 6).

In Westfield, at least one individual might have worked at a job in which occupation exposure potentially related to the development of liver cancer may have been possible. Occupation was reported as retired or unknown for 20% of adults diagnosed with liver cancer (n = 4).

7. 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. NHL occurred about equally among males (51%) and females (49%) in Massachusetts from 1982 to 2000. The American Cancer Society estimates that approximately 56,390 Americans will be diagnosed with NHL in 2005, making it the sixth most common cancer in the United States among both men and women, excluding non-melanoma skin cancers (ACS 2005a). 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, 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 Easthampton during 1982–2000 was 62 years, which is consistent with the average age of 63 years seen statewide. Fifty-six percent (n = 25) of NHL diagnoses occurred in males. Males comprised 51% of NHL diagnoses in Massachusetts during the same time period. In CT 8223, where there was a statistically significant elevation among males from 1994 to 2000, the average age was 57 years.

In Holyoke, the average age of individuals diagnosed with NHL was 64 years. There were five diagnoses in children aged 0–19 years from 1982 to 2000, while about three diagnoses were

expected for this age group. NHL diagnoses were almost equally split between males (48%) and females (52%).

In Southampton, the average age of individuals diagnosed with NHL was 61 years. Three of the diagnoses occurred in males and four occurred in females.

In Westfield, the average age of individuals diagnosed with NHL was 63 years. There were two diagnoses in children from 1982 to 2000, while about two diagnoses were expected. Fifty-four percent of NHL diagnoses occurred in males and 46% occurred in females.

b) Occupation

Review of occupational information for individuals diagnosed with NHL in Easthampton revealed that at least six individuals (13%) 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, and occupation was reported as retired or unknown for 22% of individuals (n = 10).

In Holyoke, at least eight individuals (6%) diagnosed with NHL might have worked a job in which occupational exposures potentially related to the development of NHL may have been possible. Occupation was reported as retired or unknown for 33% of adults diagnosed with NHL (n = 44).

In Southampton, one of the four individuals diagnosed with NHL who reported an occupation might have worked a job in which occupational exposures potentially related to the development of NHL may have been possible. Occupation was reported as retired or unknown for three of the seven individuals diagnosed with NHL.

In Westfield, at least five individuals (5%) diagnosed with NHL might have worked a job in which occupational exposures potentially related to the development of NHL may have been possible. Occupation was reported as retired or unknown for 39% of adults diagnosed with NHL (n = 36).

8. Pancreatic Cancer

The risk of developing pancreatic cancer increases with age, and the majority of cases occur between age 60 and 80. Men are approximately 30% more likely to develop pancreatic cancer than are women (ACS 2000b), although women in Massachusetts were diagnosed slightly more often than men from 1982 to 2000. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2000b). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al. 1996).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS 2000b, Ji et al. 2000, Porta et al. 1999). Exposure to certain dyes and certain chemicals related to gasoline, in addition to asbestos and ionizing radiation, have also been associated with the development of pancreatic cancer in some studies, however, other studies have found no link between these agents and pancreatic cancer (ACS 2000b, Anderson et al. 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons) and polycyclic aromatic hydrocarbons, nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this finding (Ojajarvi et al. 2000).

a) Age and Gender

A review of individuals diagnosed with pancreatic cancer in Easthampton from 1982–2000 revealed that slightly more females (53%) than males (47%) were diagnosed. Both males and females were diagnosed slightly more often than expected, although the elevations were not statistically significant. The mean age at diagnosis was 70 years, which is the same as the average age for statewide pancreatic cancer incidence. Thirty-three of the 34 individuals were age 50 or older at the time of diagnosis. Among the individuals diagnosed from 1994 to 2000, when there was a statistically significant elevation townwide for males and females combined, the mean age was 70.

In Holyoke, most of the individuals diagnosed with pancreatic cancer were female (59%, n = 58). Males were diagnosed approximately as expected, and females were diagnosed slightly more often than expected, although not statistically significantly. The average age of individuals diagnosed with pancreatic cancer was 71 years. All but two of the individuals were age 50 or older at the time of diagnosis. In CT 8121, where there was a statically significant elevation among females from 1988 to 1993, the average age was also 71.

In Southampton, males experienced pancreatic cancer less than the rate expected and females were diagnosed slightly higher than the expected rate. There were two males and five females diagnosed. The seven individuals diagnosed with pancreatic cancer in Southampton were all over the age of 60.

In Westfield, the majority of pancreatic cancer diagnoses were male (59%, n = 39). Pancreatic cancer among males occurred slightly higher than the rate expected and less than expected for females. The average age of individuals diagnosed with pancreatic cancer was 68 years. Most of the individuals (92%) were age 50 or older at the time of diagnosis.

b) Tobacco Use

Of the 11,549 individuals diagnosed with pancreatic cancer from 1982 to 2000 in Massachusetts, 8,523 reported a smoking status. Of those individuals with a reported smoking status, 57% were current/former smokers and 43% were nonsmokers. Smoking history was unknown for 3,026 (26%) individuals.

In Easthampton, 22 out of 34 individuals diagnosed with pancreatic cancer during the years 1982–2000 reported a smoking status. Of those 22 individuals with a reported smoking status, 36% (n = 8) were current/former smokers and 64% (n = 14) were nonsmokers. Smoking history was unknown for 12 (35%) individuals. For the time period 1994–2000, when there was a statistically significant elevation townwide, 15 out of 21 individuals reported a smoking status. Of those 15 with a reported smoking status, 33% (n = 5) were current/former smokers and 67% (n = 10) were nonsmokers. The townwide elevation from 1994 to 2000 was in part due to a statistically significant elevation among males in CT 8224 during that time period. Of the nine males diagnosed with pancreatic cancer in CT 8224 from 1994 to 2000, six reported a smoking

status. Of those six males with a smoking status, four were current/former smokers and two were nonsmokers.

In Holyoke, 81 out of 99 individuals diagnosed with pancreatic cancer reported a smoking status. Of those 81 individuals with a reported smoking status, 48% (n = 39) were current/former smokers and 52% (n = 42) were nonsmokers. Smoking history was unknown for 18 (18%) individuals. In CT 8121, where there was a statistically significant elevation among females from 1988 to 1993, 10 out of 12 females diagnosed with pancreatic cancer reported a smoking status. Of those 10 individuals with a reported smoking status, 50% (n = 5) were current/former smokers and 50% (n = 5) were nonsmokers.

Of the seven individuals diagnosed with pancreatic cancer in Southampton, six reported a smoking status. Of those six individuals with a reported smoking status, two were current/former smokers and four were nonsmokers.

In Westfield, 49 out of 66 individuals diagnosed with pancreatic cancer reported a smoking status. Of those 49 individuals with a reported smoking status, 53% (n = 26) were current/former smokers and 47% (n = 23) were nonsmokers. Smoking history was unknown for 17 (26%) individuals.

In summary, it is likely that smoking played a role in the development of pancreatic cancer among some residents of Easthampton, Holyoke, Southampton, and Westfield.

c) Occupation

Review of occupational information for individuals diagnosed with pancreatic cancer in Easthampton revealed that at least two individuals might have worked at a job in which occupational exposures potentially related to the development of pancreatic 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, and occupation was reported as retired or unknown for 29% of individuals (n = 10).

In Holyoke, at least 13 individuals (13%) diagnosed with pancreatic cancer might have worked a job in which occupational exposures potentially related to the development of pancreatic cancer

may have been possible. Occupation was reported as retired or unknown for 32% of individuals diagnosed with pancreatic cancer (n = 32).

In Southampton, two of the five individuals diagnosed with pancreatic cancer who reported an occupation might have worked a job in which occupational exposures potentially related to the development of pancreatic cancer may have been possible. Occupation was reported as retired or unknown for two of the seven individuals diagnosed with pancreatic cancer.

In Westfield, at least seven individuals (11%) diagnosed with pancreatic cancer might have worked a job in which occupational exposures potentially related to the development of pancreatic cancer may have been possible. Occupation was reported as retired or unknown for 38% of individuals diagnosed with pancreatic cancer (n = 25).

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 the residences of individuals diagnosed with the eight cancer types from 1982 to the present was conducted, particularly as the geographic distribution relates to areas of environmental concern. In particular, the analysis focused on the geographic distribution of individuals living in the Plains area of Easthampton, where residents likely received most of their drinking water from the Hendrick Street Wellfield and Pines Well, and individuals diagnosed within the potential extent of TCE-contaminated groundwater. The inclusion of individuals living within the potential extent of TCE-contaminated groundwater is very conservative and may include individuals who did not consume TCE-contaminated drinking water. In addition to the aforementioned individuals, residents who were diagnosed with one of the eight cancer types from 2001 to the present and lived in the Plains area of Easthampton or within the potential extent of TCE-contaminated groundwater were included in the qualitative evaluation of geographic distribution.

Place of residence at the time of diagnosis was mapped for each individual diagnosed with one of the eight cancer types in order to assess any possible geographic concentrations of diagnoses in relation to each other or in relation to opportunities for TCE exposure or other potential locations of environmental concern (i.e., MassDEP 21E hazardous material and oil releases) located in

Easthampton, Holyoke, Southampton, or Westfield. 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 one of the eight cancer types, no apparent concentrations of cancer diagnoses (of any type) were observed in any one area of Easthampton, Holyoke, Southampton, or Westfield that were not associated with areas of higher population density. For example, many of the males residing in Easthampton CT 8223 who were diagnosed with NHL from 1994 to 2000, when there was a statistically significant elevation, lived in a densely populated area. Further evaluation revealed that these individuals were diagnosed with histological types of NHL that are consistent with the distribution of histologies observed statewide (i.e., most of these males were diagnosed with the most common type of NHL diagnosed in the state). Also, the age distribution of the individuals was consistent with the statewide age distribution.

The geographic distribution was reviewed for all statistically significant elevations in the four communities. To summarize, the statistically significant elevations observed in the four communities were as follows:

- bladder cancer among males in Southampton from 1982 to 2000,
- bladder cancer among males and females combined and males alone in Southampton from 1988 to 1993,
- bladder cancer among males and females in Westfield CT 8125 from 1982 to 2000,
- Hodgkin's disease among females in Westfield CT 8125 from 1982 to 2000,
- leukemia among males and females combined and females alone in Holyoke from 1988 to 1993,

- leukemia among males in Holyoke CT 8121 from 1994 to 2000,
- NHL among males in Easthampton CT 8223 from 1994 to 2000,
- NHL among males and females combined and females alone in Westfield from 1982 to 2000,
- pancreatic cancer among males and females combined who were diagnosed in Easthampton from 1994 to 2000,
- pancreatic cancer among males in Easthampton CT 8224 from 1994 to 2000, and
- pancreatic cancer among females in Holyoke CT 8121 from 1988 to 1993.

There were no geographic patterns observed for any of these elevations that were not associated with areas of high population density.

None of the eight cancer types were statistically significantly elevated from 1982 to 2000 in CT 8224, which includes the Plains area in southern Easthampton. Because of their proximity to the wells, Plains area residents likely received more drinking water from the Easthampton municipal wells that were contaminated with TCE, relative to Easthampton residents in other areas of town. Seven of the eight cancer types evaluated were diagnosed among Plains area residents. Of the six cancer types that have possible associations with TCE exposure based on the scientific literature, there were three diagnoses of bladder cancer, two diagnoses of Hodgkin's disease, four diagnoses of kidney cancer, six diagnoses of leukemia, no diagnoses of liver cancer, and six diagnoses of NHL in the Plains area. No apparent geographic concentrations of individuals diagnosed with any of the eight cancer types from 1982 to the present were noted in the Plains area.

No apparent geographic concentrations of individuals diagnosed with any of the eight cancer types from 1982 to the present were noted in the areas of Holyoke, Southampton, and Westfield that are within the potential extent of TCE-contaminated groundwater. Most individuals in this area who were diagnosed with one of the cancer types were located where population density is greatest. In addition, no unusual geographic patterns were noted in the Dupuis Road

neighborhood of Holyoke, where the community expressed concerns that residents in the 1950s were exposed to the combustion products of PCBs when wastes were burned at a Dupuis Road property.

No other unusual spatial patterns or concentrations of cases at the neighborhood level that would suggest a common factor (environmental or non-environmental) 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 each of the four communities, the majority of individuals diagnosed with each type of cancer tended to be located in areas of the town where population and housing density are greatest.

Information about which Holyoke, Southampton, and Westfield residences within the potential extent of TCE-contaminated groundwater had private wells and which residences were supplied with municipal water (both TCE-impacted and those not impacted by environmental contaminants) was obtained from the MassDEP and local water departments. This information was compared with available residential history information for the individuals who were diagnosed with one of the eight cancer types between 1982 and 2000 in order to determine how long the individuals lived at their residence prior to diagnosis. Information for residential histories was obtained from annual resident lists for Holyoke (City of Holyoke 1970–1998) and Westfield (City of Westfield 1970–1998) and from the Hampden County and Hampshire County registries of deeds (Office of the Secretary of the Commonwealth 2005). Residential histories were constructed for each individual who lived within the potential extent of TCE-contaminated groundwater at the time of diagnosis. Residential histories were also constructed for individuals diagnosed while living in the Plains area of southern Easthampton where residents received more municipal water from the Hendrick Street Wellfield and Pines Well relative to other Easthampton residents. Although it is not possible to determine what may have caused any one person's diagnosis with cancer, the length of time in which an individual lived in a particular residence can help determine the importance that their location might have in terms of exposure to a potential environmental source. The residential history information is discussed in Section VIII along with information about the various sources of drinking water for residences within the potential extent of TCE-contaminated groundwater. Available risk factor information for those

individuals who were diagnosed with one of the eight cancer types is also included in the analysis.

VIII. DISCUSSION

This public health assessment provides a review of possible environmental exposures related to TCE in the Barnes Aquifer and an evaluation of cancer incidence in Easthampton, Holyoke, Southampton, and Westfield in western Massachusetts. In the 1950s, TCE wastes were released at two Holyoke residential properties and the former Southampton Sanitary Engineering in Southampton. PCB wastes were also released at the two residential properties in Holyoke. The MassDEP believes that the wastes originated from the former General Electric facility on Jackson Street in Holyoke. TCE contamination now extends approximately 4.5 miles through the Barnes Aquifer from the source properties north to municipal wells in Easthampton. This evaluation was initiated based on community concerns about possible environmental exposure in relation to TCE contamination in public and private drinking water wells whose source is the Barnes Aquifer and community concerns about cancer. Community concerns also included possible exposures to PCBs in soil, PCBs and benzene in drinking water, and dioxins in air.

The MDPH evaluated cancer incidence data for Easthampton, Holyoke, Southampton, and Westfield and for the census tracts within those communities where some residents were at risk of exposure to TCE-contaminated drinking water from the Barnes Aquifer. Available environmental information was reviewed for Barnes Aquifer drinking water and the source properties to determine possible pathways of exposure for residents. In addition, the geographic pattern of cancer diagnoses was evaluated at the neighborhood level to identify any unusual concentrations of diagnoses, with a particular focus on neighborhoods where residents were at risk of exposure to TCE.

There are completed exposure pathways that occurred in the past related to TCE contamination in the Barnes Aquifer. Exposure to TCE in municipal drinking water from the Hendrick Street Wellfield and Pines Well in the past probably occurred for many Easthampton residents from the early 1980s to about 1997. A potential exposure pathway could have occurred from as early as the 1960s to the 1980s. For about 600 residents in western Holyoke who were supplied with drinking water from the Pequot Well, there is a potential exposure pathway from 1974 to 1980

and a completed exposure pathway from 1980 to 1987. However, upon considering conservative exposure doses for both of these scenarios, adverse health effects or unusually increased cancer risk due to exposure to past contamination in municipal drinking water seemed unlikely. Present and future exposure pathways related to TCE in municipal drinking water were eliminated because Holyoke Water Works closed the Pequot Well in 1987 and Easthampton installed a water treatment plant in 1997.

Past exposure to TCE in private well water from the contaminated section of the Barnes Aquifer occurred for some residents of Holyoke and Southampton (TCE was not detected above the drinking water comparison value in private wells in Easthampton and Westfield). The majority of residents with TCE-contaminated private wells are no longer being exposed because they accepted bottled water and whole house carbon filters or connected their households to a municipal water supply not impacted by environmental contaminants. However, since a filter requires maintenance, there is the potential for present or future exposures if residents do not properly maintain them or if they use unfiltered water. There also exists a potential exposure pathway for a small number of residents who declined to have their private wells tested.

The maximum TCE concentration (34.2 ppb) detected in any drinking water sample (since 1980 in municipal wells and since 1997 in private wells) was from a private well in Southampton. However, based on the contaminant levels detected, the frequency and duration of contact assumed, and a review of the scientific literature, it is unlikely that exposures to TCE in Barnes Aquifer drinking water resulted in adverse health effects.

Past exposures to PCBs in surface soil may have been possible for children who lived at two Holyoke residential properties where wastes were released and who may have played in surface soil there. However, upon considering conservative exposure dose scenarios, adverse health effects or unusually increased cancer risk due to past exposure to PCBs were unlikely. Since PCB-contaminated soil was removed from the two residential properties and replaced with clean soil, in addition to the placement of Activity and Use Limitation (AUL) deed restrictions on the properties, present and future exposures to PCBs in soil were eliminated as exposure pathways.

Community members expressed concerns that PCBs and benzene might have migrated via groundwater to drinking water wells in the same way that TCE migrated via groundwater to

drinking water wells. Because PCBs and benzene have not been demonstrated to have migrated from the source properties via groundwater, exposures to PCBs or benzene in drinking water were eliminated as past, present, and future exposure pathways for residents.

Residents also expressed concerns that individuals living at or near the Dupuis Road property in Holyoke in the 1950s were exposed to the combustion products of PCBs in smoke when wastes were reportedly burned. However, air monitoring data were not available for that time. Also, surface soil data were not available for PCB combustion products that could have been deposited at neighboring properties from ambient air, which could help to evaluate potential past exposure opportunities. Because no environmental data were available, the likelihood of adverse health effects that might result from potential past inhalation exposure to PCB combustion products in ambient air could not be evaluated quantitatively. Instead, the pattern of cancer was evaluated for the Dupuis Road neighborhood, and no unusual geographic patterns or concentrations of diagnoses were noted.

Cancer in general has a long period of development or latency period (i.e., the interval between first exposure to a disease-causing agent and the appearance of symptoms of the disease [Last 1995]). In particular, solid tumors such as bladder, kidney, and liver cancer generally have a long latency period that ranges from at least 10 to 20 years and may be as long as 50 years (Levy and Wegman 1995). Because the TCE waste released in Holyoke and Southampton could have reached groundwater and affected private wells as early as the 1950s, the community has expressed concern that Massachusetts Cancer Registry (MCR) data are not available prior to 1982, when the MCR first began collecting cancer diagnoses data. Although earlier data are not available, it is still useful to analyze cancer incidence data from 1982 to 2000 due to the long latency periods of some of the cancer types evaluated. In addition, if exposure to TCE resulted in a trend in cancer incidence prior to 1982 and TCE exposure continued into more recent years, one would expect to observe a trend in the years following 1982.

Using data from the MCR, the MDPH evaluated the incidence of eight cancer types that were selected based on a potential association with TCE and residents' concerns about particular cancer types. Five census tracts were chosen for evaluation because they include residents who were at risk of exposure to TCE in Barnes Aquifer drinking water. It is important to note that

while most residents of Easthampton CT 8223 and CT 8224 were at times at risk of exposure to TCE from the Barnes Aquifer prior to 1997, most residents of Holyoke CT 8121, Southampton CT 8225, and Westfield CT 8125 were not at risk of exposure (i.e., estimates show that about 1,900 of the 26,416 residents of the three latter census tracts were at risk of exposure to TCE in drinking water from the Barnes Aquifer).

The time period of the cancer incidence analysis, 1982 to 2000, includes the most recent and complete cancer incidence data available from the MCR at the time of this evaluation. No consistent trends in elevations were observed for the eight cancer types evaluated from 1982 to 2000 for Easthampton, Holyoke, Southampton, and Westfield and for the census tracts where some residents were at risk of exposure to TCE in drinking water. A detailed discussion of some cancer types that were statistically significantly elevated in census tracts where some residents were at risk of exposure to TCE in Barnes Aquifer drinking water follows.

From 1994 to 2000, NHL among males in Easthampton CT 8223 was statistically significantly elevated (10 diagnoses observed versus 4.3 expected) and pancreatic cancer among females in the same census tract was borderline statistically significantly elevated (7 observed vs. 2.8 expected) (Table 9d). The geographic pattern of males diagnosed with NHL and females diagnosed with pancreatic cancer during this time period closely matched areas of population density. Males diagnosed with NHL had a variety of histological types, which was consistent with the distribution of histologies observed statewide. The average age at the time of diagnosis for the males diagnosed with NHL and the women diagnosed with pancreatic cancer was consistent with statewide trends. During the two earlier time periods evaluated (1982–1987 and 1988–1993), NHL among males and pancreatic cancer among females in CT 8223 occurred about as expected or less than expected.

From 1994 to 2000, pancreatic cancer among males in Easthampton CT 8224 was statistically significantly elevated (9 diagnoses observed versus 3.7 expected) (Table 10d). Based on a review of available risk factor information, smoking may have played a role in some individuals' diagnoses. Easthampton CT 8224 includes the Plains area where residents, because of their proximity to contaminated municipal wells and the way Easthampton municipal water is distributed, likely received more water from the Hendrick Street Wellfield and Pines Well

relative to the rest of Easthampton. Three of the nine males diagnosed during this time period lived in the Plains area. One of the three individuals likely lived in the area less than 5 years prior to diagnosis; therefore, their diagnosis was not likely related to place of residence. Of the two remaining males, one was a current/former smoker, which is the most important risk factor for pancreatic cancer. During the two earlier time periods evaluated (1982–1987 and 1988–1993), pancreatic cancer among males in CT 8224 occurred less than expected and about as expected, respectively.

A statistically significant elevation in leukemia diagnoses among males occurred in Holyoke CT 8121 from 1994 to 2000 (14 diagnoses observed versus 7.4 expected) (Table 12d). Based on the location of their residences at the time of diagnosis, none of the 14 individuals were at risk of exposure to TCE in Barnes Aquifer drinking water from the Pequot Well, Coronet Homes Well, or private wells. The geographic distribution of leukemia diagnoses corresponded to the distribution of population in this census tract. A variety of leukemia histologies were diagnosed among the males, and the average age at diagnosis was 67 years old. During the two earlier time periods evaluated (1982–1987 and 1988–1993), leukemia among males occurred about as expected and less than expected.

Among females in Holyoke CT 8121, a statistically significant elevation for pancreatic cancer was observed from 1988 to 1993 (12 diagnoses observed versus 6.2 expected) (Table 12c). The average age at diagnosis was consistent with statewide trends, and five of the 10 women with a known smoking history were current/former smokers. The geographic distribution of residences of women diagnosed with pancreatic cancer during this time period corresponded to the distribution of the overall population. Of the 12 females, 11 were not at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences at the time of diagnosis. It is unknown whether the remaining individual, who resided within the potential extent of TCE-contaminated groundwater, could have been exposed to TCE. In CT 8121 during the other two time periods (1982–1987 and 1994–2000), about one or two more cases of pancreatic cancer among women in CT 8121 were observed over the expected number, but neither elevation was statistically significant.

Bladder cancer among Southampton males was statistically significantly elevated during the 1982–2000 time period, with the overall elevation attributed to elevations during the two earliest time periods (6 diagnoses observed versus 2.8 expected during 1982–1987, and 8 diagnoses observed versus 3.0 expected during 1988–1993, the latter of which was statistically significant) (Tables 13b–13c). Bladder cancer occurred slightly less often than expected among males from 1994 to 2000 (3 diagnoses observed versus 3.8 expected) (Table 13d). Overall, among the 14 individuals diagnosed with bladder cancer during the two earliest time periods, eight reported a smoking history, and six of these eight were current or former smokers. The geographic distribution of bladder cancer among males in Southampton was generally consistent with population density. Ten of the 14 males diagnosed from 1982 to 1993 were not at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences at the time of diagnosis. It is unknown whether three of the males, who resided at the time of diagnosis within the potential extent of TCE-contaminated groundwater, could have been exposed. The remaining individual could have been exposed to TCE; therefore, if exposure did occur, it could have played a role in the development of bladder cancer. Two of the four remaining males reported smoking history information and were both current/former smokers. The average age at diagnosis of the four males was 73 years old, which is consistent with statewide trends. Two of the males were likely long-term residents (15+ years), and residential histories were unknown for the other two males.

In Westfield CT 8125, bladder cancer was statistically significantly elevated among males and females from 1982 to 2000 (28 diagnoses observed versus 18.1 expected) (Table 15a). The elevation was largely due to elevations among males for each of the three smaller time periods, when two to four more males than expected were diagnosed with bladder cancer (Tables 15b–15d). None of the elevations among males were statistically significant. Of the 22 males diagnosed from 1982 to 2000 with bladder cancer in CT 8125, 21 were not at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences at the time of diagnosis. Most of the males resided in the southern part of the census tract, where population and housing density are greatest. It is unknown whether the remaining individual, who resided within the potential extent of TCE-contaminated groundwater, may have been exposed to TCE from Barnes Aquifer drinking water. According to a review of available risk factor information, this individual reported being a current/former smoker. Therefore, smoking may have played a role

in the development of bladder cancer for this individual. Among the 22 males in CT 8125 who were diagnosed with bladder cancer from 1982 to 2000, 17 had a known smoking history, and 14 of the 17 reported being current/former smokers. Therefore, information on bladder cancer in this census tract is consistent with patterns seen elsewhere in the state and in the scientific literature.

Hodgkin's disease among females in Westfield CT 8125 was statistically significantly elevated from 1982 to 2000 (6 diagnoses observed versus 2.1 expected) (Table 15a). The diagnoses were fairly evenly distributed through time, with three, one, and two diagnoses occurring in the first, middle, and most recent time periods, respectively (Tables 15b–15d). Most of the individuals lived in the densely populated southern area of the census tract and none were at risk of exposure to TCE from the Barnes Aquifer, based on the location of their residences at the time of diagnosis. According to a review of available risk factor information, the individuals were diagnosed with Hodgkin's disease between the ages of 12 and 33, which is consistent with the peak in diagnoses that typically occurs among young adults.

In studies of community exposure to TCE in drinking water, the strongest support for increased cancer incidence is for leukemia (Wartenberg et al. 2000). For the town of Easthampton, where most residents were likely at risk of some exposure to TCE-contaminated municipal water in the past, there were fewer diagnoses of leukemia than expected from 1982 to 2000, based on the state rate of leukemia. Leukemia diagnoses in CT 8223 occurred less often than expected during all time periods. In CT 8224, which includes the Plains area that received more municipal water from TCE-contaminated wells relative to unaffected municipal wells, there was about one additional leukemia diagnosis above the expected number from 1982 to 2000. Leukemia was diagnosed at about the expected rate in CT 8224 for each smaller time period. Therefore, although many individuals in Easthampton could have been exposed to some level of TCE in municipal drinking water, it does not appear that exposures were of sufficient concentration or duration to result in elevated leukemia diagnoses. In addition, none of the approximately 1,000 Holyoke residents who lived at a residence supplied by the Pequot Well or Coronet Homes Well, municipal wells that were contaminated with TCE, were diagnosed with leukemia from 1982 to the present.

In summary, analysis of the geographic distribution of residences of individuals diagnosed with cancer, available risk factor information, and residential history information did not reveal any atypical patterns that would suggest that a common factor is related to the incidence of cancer in Easthampton, Holyoke, Southampton, Westfield, or in the census tracts of concern. That is, no unusual concentrations of individuals diagnosed with the eight cancer types evaluated were observed among the populations at potential risk of TCE exposure or in any other areas of the four communities. In general, cancer patterns observed in Easthampton, Holyoke, Southampton, and Westfield were similar to those seen in the general population and in Massachusetts. Data reviewed suggest that smoking likely played some role in the diagnoses of certain cancers (bladder, esophageal, kidney, and pancreatic cancers) among some individuals. Also, occupational exposures may have played a role for some individuals in the development of the eight cancer types. However, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in the four communities due to incomplete information for some risk factors (e.g., occupation).

In all, the information reviewed and analyzed for this public health assessment included available environmental data, cancer incidence data, available risk factor information for individuals diagnosed with cancer, residential history information, and a review of the relevant scientific literature. Based on this information, it does not appear that a common factor (environmental or non-environmental) played a major role in the overall incidence of cancer in the census tracts where some residents were at risk of exposure to TCE from the Barnes Aquifer or in Easthampton, Holyoke, Southampton, or Westfield as a whole during the 19-year time period, 1982–2000.

IX. ATSDR CHILD HEALTH CONSIDERATIONS

The 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 Easthampton, Holyoke, Southampton, and Westfield are discussed in Section VII ("Analysis of Cancer Incidence") of this report. As discussed previously, risk of exposure to TCE in municipal drinking water existed in the past for children in Easthampton and some children in west Holyoke prior to treatment and well closures. Risk of exposure to TCE in private well water also existed in the past for some children living in certain parts of Holyoke, Southampton, and Westfield. At present, most children are not at risk of exposure to TCE in private well water because most residences have whole house filters or are now connected to uncontaminated municipal water. A potential exposure pathway still exists for a small number of children living at residences where private well testing was refused or where residents use unfiltered water.

In addition, exposure to contaminants in air and soil may have been possible in the past for children living at or near the Holyoke residential properties where wastes were released. However, it is unlikely that anyone would have had contact with soil at the properties for a sufficient frequency and duration of time to result in health effects. Present and future exposures are not of concern because PCB-contaminated surface soils were removed and Activity and Use Limitation deed restrictions were placed on both properties.

X. LIMITATIONS

There are several limitations encountered when analyzing environmental data. As a result, 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 cancer types 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. For example, 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 Easthampton, Holyoke, Southampton, and Westfield.

XI. CONCLUSIONS

- In the past, Easthampton residents and some western Holyoke residents were at risk of exposure to TCE in municipal drinking water from the Barnes Aquifer. Some residents of western Holyoke and eastern Southampton were at risk of exposure to TCE in Barnes Aquifer drinking water from private wells. Based on the contaminant levels detected since 1980 in municipal wells and since 1997 in private wells, the frequency and duration of contact assumed, and a review of the scientific literature, it is unlikely that exposures resulted in adverse health effects.

- Holyoke and Southampton residents with TCE-contaminated private well water are not at risk of exposure if they properly maintain whole house charcoal filters or connected their households to unaffected municipal water. However, a potential exposure pathway could remain if residents do not properly maintain their filters or use unfiltered water.

- Children who may have played in surface soil at two Holyoke residential properties may have been at risk of exposure to PCBs; however, based on the levels detected and the frequency and duration of contact assumed, it is unlikely that potential exposures could have resulted in adverse health effects. The contaminated soils have since been removed.

- Potential exposures to PCBs in private well water were ruled out because PCBs have not been demonstrated to have migrated via groundwater from the release properties.

- No consistent trends in elevations were observed from 1982 to 2000 for any of the eight cancer types [Hodgkin's disease, leukemia, non-Hodgkin's lymphoma (NHL), and cancers of the bladder, esophagus, kidney, liver, and pancreas] in Easthampton, Holyoke, Southampton, Westfield, or the census tracts where some residents were at risk of exposure to TCE in drinking water from the Barnes Aquifer.

- In Easthampton CT 8223, NHL among males was statistically significantly elevated from 1994 to 2000. The histological types of NHL were consistent with the statewide distribution, and no unusual geographic concentrations of diagnoses were observed. There was a statistically significant elevation in pancreatic cancer among males and females townwide and males in Easthampton CT 8224 from 1994 to 2000. Based on available risk factor information, smoking may have played a role in some individuals' diagnoses.

- In Holyoke CT 8121, males were diagnosed with leukemia statistically significantly more often than expected from 1994 to 2000. Based on the location of their residences at the time of diagnosis, none of the 14 males were at risk of exposure to TCE in Barnes Aquifer drinking water. Pancreatic cancer was statistically significantly elevated for females from 1988 to 1993. Eleven of the 12 females were not at risk of exposure to TCE from the Barnes Aquifer, based on their residences. It is unknown whether the remaining individual could have been exposed to TCE.

- In Southampton, a statistically significant elevation in bladder cancer among males from 1982 to 2000 was attributed to elevations during two time periods, 1982–1987 and 1988–1993. Ten of the 14 males diagnosed from 1982 to 1993 were not at risk of exposure to

TCE from the Barnes Aquifer, based on the location of their residences. It is unknown whether three of the males could have been exposed. The remaining individual could have been exposed to TCE; therefore, if exposure did occur, it could have played a role in the development of bladder cancer. Based on available risk factor information, it is likely that smoking played a role in the development of bladder cancer among some males.

- In Westfield CT 8125, a statistically significantly elevation in bladder cancer for males and females from 1982 to 2000 was due to elevations among males during the three smaller time periods. Of the 22 males diagnosed, 21 were not at risk of exposure to TCE from the Barnes Aquifer, based on the locations of their residences. It is unknown whether the remaining individual could have been exposed; however, based on available risk factor information, it is likely that smoking played a role in the individual's diagnosis. Based on their residences, none of the six females diagnosed with Hodgkin's disease, which was statistically significantly elevated from 1982 to 2000, were at risk of exposure to TCE from the Barnes Aquifer.

- A review of the geographic distribution of residences of individuals diagnosed with any of the eight cancer types in Easthampton, Holyoke, Southampton, and Westfield revealed no apparent spatial patterns at the neighborhood level. Further, no unusual concentrations of individuals diagnosed with cancer were observed among residents potentially exposed to TCE or in any other area of the four communities.

- Residents living in the Dupuis Road neighborhood in Holyoke could have been exposed to air contaminants when PCB wastes were reportedly burned at a property there. Because no environmental data were available for that time, it was not possible to quantitatively evaluate the potential for adverse health effects. However, a qualitative review of cancer diagnoses in the Dupuis Road neighborhood revealed no unusual pattern or concentration of diagnoses.

- Based on the information reviewed in this evaluation, it does not appear that a common factor (environmental or non-environmental) played a major role in the overall incidence of cancer in the census tracts where some residents were at risk of exposure to TCE from the Barnes Aquifer or in the communities of Easthampton, Holyoke, Southampton, and

Westfield as a whole during the 19-year time period, 1982–2000. Information reviewed included available environmental data, risk factor information for individuals diagnosed with cancer, residential history information, and a review of the relevant scientific literature.

The 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 the MDPH evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to the cancer types evaluated in this analysis, the ATSDR would classify the TCE-contaminated section of the Barnes Aquifer as posing an indeterminate public health hazard in the past due to incomplete historical sampling data for private wells prior to 1997. Most exposure opportunities have been eliminated through municipal water treatment and well closures, connections to municipal water not impacted by contaminants, and whole house charcoal filters; however, for some residents with private wells (i.e., residents of a few households that declined testing, residents who might not properly maintain their filters, and residents who use unfiltered water), the ATSDR would classify the contaminated section of the Barnes Aquifer as posing an indeterminate public health hazard at present and in the future.

XII. RECOMMENDATIONS

1. Holyoke, Southampton, and Westfield residents who continue to use private wells without whole house carbon filters in the vicinity of the contaminated section of the Barnes Aquifer, including the small number of Easthampton residents on Fort Hill Road that use private wells, should test their wells to ensure that TCE levels remain below the U.S. EPA Maximum Contaminant Level (MCL) of 5 ppb.

2. If TCE is detected in a private well above the U.S. EPA MCL of 5 ppb, residents should properly maintain a whole house carbon filter or connect to municipal water, if possible. The MDPH supports the City of Holyoke and the Town of Southampton exploring the feasibility of connecting remaining Holyoke homes with TCE-contaminated private wells to municipal water.

3. The Easthampton Board of Health requires private well testing at the time of property transfer for all residences with private wells and for newly constructed wells. The MDPH recommends that the City of Holyoke and the Town of Southampton consider a similar requirement and/or notification of the existence of a whole house charcoal filter at the time of property transfer for residences in the vicinity of the contaminated section of the Barnes Aquifer. The MDPH is available to offer guidance to help determine the geographic boundaries of such a requirement.

4. The MDPH recommends that Holyoke and Southampton evaluate the feasibility of a testing and approval process for new private well construction in the vicinity of the TCE-contaminated section of the Barnes aquifer. The MDPH is available to offer guidance to help determine the geographic boundaries of such a requirement.

5. In order to address a data gap regarding potential past exposure for residents to air contaminants during the reported burning of PCB wastes, the MDPH recommends that the MassDEP continue its efforts toward identifying Potentially Responsible Party(s) that could conduct or oversee surface soil sampling and analysis for the combustion products of PCBs (i.e., chlorinated dibenzodioxins and chlorodibenzofurans) at residential properties neighboring the Dupuis Road property in Holyoke where burning occurred. The MDPH recommends that sampling be conducted in areas where further study suggests that contaminants in ambient air might have been deposited. The MDPH is available to review and comment on any sampling protocol developed for this effort.

XIII. PUBLIC HEALTH ACTION PLAN

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

- Upon request, the MDPH will review new environmental data related to Barnes Aquifer TCE contamination as it becomes available and will work with other environmental agencies to identify and fill in data gaps.

- If requested, the MDPH will review and comment on any proposed plan for assessing the presence of PCB combustion products in surface soil at residential properties neighboring the Dupuis Road residence where PCB wastes were reportedly burned in the 1950s. If new soil sampling data are generated, the MDPH will further characterize opportunities for exposure upon request.

- The MDPH/BEH will continue to monitor the incidence of all cancer types in the communities of Easthampton, Holyoke, Southampton, and Westfield through city/town cancer incidence reports published by the Massachusetts Cancer Registry.

The ATSDR and MDPH will evaluate and expand the Public Health Action Plan when needed. New environmental, toxicological, or health outcome data may determine the need for additional actions related to the Barnes Aquifer.

XIV. REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1997. Toxicological profile for trichloroethylene. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2001a. Evaluating exposure for possible health effects. Cited 2006 Jan 5. Available at: URL: http://www.atsdr.cdc.gov/training/public-health-assessment-overview/html/module3/sv12.html.

Agency for Toxic Substances and Disease Registry (ATSDR). 2001b. Toxicological profile for arsenic. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005a. Drinking water comparison values. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005b. Public health assessment guidance manual. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005c. Soil comparison values. Atlanta: U.S. Department of Health and Human Services.

American Cancer Society. 1999. Hodgkin's Disease. Cited 2004 July 13. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2000a. Bladder cancer. Cited 2004 Mar 10. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2000b. Pancreatic Cancer. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2001a. Kidney cancer (adult): renal cell carcinoma. Cited 2004 Mar 26. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2001b. Liver cancer. Cited 2004 Mar 23. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2003a. Cancer facts and figures 2003a. Atlanta: American Cancer Society, Inc.

American Cancer Society (ACS). 2003b. Non-Hodgkin's lymphoma. Cited 2004 Mar 19. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2005a. Cancer Facts & Figures 2005. Atlanta: American Cancer Society, Inc.

American Cancer Society (ACS). 2005b. Leukemia—Acute Myeloid. Cited 2005 Dec 14. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2005c. Leukemia—Chronic Lymphocytic. Cited 2005 Dec 15. Available at: URL: http://www3.cancer.org/cancerinfo/.

American Cancer Society (ACS). 2005d. Leukemia—Chronic Myeloid. Cited 2005 Dec 15. Available at: URL: http://www3.cancer.org/cancerinfo/.

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

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

Berg JW. 1996. Morphologic classification of human cancer. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press.

Bruning T, Bolt HM. 2000. Renal toxicity and carcinogenicity of trichloroethylene: key results, mechanisms, and controversies. Crit Rev in Tox 30(3):253-285.

Bull RJ. 2000. Mode of action of liver tumor induction by trichloroethylene and its metabolites, trichloroacetate and dichloroacetate. Enviro Health Perspect 108:241-258.

Clewell HJ, Andersen ME. 2004. Applying mode-of-action and pharmacokinetic considerations in contemporary cancer risk assessments: an example with trichloroethylene. Crit Rev in Toxico 34(5):385-445.

Cohn, P, Klotz J, Bove F, Berkowitz M, Fagliano J. 1994. Drinking water contamination and the incidence of leukemia and non-Hodgkin's lymphoma. Environ Health Perspect 102:556-561.

Costas, K, Knorr RS, Condon SK. 2002. A case-control study of childhood leukemia in Woburn, Massachusetts: the relationship between leukemia incidence and exposure to public drinking water. Sci Total Environ 300:23-35.

Easthampton Water Works (EWW). 2001. Easthampton Water Works 2000 water quality report. Easthampton, Massachusetts.

Easthampton Water Works (EWW). 2002. Data sheets concerning TCE testing of municipal water. Easthampton, Massachusetts.

Environmental Systems Research Institute (ESRI). 2005. ArcGIS, Arcview license, ver. 9.1, Redlands, California.

Fagliano J, Berry M, Bove F, Burke T. 1990. Drinking water contamination and the incidence of leukemia: an ecologic study. Am J Public Health 80:1209-1212.

Green T. Trichloroethylene and human cancer. 2001. Human Ecol Risk Assess. 7(4):677-85.

Holyoke Water Works (HWW). 2000. Annual water system report. Holyoke, Massachusetts.

Holyoke Water Works (HWW). 2002. Data sheets concerning tests of Pequot Well and Coronet Homes Well. Holyoke, Massachusetts.

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

Johansson SL, Cohen SM. 1997. Epidemiology and etiology of bladder cancer. Semin Surg Oncol 13:291-8.

Lash LH, Fisher JW, Lipscomb JC, and Parker JC. 2000. Metabolism of trichloroethylene. Environ Health Perspect 108(Suppl 2): 177-200.

Last JM. A Dictionary of Epidemiology. 1995. International Epidemiological Association, Inc. Oxford University Press: New York.

Levy BS, Wegman DH. (eds). 1995. *Occupational Health: Recognizing and Preventing Work-Related Disease*. Third Edition. Boston, New York, Toronto, London: Little, Brown and Company.

Linehan WM, Shipley WU, Parkinson DR. Cancer of the kidney and ureter. 1997. In: Devita V, Hellman S, Rosenberg S, editors. Cancer: principles and practice of oncology. 5th ed. Philadelphia: Lippincott-Raven Publishers. p. 1271-97.

Linet MS, Cartwright RA. 1996. The leukemias. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press.

London WT, McGlynn KA. 1996. Liver cancer. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press.

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

Massachusetts Department of Environmental Protection (MassDEP). 1988. Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). October 1, 2000. Western Regional Office. Memo to file from Joanne Flescher re: Hendrick Street Site Discovery Project.

Massachusetts Department of Environmental Protection (MassDEP). 2002. Site Discovery Program. Private well data, 1997-2000, from Hendrick St. Wellfield/Barnes Aquifer TCE investigation. Springfield, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). 2003. Source Water Assessment Program. Source water assessment and protection report for Easthampton Water Department. Springfield, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). 2004a. Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). 2004b. Bureau of Waste Site Cleanup, Site Discovery Program. Summary of DEP's Site Discovery Program investigation of the Barnes Aquifer/Hendrick Street water supply TCE contamination. Springfield, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). 2004c. Office of Research and Standards. Standards and guidelines for contaminants in Massachusetts drinking waters. Boston, Massachusetts.

Massachusetts Department of Environmental Protection (MassDEP). 2005a. Bureau of Waste Site Cleanup. Downloadable Site Lists. Available at: http://www.state.ma.us/dep/bwsc/sites/sdown.htm.

Massachusetts Department of Environmental Protection (MassDEP). 2005b. Bureau of Waste Site Cleanup, Site Discovery Program. Data sheets concerning private well data, 2001-2005, from Hendrick St. Wellfield/Barnes Aquifer TCE investigation. Springfield, Massachusetts.

Massachusetts Department of Environmental Quality Engineering (MDEQE). June 1980. Division of Water Supply. A program for the identification of volatile halogenated organics in the groundwaters of Massachusetts. Boston, Massachusetts.

Massachusetts Geographic Information System (MassGIS). 2004. 1:5,000 Color ortho imagery. Boston, Massachusetts.

McLaughlin JK, Blot WJ, Devesa SS, Fraumeni JF, Jr. 1996. Renal cancer. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press. p. 1142-52.

Morgan JW, Cassady RE. 2002. Community cancer assessment in response to long-time exposure to perchlorate and trichloroethylene in drinking water. J Occup Environ Med 44(7):616-21.

Mueller NE. 1996. Hodgkin's Disease. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

Office of the Secretary of the Commonwealth. 2005. Registry of Deeds. Available at: http://www.sec.state.ma.us/rod/rodidx.htm.

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

Pine and Swallow, Inc. 2000. Executive summary on the investigation of the Barnes Aquifer/Hendrick Street water supply TCE contamination. Groton, Massachusetts.

Porta M, Malats N, Jariod M, et al. 1999. Serum concentrations of organochlorine compounds and K-ras mutations in exocrine pancreatic cancer. Lancet 354:2125-29.

Rothman K and Boice J. 1982. Epidemiological Analysis with a Programmable Calculator. Boston: Epidemiology Resources, Inc.

Silverman D, Morrison A, Devesa S. 1996. Bladder cancer. In: Schottenfeld D, Fraumeni, JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press.

Southampton Water Department (SWD). 2000. 1999 Drinking water quality report. Southampton, Massachusetts.

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

U.S. Department of Commerce (U.S. DOC). 1980. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: U.S. Government Printing Office.

U.S. Department of Commerce (U.S. DOC). 1990. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: U.S. Government Printing Office.

U.S. Department of Commerce (U.S. DOC). 2000. Census of Population: General Population Characteristics, Massachusetts. U.S. Department of Commerce, Washington, DC: U.S. Government Printing Office.

U.S. Environmental Protection Agency (U.S. EPA). 2000. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment Bulletins. EPA Region 4, originally published November 1995, Website version last updated May 2000: http://www.epa.gov/region4/waste/ots/healtbul.htm

U.S. Environmental Protection Agency (U.S. EPA). 2001. Trichloroethylene Health Risk Assessment: Synthesis and Characterization. Washington, DC: U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency (U.S. EPA). 2004. Human health risk assessment. Cited 2004 Jan 29. Available at: URL: http://www.epa.gov/reg3hwmd/risk/human/index.htm.

Vartiainen T, Pukkala E, Strandman T, Kaksonen K. 1993. Population exposure to tri-and tetrachloroethylene and cancer risk: two cases of drinking water pollution. Chemosphere 27:1171-1181.

Wartenberg D, Reyner D, Siegel Scott C. 2000. Trichloroethylene and cancer: Epidemiologic evidence. Environ Health Perspect 108(Suppl 2):161-176.

Weiss LM. 2000. Epstein-Barr virus and Hodgkin's disease. Curr Oncol Rep 2(2):199-204.

Wernke MJ, Schell JD. 2004. Solvents and malignancy. Clin Occup Environ Med 4:513-527.

Westfield Water Department (WWD). 1999. Water quality report. Westfield, Massachusetts.

Wong O. 2004. Carcinogenicity of trichloroethylene: an epidemiologic assessment. Clin Occup Environ Med 4:557-589.

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

Yu MC, Yuan JM, Govindarajan S, Ross RK. 2000. Epidemiology of hepatocellular carcinoma. Can J Gastroenterol 14(8):703-9.

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

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

PREPARER

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

CERTIFICATION

The Public Health Assessment, *Evaluation of Environmental Concerns Related to the Barnes Aquifer and Cancer Incidence, 1982–2000, in Easthampton and Southampton, Hampshire County, and Holyoke and Westfield, Hampden 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. Editorial review was completed by the cooperative agreement partner.

Alan S. Crawford, REHS/RS
Technical Project Officer, CAPEB, DHAC
Agency for Toxic Substances & Disease Registry

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

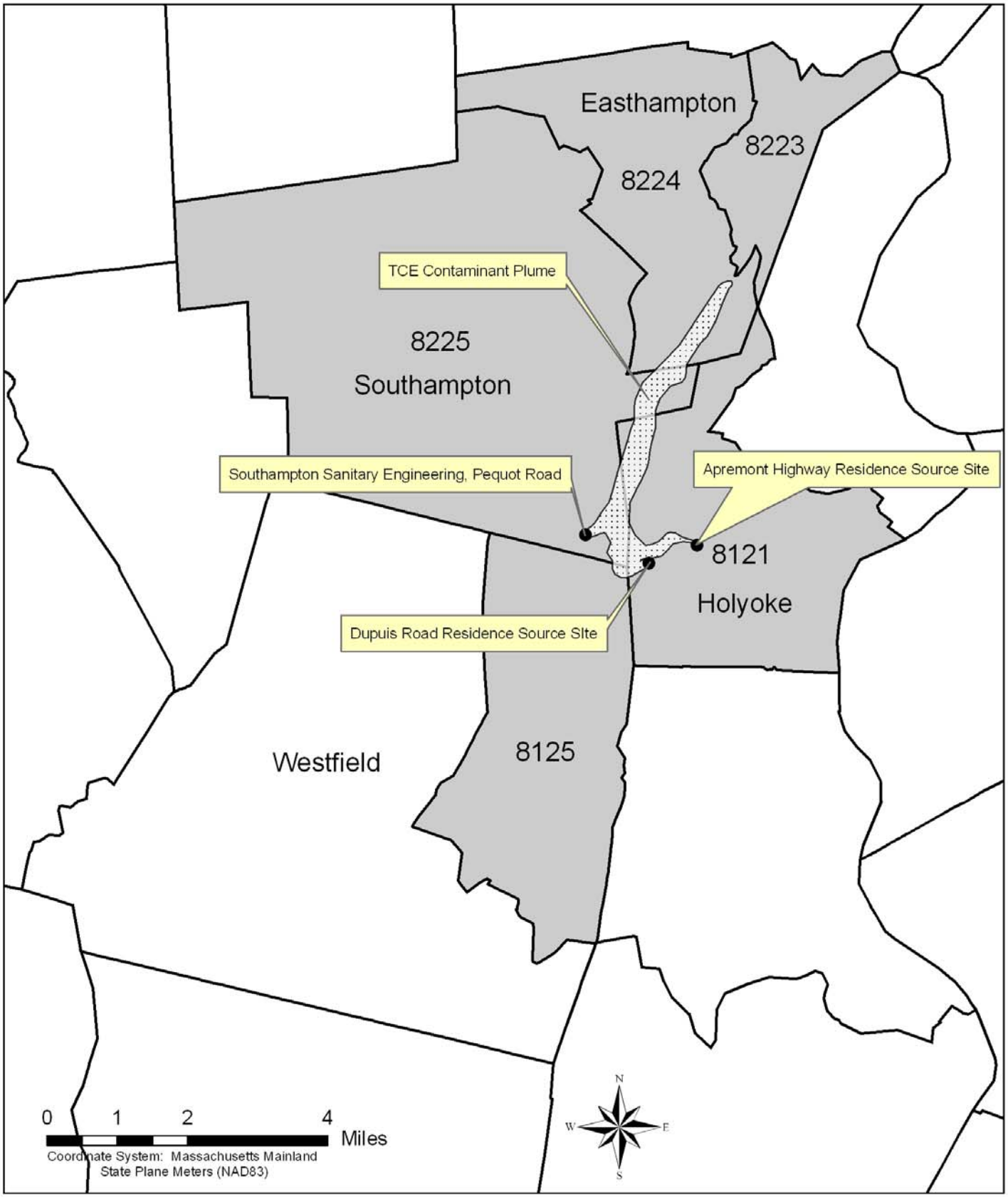
Alan W. Yarbrough, M.S.
Team Lead, CAPEB, DHAC
Agency for Toxic Substances & Disease Registry

FIGURES

Figure 1
Communities Encompassed by Evaluation
Easthampton, Holyoke, Southampton, and Westfield, Massachusetts

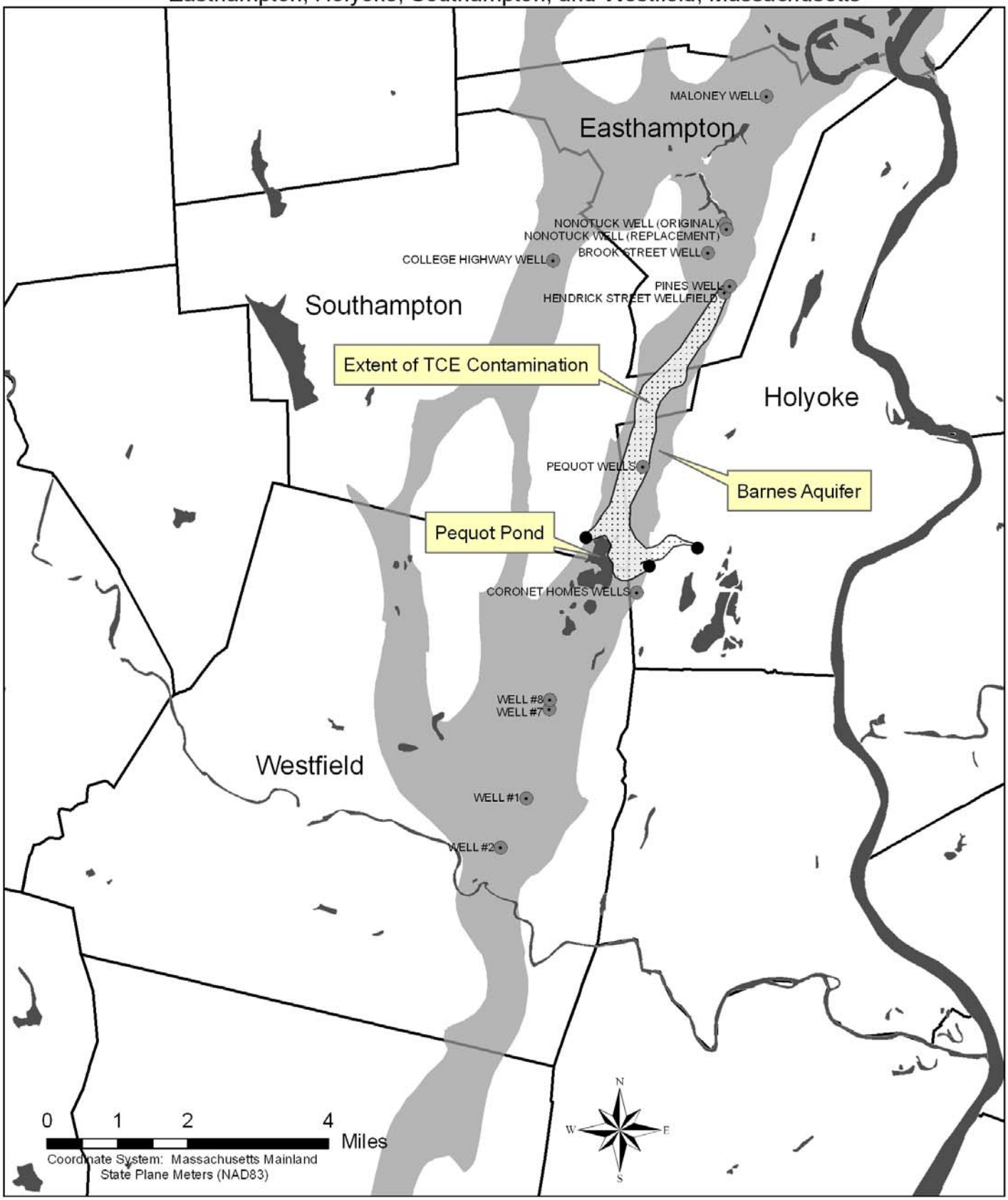
Geographic data supplied by:
Massachusetts Executive Office of Environmental Affairs, MassGIS;
Geographic Data Technology, Inc.; U.S. Bureau of the Census.

Figure 2
Census Tracts Encompassed by Evaluation
Easthampton, Holyoke, Southampton, and Westfield, Massachusetts



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

Figure 3
Public Wells in the Barnes Aquifer
Easthampton, Holyoke, Southampton, and Westfield, Massachusetts

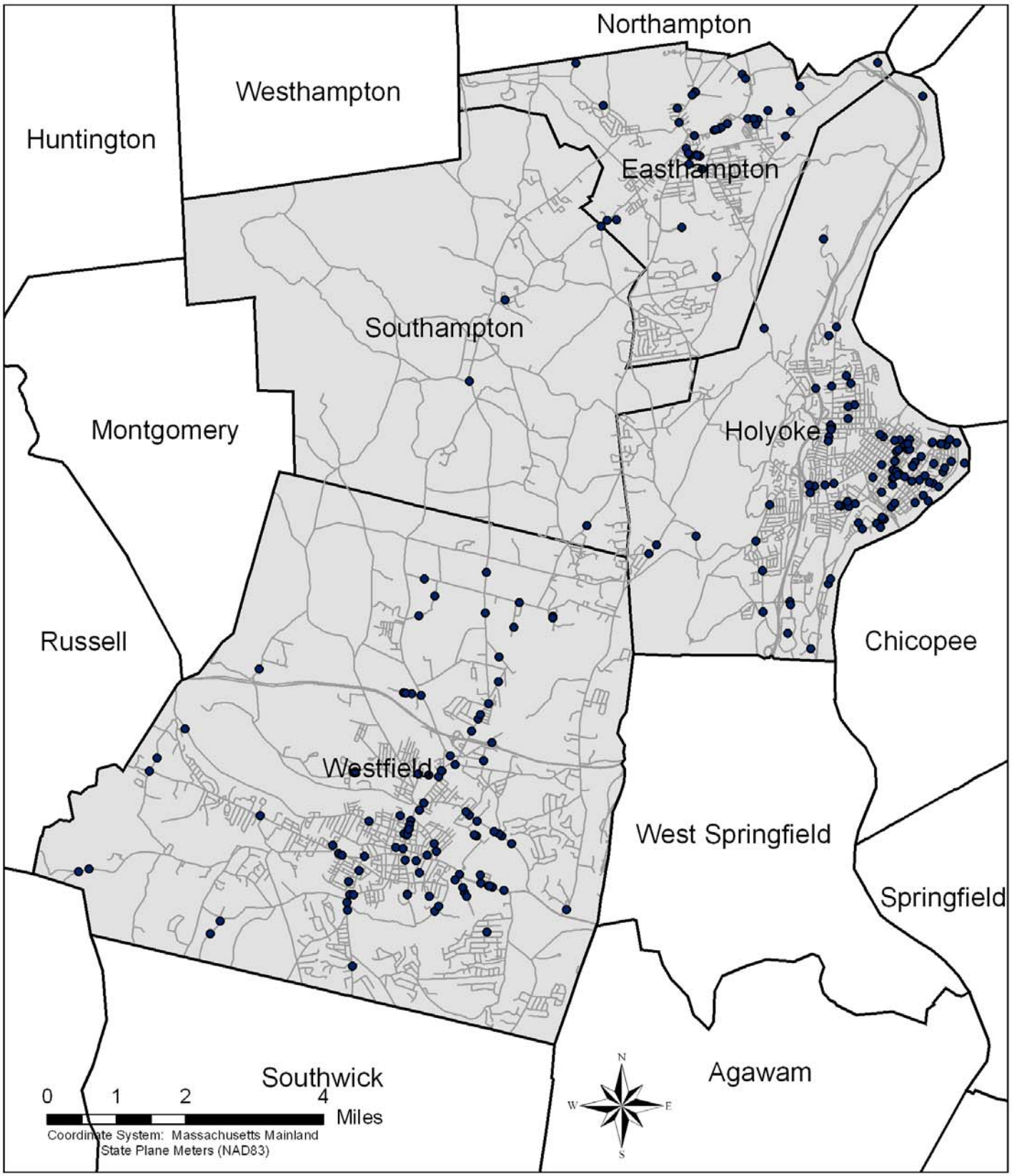


Bureau of BEH Environmental Health

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

Public Well
Aquifer Deposits
Surface Water
Source sites

Figure 4
Location of Massachusetts Department of Environmental Protection 21E Hazardous Waste and Oil Releases
Easthampton, Holyoke, Southampton and Westfield, Massachusetts



Bureau of BEH Environmental Health

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

MassDEP 21E Site
Roads

TABLES

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

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0000064	GASSTATION	EASTHAMPTON EXXON	32 UNION ST	EH	1/12/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNFA
Mapped	1-0000065	MUNICIPAL	EASTHAMPTON LANDFILL TOWN OF	OLIVER ST	EH	1/15/1987	WASTE OIL	LANDFILL, UNCONTAIN	ADQREG
Not Mapped	1-0000066		EASTHAMPTON LANDFILL	LOUDVILLE RD	EH	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		ADQREG
Mapped	1-0000067		FERRY STREET SITE	FERRY ST	EH	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000068	COMMERCIAL	MAGNAT CO	ONEIL ST	EH	10/24/1985	UNKNOWN CHEMICAL OF UNKNOWN TYPE	SEPTIC TANK	WCSPRM
Mapped	1-0000296	INDUSTRIAL, MANUFACT	NATIONAL FELT CO	MECHANIC ST	EH	8/14/1987	OIL	UST	RAO
Mapped	1-0000415		STANLEY HOME PRODUCTS	116 PLEASANT ST	EH	7/15/1989	PETROLEUM BASED OIL		RAO
Mapped	1-0000504	INDUSTRIAL	STONINGTON CORP.	45 FERRY ST	EH	10/15/1988	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0000512		KELLOGG BRUSH ARTHUR	ARTHUR ST	EH	8/1/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		PENNFA
Mapped	1-0000513	INDUSTRIAL	KELLOG BRUSH MANUFACTURING	122 PLEASANT ST	EH	10/15/1988	UNKNOWN CHEMICAL OF TYPE - OIL		RAO
Mapped	1-0000514		PLEASANT STREET PROPERTY	13-15 PLEASANT ST	EH	10/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		PENNFA
Mapped	1-0000608	GASSTATION, WETLANDS	INLAND CITGO STATION	101-109 PLEASANT ST	EH	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	REMOPS
Not Mapped	1-0000639	MUNICIPAL	PROPERTY OFF HENDRICKS ST	OFF HENDRICKS ST	EH	7/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		TIER1A
Mapped	1-0000674	COGASPLANT	EASTHAMPTON AREA WORK CENTER	19 LIBERTY ST	EH	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		TIER1C
Mapped	1-0000689	MANUFACT	CATALYTIC PAINT CO	ARTHUR ST	EH	1/10/1991	UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	RAO
Mapped	1-0000723		WWTP SLUDGE LAGOON	FERRY ST	EH	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		LSPNFA
Mapped	1-0000728	INDUSTRIAL	FMR J P STEVENS	27 PAYSON AVE	EH	1/15/1990	WASTE OIL	LAGOON	RAO
Mapped	1-0000776	GASSTATION	COTTAGE STREET MOTORS	47 COTTAGE ST	EH	3/12/1990	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0001020	FORMER, GASSTATION	HAMPSHIRE CHRYSLER PLYMOUTH	150 NORTHAMPTON ST	EH	7/15/1993	WASTE OIL	UST	RAO
Mapped	1-0001071	INDUSTRIAL	EASTHAMPTON TIRE OUTLET	141 NORTHAMPTON ST	EH	9/27/1993	WASTE OIL	UST	INVSUB
Mapped	1-0010035	MUNICIPAL	SEWER PUMP STATION	NONATUCK PARK	EH	10/25/1993	DIESEL FUEL, FUEL OIL #2	UST	RAO
Mapped	1-0010201	INDUSTRIAL	FREIGHT ELEVATOR INSIDE STANHOME INC	116 PLEASANT ST	EH	2/14/1994	PETROLEUM BASED OIL (150 GAL), PETROLEUM BASED OIL (53 GAL)	PIPE	RAONR
Mapped	1-0010339	INDUSTRIAL	KELLOGG BRUSH INJECTION MOLDING DEPT FL	122 PLEASANT ST	EH	5/16/1994	PETROLEUM BASED OIL (60 GAL), PETROLEUM BASED OIL (60 GAL)	PIPE	RAO
Mapped	1-0010464	COMMERCIAL	CITGO STATION	105 PLEASANT ST	EH	8/8/1994	DIESEL FUEL, GASOLINE	UST	REMOPS
Mapped	1-0010486	COMMERCIAL	NEAR WEST ST	54 NORTHAMPTON ST	EH	8/25/1994	FUEL OIL #2, FUEL OIL #2 (75 PPMV)	UST	RAO
Mapped	1-0010723	COMMERCIAL	GASOLINE STATION	124 NORTHAMPTON ST	EH	2/17/1995	GASOLINE, UNKNOWN CHEMICAL OF TYPE - OIL	UST	RAO
Mapped	1-0010866	COMMERCIAL	LAZY D FARMS	283 EAST ST	EH	5/12/1995	GASOLINE, PETROLEUM BASED OIL (600 PPMV)	UST	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0010892		7/11 STATION	97 UNION ST	EH	6/2/1995	GASOLINE (100 PPM), TOTAL PETROLEUM HYDROCARBONS (TPH) (100 PPMV)	UST	RAONR
Mapped	1-0011353	COMMERCIAL	ABANDONED FOOD & FUEL GAS STATION	72 UNION ST	EH	5/2/1996	GASOLINE	UST	RAO
Not Mapped	1-0011609	ROADWAY	TTU ACCIDENT	PARK ST	EH	11/26/1996	DIESEL FUEL (40 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE	VEHICLE	RAO
Mapped	1-0011629	INDUSTRIAL	JPS ELASTOMERICS CORP	412 MAIN ST	EH	12/10/1996	UNKNOWN CHEMICAL OF UNKNOWN TYPE (115 LBS)		RAO
Mapped	1-0011636	RESIDNTIAL	NO LOCATION AID	17 BROOK ST	EH	12/12/1996	FUEL OIL #2 (202 PPMV)	UST	RAO
Mapped	1-0011887	RESIDNTIAL	NO LOCATION AID	FORT HILL RD	EH	6/21/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (385 GAL)	DRUMS	RAO
Mapped	1-0012049	COMMERCIAL	EASTHAMPTON BP	124 NORTHAMPTON ST	EH	10/15/1997	GASOLINE (100 PPM)	UST	RAO
Mapped	1-0012059	INDUSTRIAL	GARAGE BEHIND NATIONAL NONWOVEN	150 PLEASANT ST	EH	10/21/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL)	DRUMS	RAO
Mapped	1-0012176		STRONG CORPORATION	40 ONEIL ST	EH	1/26/1998	FUEL OIL #2		RAO
Mapped	1-0012406	COMMERCIAL	PRIDE STATION	60 UNION ST	EH	6/18/1998	GASOLINE (25 GAL)	UNKNOWN	RAO
Mapped	1-0012489	RESIDNTIAL	CIALEK RESIDENCE	17 GROVELAND ST	EH	7/30/1998	FUEL OIL #2 (169 PPM)	UST	RAO
Mapped	1-0012499	MUNICIPAL	TOWN LODGING	75 OLIVER ST	EH	8/4/1998	FUEL OIL #2 (100 PPM)	UST	RAO
Mapped	1-0012881	COMMERCIAL	OCONNELL OIL SERVICE STATION	19 PARSONS ST	EH	4/6/1999	GASOLINE (5700 PPB)	UST	TIER1C
Mapped	1-0013108	COMMERCIAL	AJ KIENLE COAL CO	20 MECHANIC ST	EH	9/15/1999	FUEL OIL #2 (150 PPMV)	UST	RAO
Mapped	1-0000088		ATLAS COPCO HOLYOKE INC	161 LOWER WESTFIELD RD	HO	7/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNDS
Mapped	1-0000089		ATLAS COPCO HOLYOKE INC	37 APPLETON ST	HO	7/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNDS
Mapped	1-0000090	GASSTATION	BURGESS GULF	582 SOUTH ST	HO	7/24/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0013515	INDUSTRIAL	FRM ZONOLITE PLANT	WEMELCO WAY	HO	6/30/2000	ASBESTOS	HISTORICAL	TIERII
Mapped	1-0013516	INDUSTRIAL	OCTOBER COMPANY INC	51 FERRY ST	HO	6/30/2000	DIESEL FUEL (55 GAL)	TANKER	RAO
Mapped	1-0013709	STATE	OXBOW STATE BOAT RAMP	ROUTE 5	HO	12/4/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (26 GAL)	DRUMS	RAO
Mapped	1-0000091		DOWNING & DOWNING	109 WINTER ST	HO	4/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNDS
Mapped	1-0000092	MANUFACT	HAMPDEN PAPERS	100 WATER ST	HO	4/8/1986	BENZENE, METHYL-	UST	DEPNFA
Mapped	1-0000093	INDUSTRIAL	HOLYOKE GAS & ELETRIC	CABOT ST	HO	10/28/1986	PETROLEUM BASED OIL	UNKNOWN	RAO
Mapped	1-0000094		FMR	RD	HO	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNDS
Mapped	1-0000095	GASSTATION	INTERSTATE CHEVRON	181 FRANKLIN ST	HO	4/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000096	MANUFACT	JAMES RIVER GRAPHICS	BERKSHIRE ST	HO	10/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNCONTAIN	RAO
Mapped	1-0000097	COMMERCIAL	MARCOTTE FORD SALES	1025 MAIN ST	HO	7/9/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	PENNFA
Mapped	1-0000098	COMMERCIAL, INDUSTRIAL, MANUFA	BASF POLYMER SYSTEMS FMR MOBIL	3 HANOVER ST	HO	1/15/1987	PETROLEUM BASED OIL	UST	REMOPS
Mapped	1-0000099		REMINGTON FORMER	686 MAIN ST	HO	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000100	GASSTATION	REPUBLIC OIL	330 MAIN ST	HO	4/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	REMOPS
Mapped	1-0000101	GASSTATION	REPUBLIC OIL	CANAL ST	HO	4/15/1987	UNKNOWN CHEMICAL OF TYPE - OIL	UST	DEPNFA
Mapped	1-0000102		SCHWARTZ	118 CABOT ST	HO	4/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	TIERII

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0000103	AUTOREPAIR	SHELDON TRANSFER & STORAGE	55 NORTH CANAL ST	HO	9/17/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	TIER1D
Mapped	1-0000271	COMMERCIAL	PARSONS PAPER	84 SARGEANT ST	HO	7/15/1987	UNKNOWN CHEMICAL OF TYPE - OIL	UST	RAO
Mapped	1-0000359	HOSPITAL, MUNICIPAL	HOLYOKE HOSPITAL	575 BEECH ST	HO	9/4/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000435	INDUSTRIAL	FORMER LUDLOW CORP	111 MOSHER ST	HO	10/15/1988	PETROLEUM BASED OIL	UST	PENNFA
Mapped	1-0000474	MUNICIPAL	HOLYOKE DPW	24 COMMERCIAL ST	HO	5/11/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000507		CHESTNUT STREET PROPERTY	167 CHESTNUT ST	HO	10/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		PENNFA
Mapped	1-0000525		SLABYS SUNOCO	220 SUFFOLK ST	HO	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000576		NORTHAMPTON STREET PROPERTY	1607 NORTHAMPTON ST	HO	1/24/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000614		EARLY AUTO CARE INC	636 MAIN ST	HO	7/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000656	INDUSTRIAL	XIDEX CORP FACILITY	195 APPLETON ST	HO	10/15/1989	UNKNOWN CHEMICAL OF TYPE - OIL	UST	RAO
Mapped	1-0000670	RESIDNTIAL	LINCOLN ST PROPERTY	85 LINCOLN ST	HO	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNFA
Mapped	1-0000676	INDUSTRIAL	CITY PAINT FACTORY	1548 NORTHAMPTON ST	HO	1/15/1990	UNKNOWN CHEMICAL OF TYPE - OIL		RAO
Mapped	1-0000700	GASSTATION	CRABTREES MOBIL 01 JFN	1530 NORTHAMPTON ST	HO	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000701	OPENSPACE	COMMERCIAL & JACKSON	COMMERCIAL ST JACKSON ST	HO	1/15/1990	PETROLEUM BASED OIL, UNKNOWN CHEMICAL OF TYPE - OIL	UST	STMRET
Mapped	1-0000704	INDUSTRIAL	WINTER ST PARCEL	20 WINTER ST	HO	11/22/1989	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0000711		HOLYOKE WATER POWER	BERKSHIRE ST	HO	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		LSPNFA
Mapped	1-0000790	DRYCLEANER, FORMER	DOMINOS PIZZA	399-401 HILLSIDE AVE	HO	10/15/1990	UNKNOWN CHEMICAL OF TYPE - OIL		TIER1D
Mapped	1-0000802	GASSTATION	GETTY PETROLEUM STATION	630 DWIGHT ST	HO	10/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000816	COGASPLANT, FORMER, INDUSTRIAL	HOLYOKE GAS WORKS FMR	7 NORTH BRIDGE ST	HO	10/15/1990	UNKNOWN CHEMICAL OF TYPE - OIL	UST	TIER1A
Mapped	1-0000907	INDUSTRIAL	MT TOM GENERATOR PLANT	ROUTE 5	HO	7/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE, UST	REMOPS
Mapped	1-0000966	HOSPITAL	HOLYOKE SOLDIERS HOME	110 CHERRY ST	HO	4/15/1992	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0001024	COMMERCIAL, GASSTATION	YEORG'S GARAGE & TIRE SERVICE	158 CHESTNUT ST	HO	7/15/1993	PETROLEUM BASED OIL	UST	TIERII
Mapped	1-0001046	GASSTATION	GALLAGHERS OLD FASHION SER	532 HIGH ST	HO	10/1/1993	UNKNOWN CHEMICAL OF TYPE - OIL	UST	DPS
Mapped	1-0001051		REARDONS GARAGE	1537 NORTHAMPTON ST	HO	7/15/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNDS
Mapped	1-0001053	INDUSTRIAL	AVERY DENNISON	1 CABOT ST	HO	8/3/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNCONTAIN, UNKNOWN	WCSPRM
Mapped	1-0001055	INDUSTRIAL	HOLYOKE GAS TAR DEPOSITS	BELOW HOLYOKE DAM COURT RIV	HO	7/15/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE	TIER1A
Mapped	1-0001056	UTILITY	NEW ENGLAND TELEPHONE CO	322 MAPLE ST	HO	7/9/1993	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0001069	SKI AREA	MOUNT TOM SKI AREA	ROUTE 5	HO	9/28/1993	PETROLEUM BASED OIL	PIPE	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0010023	COMMERCIAL	OLD STEIGERS BUILDING	253-267 HIGH ST	HO	10/19/1993	FUEL OIL #2, FUEL OIL #4	UST	RAO
Mapped	1-0010047	INDUSTRIAL	ADAMS PAKKAWOOD	191 APPLETON ST	HO	10/30/1993	UNKNOWN CHEMICAL OF TYPE - OIL (100 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (450 PPM)	TRANSFORM	RAO
Not Mapped	1-0010165	ROADWAY	NO LOCATION AID	SOUTHBOUND I91	HO	1/12/1994	DIESEL FUEL (40 GAL), KEROSENE	FUELTANK	RAO
Mapped	1-0010168	INDUSTRIAL, WATERBODY	HOLYOKE WATER POWER HADLEY FALLS STATION	GATEHOUSE RD	HO	1/14/1994	FUEL OIL #2 (1800 GAL), FUEL OIL #2 (400 GAL)	PIPE, UST	REMOPS
Mapped	1-0010176	STATE	SOLDIERS HOME	110 CHERRY ST	HO	1/20/1994	FUEL OIL #6 (15 GAL)	TANKER	RAO
Mapped	1-0010212	RESIDNTIAL	SPEARS RESIDENCE	29 MEADOW ST	HO	2/22/1994	FUEL OIL #2 (100 GAL), PETROLEUM BASED OIL (30 GAL)	PIPE, UST	RAO
Mapped	1-0010218	ROADWAY	NORTH OF EXIT 16 MM 15.3	RT 91 SOUTHBOUND EXIT 17	HO	2/23/1994	DIESEL FUEL	FUELTANK, VEHICLE	RAO
Not Mapped	1-0010256	ROADWAY	MM17 N	RT 91N MM17	HO	3/20/1994	DIESEL FUEL, DIESEL FUEL (100 GAL)	FUELTANK, TRACT TANK	RAO
Not Mapped	1-0010267	COMMERCIAL	FMR DREIKORNS BLDG	322 PARK ST	HO	3/24/1994	FUEL OIL #2, FUEL OIL #2 (4 INCH)	UST	RAO
Mapped	1-0010271	ROADWAY	NO LOCATION AID	COR OF MAPLE ST AND ESSEX ST	HO	3/29/1994	FUEL OIL #2 (10 GAL), FUEL OIL #2 (50 GAL)	PIPE, TANKER	RAO
Mapped	1-0010318	COMMERCIAL, SERVSTATIO	B & D PETROLEUM SALES INC	3 BROWN AVE	HO	5/2/1994	GASOLINE (300 PPMV), GASOLINE (320 PPM)	OVERFILL, POSSIBLE, UST	RAO
Mapped	1-0010435	RESIDNTIAL	LAFLAMME RESIDENCE	AVE	HO	7/18/1994	FUEL OIL #2	UST	RAO
Mapped	1-0010457	COMMERCIAL	FMR MAIN AUTO	600 MAIN ST	HO	8/1/1994	WASTE OIL	DRUMS	RAO
Mapped	1-0010473	COMMERCIAL, WATERBODY	HADLEY DAM	GATEHOUSE RD	HO	8/15/1994	FUEL OIL #2, FUEL OIL #2 (20 GAL)	UST	RAONR
Mapped	1-0010477	COMMERCIAL	HAZEN PAPER	THIRD LEVEL CANAL	HO	8/19/1994	2-BUTANONE, 2-BUTANONE (1 GAL), BENZENE, METHYL- (1 GAL)	PUMP LEAK	RAO
Mapped	1-0010532	INDUSTRIAL	MT TOM POWER PLANT	RT 5 SMITHS FRY	HO	9/26/1994	FUEL OIL #6 (50 GAL)	PIPE	RAO
Mapped	1-0010533	HOSPITAL	HOLYOKE HOSPITAL	575 BEECH ST	HO	9/26/1994	FUEL OIL #2	UST	RAO
Mapped	1-0010649	INDUSTRIAL	MASTEX INDUSTRIES	CABOT AND BIGELOW ST	HO	12/7/1994	1-METHYL-2-CHLOROBENZENE (27 LBS), 1-METHYL-2-CHLOROBENZENE (40 LBS)	RELEASE	RAO
Mapped	1-0010718	COMMERCIAL	OLD TAXI GARAGE	118 CABOT ST	HO	2/1/1995	FUEL OIL #2 (10 GAL), WASTE OIL	AST, UNKNOWN	RAONR
Mapped	1-0010765	COMMERCIAL	JOHNS SUNOCO	1616 NORTHAMPTON ST	HO	3/13/1995	GASOLINE (100 PPMV)	UST	RAO
Mapped	1-0010782	COMMERCIAL	CORNER MAPLE ST	145 HAMPDEN ST	HO	3/23/1995	GASOLINE, GASOLINE (100 PPMV)	UST	RAO
Mapped	1-0010849	INDUSTRIAL	HOLYOKE B & M RAILROAD	CRESCENT ST	HO	5/2/1995	DIESEL FUEL (150 GAL)	FUELTANK	RAO
Mapped	1-0010878	RESIDNTIAL	RESIDENCE	48 LIBERTY ST	HO	5/25/1995	FUEL OIL #2 (4000 MG/KG)	UST	RAO
Mapped	1-0010902	RESIDNTIAL	LACASSE APARTMENTS	SOUTH CANAL ST	HO	6/9/1995	FUEL OIL #2, FUEL OIL #2 (347 PPM)	UST	RAO
Not Mapped	1-0010915		MM 17	RTE 91 N	HO	6/19/1995	DIESEL FUEL (10 GAL), DIESEL FUEL (60 GAL)	FUELTANK	RAO
Mapped	1-0010916	COMMERCIAL, FUNERALHOM	HOLBERT FUNERAL HOME	250 SUFFOLK ST	HO	6/19/1995	FUEL OIL #2 (100 PPM), FUEL OIL #2 (150 PPM), PETROLEUM BASED OIL	UST	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0010919	COMMERCIAL	YEORGS GARAGE	158 CHESTNUT ST	HO	6/20/1995	UNKNOWN CHEMICAL OF TYPE - OIL, UNKNOWN CHEMICAL OF UNKNOWN TYPE (10.8 INCH)	UNKNOWN	RAONR
Mapped	1-0010937	INDUSTRIAL	HEATING OIL STORAGE DIKE	3 HANOVER ST	HO	7/4/1995	OIL (300 GAL)	AST	RAO
Mapped	1-0010938	INDUSTRIAL	LOADING DOCK	20 WATER ST	HO	7/7/1995	WASTE OIL	DRUMS	RAO
Mapped	1-0010975	MUNICIPAL	HOLYOKE DPW	BEHIND CITY HALL	HO	7/27/1995	GASOLINE (2300 PPM), GASOLINE (2500 PPM)	UST	RAO
Mapped	1-0010996	MUNICIPAL	CITY OF HOLYOKE DPW	63 NORTH CANAL ST	HO	8/11/1995	DIESEL FUEL, FUEL OIL #2 (110 PPM)	UST	RAO
Mapped	1-0010998	MUNICIPAL	FIRE DEPT REPAIR YARD	1034 HAPDEN ST	HO	8/14/1995	GASOLINE	UST	RAO
Mapped	1-0011057	RESIDNTIAL	SERGEANT WEST APTS	151 WEST ST	HO	9/22/1995	FUEL OIL #2	UST	RAO
Mapped	1-0011080	RESIDNTIAL	NO LOCATION AID	8 WILLIAMS ST	HO	10/6/1995	FUEL OIL #2 (9100 PPM)	UST	TIER1D
Mapped	1-0011086	SCHOOL, STATE	HCC	303 HOMESTEAD AVE	HO	10/11/1995	GASOLINE (180 PPMV)	UST	RAO
Mapped	1-0011102	COMMERCIAL, INDUSTRIAL, WATERB	ON CANAL	383 DWIGHT ST	HO	10/20/1995	FUEL OIL #6	PIPE	RAO
Mapped	1-0011163	MUNICIPAL	DPW GARAGE	63 NORTH CANAL ST	HO	12/1/1995	PETROLEUM BASED OIL (190 PPM)	UST	RAONR
Mapped	1-0011232	RESIDNTIAL	FRANCISCAN MISSIONARY	RD	HO	2/1/1996	FUEL OIL #2	UNKNOWN	RAO
Mapped	1-0011291	RESIDNTIAL	FREDDIE MAC PROPERTY	38 COIT ST	HO	3/14/1996	FUEL OIL #2 (101 PPM)	UST	RAO
Mapped	1-0011312	MUNICIPAL	FIRE STATION #3	1579 NORTHAMPTON ST	HO	4/3/1996	GASOLINE	PIPE	RAO
Mapped	1-0011337	OPENSPACE	CENTRAL AUTO SALES	1010 MAIN ST	HO	4/22/1996		AST, DRUMS, TANKER	TIER1D
Mapped	1-0011360	INDUSTRIAL	CHEMICAL FINISHING INC	110 NORTH BRIDGE ST	HO	5/6/1996	UNKNOWN CHEMICAL OF UNKNOWN TYPE	DRUMS	RAO
Mapped	1-0011380	COMMERCIAL	LOG CABIN RESTRAURANT	EASTHAMPTON RD	HO	5/23/1996	FUEL OIL #2 (125 PPMV), FUEL OIL #2 (31 PPMV)	UST	RAO
Mapped	1-0011399	COMMERCIAL, RESIDNTIAL	SPEAR PROPERTY	693 DWIGHT ST	HO	6/6/1996	FUEL OIL #2 (165 PPM)	UST	RAONR
Mapped	1-0011517	COMMERCIAL, INDUSTRIAL	HAMPDEN PAPERS	100 WATER ST	HO	9/13/1996	DIESEL FUEL (17 GAL)	FUELTANK	RAO
Mapped	1-0011534	COMMERCIAL	OIL TANK	158 CHESTNUT ST	HO	9/26/1996	FUEL OIL #2	UST	RAONR
Mapped	1-0011549	COMMERCIAL	A.J. VIRGILIO CONSTRUCTION CO	115 CHAPIN ST	HO	10/10/1996	DIESEL FUEL, FUEL OIL #2, WASTE OIL	DRUMS	RAO
Mapped	1-0011569	COMMERCIAL, RESIDNTIAL	NO LOCATION AID	INT RTE 202 HOMESTEAD AVE	HO	10/24/1996	UNKNOWN CHEMICAL OF TYPE - OIL (30 GAL)	TRANSFORM	RAO
Mapped	1-0011840	COMMERCIAL	REAR OF FOOD MART	SOUTH ST	HO	5/21/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (30 GAL)	DRUMS	RAO
Mapped	1-0011868	MEDIAN, OPENSPACE, ROADWAY	MT TOM POWER STACK	RTE I-91 S	HO	6/12/1997	DIESEL FUEL (50 GAL)	VEHICLE	RAO
Mapped	1-0011916	MUNICIPAL, OPENSPACE	SPRINGDALE PARK	827 MAIN ST	HO	7/14/1997	WASTE OIL (50 GAL)	DRUMS	RAO
Mapped	1-0011975	RESIDNTIAL	SISTERS OF PROVIDENCE MOTHERHOUSE	5 GAMELIN ST	HO	8/22/1997	GASOLINE (155 PPMV)	UST	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0011977	COMMERCIAL	ANATEC	DWIGHT ST	HO	8/25/1997	DIESEL FUEL (25 GAL)	SADDLE TANK	RAO
Mapped	1-0012055	INDUSTRIAL	ANITEC	383 DWIGHT ST	HO	10/15/1997	SULFURIC ACID	SCRUBBER	RAO
Mapped	1-0012080	COMMERCIAL	BASF CORPORATION	HANOVER ST	HO	11/6/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (100 GAL)	AST	RAO
Mapped	1-0012266		AT CANAL STREET	CABOT ST	HO	3/30/1998	DIESEL FUEL (-70 GAL)	VEHICLE	RAO
Mapped	1-0012301	INDUSTRIAL	ATLAS COPCO COMPRESSORS INC	161 LOWER WESTFIELD RD	HO	4/22/1998	DIESEL FUEL (-20 GAL)	VEHICLE	RAO
Mapped	1-0012319	COMMERCIAL	F L ROBERTS	679 MAIN ST	HO	4/28/1998	GASOLINE (300 PPM)	UST	RAO
Mapped	1-0012484	COMMERCIAL	JC PENNY LOADING DOCK	HOLYOKE MALL	HO	7/24/1998	GASOLINE (-20 GAL)	VEHICLE	RAO
Mapped	1-0012611	RESIDNTIAL	VACANT LOT	129 TO 133 WALNUT ST	HO	9/28/1998	UNKNOWN CHEMICAL OF UNKNOWN TYPE (-3 GAL)	DRUMS	TIER1D
Mapped	1-0012759	MUNICIPAL	FIRE STATION	206 MAPLE ST	HO	12/23/1998	GASOLINE (260 PPM)	UST	RAO
Mapped	1-0012771	MUNICIPAL	FIRE STATION #6	820 HOMESTEAD AVE	HO	12/30/1998	GASOLINE (100 PPM)	UST	RAO
Mapped	1-0012772	MUNICIPAL	HOLYOKE DPW	63 CANAL ST	HO	12/30/1998	GASOLINE (100 PPM)	UST	RAO
Mapped	1-0012784	MUNICIPAL	HOLYOKE WATER WORKS	20 COMMERCIAL ST	HO	1/16/1999	GASOLINE (118 PPMV)	UST	RAO
Mapped	1-0012833	COMMERCIAL	NEW WALGREEN CONSTRUCTION SITE	1588 NORTHAMPTON ST	HO	2/22/1999	FUEL OIL #2 (110 PPM)	UST	RAO
Mapped	1-0012879	INDUSTRIAL, WATERBODY	NEXT TO HALLMARK BUILDING	526 SOUTH WATER ST	HO	4/3/1999	WASTE OIL	DRUMS, VEHICLE	TIERII
Mapped	1-0012884	RESIDNTIAL	NO LOCATION AID	263 ELM ST	HO	4/8/1999	FUEL OIL #2 (120 PPM)	UST	TIER1D
Mapped	1-0012908	MUNICIPAL	SULLIVAN SCRAP	107 APPLETON ST	HO	4/27/1999	1,1'-BIPHENYL, CHLORO-DERIVS. (500 PPM)	TRANSFORM	RAO
Mapped	1-0012915	COMMERCIAL	FILENES PARKING LOT HOLYOKE MALL	MALL RING RD	HO	5/1/1999	UNKNOWN CHEMICAL OF UNKNOWN TYPE (345 GAL)	TRANSFORM	RAO
Mapped	1-0013089	RESIDNTIAL	NO LOCATION AID	75 MOUNTAINVIEW DR	HO	9/3/1999	FUEL OIL #2 (100 PPM)	UST	RAO
Not Mapped	1-0013194	RIGHTOFWAY, STATE	MM 18	RTE 91 N	HO	11/17/1999	DIESEL FUEL (10 GAL)	VEHICLE	RAO
Mapped	1-0013243	RESIDNTIAL	ABANDONED APARTMENT BLDG	1 WORCESTER PL	HO	12/16/1999	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, WASTE OIL	DRUMS	TIER1D
Mapped	1-0013278	RESIDNTIAL	RESIDENCE	92 CHAPIN ST	HO	1/18/2000	FUEL OIL #2 (275 GAL)	AST	RAO
Mapped	1-0013507	RESIDNTIAL	NO LOCATION AID	74 MADISON AVE	HO	6/26/2000	FUEL OIL #2 (119 PPM)	UST	RAO
Not Mapped	1-0013551	COMMERCIAL, RESIDNTIAL, ROADWA	UNDERPASS BRIDGE	MAIN AND LYMAN ST	HO	7/21/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (30 GAL)	VEHICLE	RAO
Mapped	1-0013568	COMMERCIAL	UNION MART GASOLINE STATION	297 APERMONT HWY	HO	7/31/2000	BENZENE (1000 PPB), BENZENE, METHYL- (1400 PPB), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (1700 PPB)	UNKNOWN	RAO
Mapped	1-0013592	COMMERCIAL	SEALED AIR CORP	2030 LOWER HOMESTEAD AVE	HO	8/17/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (21 GAL)	TRANSFORM	RAO
Mapped	1-0013692	RESIDNTIAL	RESIDENCE	1873 NORTHAMPTON ST	HO	11/17/2000	FUEL OIL #2 (25 GAL)	AST, PIPE	RAO
Mapped	1-0013699	COMMERCIAL	HAROLDS GARAGE INC	19 HOLYOKE ST	HO	11/27/2000	DIESEL FUEL (835 GAL)	TANKER	RAO
Mapped	1-0013735	RESIDNTIAL	LUSSIER RESIDENCE	30 DUPUIS RD	HO	12/29/2000	1,1'-BIPHENYL, CHLORO-DERIVS. (23 PPM)	UNKNOWN	TIERII
Mapped	1-0013736	RESIDNTIAL	ODABASHEN RESIDENCE	94 APREMONT HWY	HO	12/29/2000	1,1'-BIPHENYL, CHLORO-DERIVS. (411 PPM)	UNKNOWN	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0000264	FORMER, REFINISHER	FORMER MR STRIPPER	6 COLEMAN RD	SH	11/17/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE	BURIED, PIPE	DEPNFA
Mapped	1-0010766	COMMERCIAL	MIDTOWN MOTORS N OF POMEROY MDW RD	151 COLLEGE HWY	SH	3/14/1995	GASOLINE (100 PPMV)	UST	RAO
Not Mapped	1-0010955	RESIDNTIAL, ROADWAY	POLE 18	PLEASANT ST	SH	7/15/1995	UNKNOWN CHEMICAL OF TYPE - OIL (18 GAL)	TRANSFORM	RAO
Mapped	1-0011109	RESIDNTIAL, ROADWAY	CORNER OF FOMER RD	COLLEGE HWY	SH	10/28/1995	UNKNOWN CHEMICAL OF TYPE - OIL (10 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (3 GAL)	TRANSFORM, VEHICLE	RAO
Mapped	1-0013737	COMMERCIAL, RESIDNTIAL	4.5 MILE LONG GW CONTAMINATION PLUME	82 PEQUOT RD	SH	12/20/2000	ETHENE, TRICHLORO- (26 PPB)	UNKNOWN	TIER1D
Mapped	1-0000227	COMMERCIAL, FORMER, JUNKYARD	CAMEROTAS AUTO SALVAGE	NECK RD PO BOX 537	WF	9/17/1986	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, UNKNOWN CHEMICAL OF TYPE - OIL, UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	RAO
Mapped	1-0000228	MANUFACT	DECORATED PROD CO	1 ARCH RD	WF	8/14/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE	LAGOON	RAO
Mapped	1-0000229	GASSTATION	CENTER CITY SERVICE STATION	1 FRANKLIN ST	WF	2/13/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNFA
Mapped	1-0000230	GASSTATION	MOBIL GAS	27 SOUTHWICK RD	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNFA
Mapped	1-0000232	MANUFACT	COLUMBIA MFG CO MTD	CYCLE ST	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	LAGOON	DEPNFA
Mapped	1-0000233	MANUFACT	PTS ELECTRONICS	300 UNION ST	WF	4/15/1987	UNKNOWN CHEMICAL OF TYPE - OIL	SEPTIC TANK	DEPNFA
Mapped	1-0000234	GASSTATION	REPUBLIC OIL	322 EAST MAIN ST	WF	4/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000235	INDUSTRIAL	SAVAGE INDUSTRIES	SPRINGDALE RD	WF	2/12/1987	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	UNCONTAIN, UST	RAO
Mapped	1-0000236	MANUFACT	TELL TOOL	TPKE INDUSTRIAL RD	WF	4/15/1987	UNKNOWN CHEMICAL OF TYPE - OIL	DRYWELL	DEPNFA
Mapped	1-0000237		WALTHAM GRINDING	30 EMERY ST	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000238	INDUSTRIAL, MANUFACT	INTERNATIONAL SALT	163 UNION ST	WF	4/15/1987	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNCONTAIN, UNKNOWN	RAO
Mapped	1-0000288		BARNES AIR NATIONAL GUARD	BARNES MUN A P BUCK PONDS RD	WF	10/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE (.5 INCH)	UNKNOWN	TIER1A
Mapped	1-0000315	INDUSTRIAL	WESTFIELD COATING	221 UNION ST	WF	10/15/1988	UNKNOWN CHEMICAL OF TYPE - OIL	LAGOON	RAO
Mapped	1-0000326	GASSTATION	GETTY POWER TEST	278 ELM ST	WF	10/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000328		WESTFIELD NEWS	64 SCHOOL ST	WF	1/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000343	COMMERCIAL	KANTANYS VOLKSWAGON	342 MAIN ST	WF	7/7/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNCONTAIN	RAONR
Mapped	1-0000356	GASSTATION	FIRESTONE	322 EAST MAIN ST	WF	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNDS
Mapped	1-0000381		TOWNSEND ASSOCIATES	79 MAINLINE DR	WF	10/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		LSPNFA
Mapped	1-0000427		LEAKING STORAGE TANK	163 UNION ST	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000428		PREFERRED ELECTRONICS INC	MAIN LINE DR	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000429		WESTFIELD FORD	234 EAST MAIN ST	WF	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000453	MANUFACT	DIGITAL	1111 SOUTHAMPTON RD	WF	1/15/1989	PETROLEUM BASED OIL	UST	WCSPRM

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0000485	GASSTATION	BOBS AUTOMOTIVE	97 SOUTH MAPLE ST	WF	6/20/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000489		BP STATION	88 SOUTH MAPLE ST	WF	6/21/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000510		AXTON CROSS CHEMICAL	TPKE INDUSTRIAL RD	WF	10/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000529	COMMERCIAL, FORMER, TANK FARM	ELM STREET PROPERTY	224 ELM ST	WF	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000540		RICHARDSON SILKSCREEN	798 APREMONT WAY	WF	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000544	FORMER, FUEL DEPOT	SCARFO REALTY	30 CLINTON AVE	WF	10/31/1988	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0000559		FILMTECH INC	181 NOTRE DAME ST	WF	1/15/1989	PETROLEUM BASED OIL		RAO
Mapped	1-0000571		PARKSIDE PARTS INC	17 PARKSIDE AVE	WF	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		WCSPRM
Mapped	1-0000586		MULTIFUELS CORPORATION	136 MEADOW ST	WF	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000589		WING FARM	TIMBERSWAMP RD	WF	1/27/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000601	TANK FARM	AGWAY MULTIFUELS	ARCH RD	WF	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE	RAO
Mapped	1-0000602		RYANS PACKAGE STORE	11 FRANKLIN ST	WF	3/23/1989	PETROLEUM BASED OIL	UST	DEPNDS
Mapped	1-0000604		PEER BROTHERS TRUCKING	253 UNION ST	WF	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000626		NATIONAL GUARD ARMORY	137 FRANKLIN ST	WF	4/27/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000633		PREFERRED ELECTRONICS 2	53 MAINLINE DR	WF	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000650		FOWLER FARMS	SOUTHWICK RD	WF	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		PENNFA
Mapped	1-0000665	GASSTATION	E MAIN MOBIL 01 PL6	460 EAST MAIN ST	WF	10/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000707	COMMERCIAL	AFM CORP	ELISE ST	WF	12/15/1989	WASTE OIL	DRYWELL	RAO
Mapped	1-0000734	COMMERCIAL	PLAZA MALL	288 SOUTHAMPTON RD	WF	4/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		LSPNFA
Mapped	1-0000758	INDUSTRIAL	N E CONCRETE & PIPE	69 NECK RD	WF	2/16/1990	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0000767	COMMERCIAL, MUNICIPAL	SACKETT STREET	30 SACKETT ST	WF	10/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE		TIER1A
Mapped	1-0000836	GASSTATION	MURRAYS MOBIL STATION 01 FKM	162 SOUTHAMPTON RD	WF	10/15/1990	UNKNOWN CHEMICAL OF TYPE - OIL	UST	RAO
Mapped	1-0000840	COMMERCIAL, FORMER, GASSTATION	TERRYS AUTO FMR	235 ELM	WF	10/15/1990	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0000851	GASSTATION	PVRR PARCEL	ADJACENT 235 ELM ST	WF	1/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000860		FMR POST OFFICE	25 RAILROAD AVE	WF	10/1/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE		DEPNFA
Mapped	1-0000868	GASSTATION	SHELL OIL STATION	259 NORTH ELM ST	WF	1/9/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	DPS
Mapped	1-0000869	GASSTATION	LECRENSKIS MOBIL	33 MAIN ST	WF	1/9/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE, UST	RAO
Mapped	1-0000875	GASSTATION	J J MOBIL MART 01 FMO	181 ELM ST	WF	4/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0000888	MANUFACT	ANDERSON & SONS	214 NORTH ELM ST	WF	4/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE		REMOPS
Mapped	1-0000910		MERIT STATION	310 EAST MAIN ST	WF	7/15/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0000918	INDUSTRIAL	DAY LUMBER COMPANY	SOUTH BROAD ST PO BOX 9	WF	7/15/1993	WASTE OIL	UST	RAO
Mapped	1-0000924	INDUSTRIAL	ATLANTIC VALVE COMPANY	TPKE INDUSTRIAL PARK	WF	10/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE	DRYWELL, UST	RAO
Mapped	1-0000955	INDUSTRIAL	PROP LEASED FROM JSLANE CO	UNION ST SPRINGDALE RD	WF	4/15/1992	WASTE OIL		PENNFA

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0000962	COMMERCIAL	WESTFIELD SAVINGS BANK	280 LOCKHOUSE RD	WF	7/15/1993	PETROLEUM BASED OIL	VEHICLE	WCSPRM
Mapped	1-0000987	FORMER, GASSTATION	STERLING RADIATOR	260 NORTH ELM ST	WF	10/30/1992	UNKNOWN CHEMICAL OF UNKNOWN TYPE		RAO
Mapped	1-0001012		STATE POLICE BARRACKS	MASSACHUSETTS TPKE MM 414	WF	4/20/1993	PETROLEUM BASED OIL	UST	RAO
Mapped	1-0001052	COLLEGE	WESTFIELD STATE COLLEGE	577 WESTERN AVE	WF	6/25/1993	FUEL OIL #6	UST	REMOPS
Mapped	1-0001073	GASSTATION	DEGRAYS SERVICE CENTER	163 MEADOW ST	WF	9/28/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE	RAO
Mapped	1-0001080	TANK FARM	WESTERN MASS HOSPITAL	91 EAST MOUNTAIN RD	WF	9/29/1993	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
Mapped	1-0010097	STATE	WESTFIELD MHD MAINT DEPOT	RT 20 (EAST MAIN ST)	WF	12/3/1993	DIESEL FUEL	UST	RAO
Mapped	1-0010141	AIR BASE, FEDERAL	BUILDING 028 UST/BARNES ANG BASE	1 TANK DESTROYER BLVD	WF	9/29/1994	FUEL OIL #2 (55 PPMV), TOTAL PETROLEUM HYDROCARBONS (TPH) (54.9 PPM)	TANKER, UST	RAONR
Mapped	1-0010144	AIR BASE, FEDERAL	BUILDING 051 UST	1 TANK DESTROYER BLVD	WF	12/23/1993	FUEL OIL #2 (73 PPMV), TOTAL PETROLEUM HYDROCARBONS (TPH) (73.2 PPM)	TANKER, UST	RAONR
Mapped	1-0010161	AIR BASE, FEDERAL	BARNES ANG BASE/BUILDING 027 UST	1 TANK DESTROYER BLVD	WF	1/7/1994	UNKNOWN CHEMICAL OF TYPE - OIL (483 PPMV), WASTE OIL (138 PPM), WASTE OIL (483 PPM)	UST	RAONR
Mapped	1-0010198	INDUSTRIAL, MUNICIPAL, WATERBO	HYDROPOWER GENERATING STATION	1717 GRANVILLE RD	WF	2/7/1994	MINERAL OIL (1000 GAL), MINERAL OIL (1475 GAL)	TRANSFORM	RAO
Mapped	1-0010202	RESIDNTIAL	RESIDENTIAL RT 10 TO CITYVIEW TO SACKETT	256 SACKETT RD	WF	2/14/1994	FUEL OIL #2 (30 GAL), FUEL OIL #2 (45 GAL)	PIPE	RAO
Mapped	1-0010298	INDUSTRIAL	CONSTRUCTION SERVICE PLANT	140 UNION ST	WF	4/15/1994	FUEL OIL #2 (200 GAL), FUEL OIL #2 (60 PPMV)	PIPE	STMRET
Mapped	1-0010333	AIR BASE, FEDERAL	ADJACENT TO BLDG 006	BARNES ANG BASE	WF	5/9/1994	OIL (500 PPMV)	UST	RAONR
Mapped	1-0010389	INDUSTRIAL	WESTFIELD COATINGS CORP	221 UNION ST	WF	6/20/1994	ETHANOL, 2-BUTOXY- (1000 GAL), ETHANOL, 2-BUTOXY- (1000 GAL)	PIPE	RAONR
Mapped	1-0010392	COMMERCIAL	FMR TERRYS AUTO SALES	235 ELM ST	WF	6/22/1994	GASOLINE (100 PPMV)	UST	RAO
Mapped	1-0010463		WESTFIELD COATINGS CORP	221 UNION ST	WF	8/8/1994	DIESEL FUEL (10 GAL), DIESEL FUEL (334 GAL)	AST	RAO
Mapped	1-0010475	SCHOOL, STATE	WESTFIELD STATE COLLEGE BOILER HOUSE	577 WESTERN AVE	WF	8/16/1994	FUEL OIL #6 (2 INCH), TOTAL PETROLEUM HYDROCARBONS (TPH) (39700 PPM), TOTAL PETROLEUM HYDROCARBONS (TPH) (4.3 PPM)	UST	RAONR
Mapped	1-0010505	RESIDNTIAL	FMR FOWLER FARMS PROPERTY	37 SOUTH MEADOW RD	WF	9/8/1994	1,3-BENZENEDICARBONITRILE, 2,4,5,6-TETRACHLORO- (520000), 6,9-METHANO-2,4,3-BENZODIOXATHIEPIN,6,7,8,9,10,10-H... (25000), PETROLEUM BASED OIL (25300 MG/KG), TOTAL PETROLEUM HYDROCARBONS (TPH)	PEST APPL	RAO
Mapped	1-0010545	WATERBODY	SPRINGFIELD WATER SUPPLY PARISH FILTERS	1515 GRANVILLE RD	WF	10/5/1994	FUEL OIL #2	UST	RAO

Table 1 (Continued)

MAPPED/NOT MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	CURRENT STATUS
Mapped	1-0010573	SCHOOL	HEADSTART OF WESTFIELD SO SIDE OF BLDG	390 SOUTHAMPTON RD	WF	10/24/1994	FUEL OIL #2, FUEL OIL #2 (10 GAL)	UST	RAO
Mapped	1-0010583	OPENSPACE, RESIDNTIAL	FMR RAYS EQUIPMENT	133 PROSPECT STREET EXT	WF	10/31/1994	WASTE OIL (100 GAL)	DRUMS	RAO
Mapped	1-0010595	AIRPORT, COMMERCIAL	KC AVIATION	33 ELISE ST	WF	11/3/1994	JET FUEL (150 PPMV), JET FUEL (50 PPMV)	UST	RAO
Mapped	1-0010623	COMMERCIAL	LECRENSKIS MOBIL	33 MAIN ST	WF	11/18/1994	GASOLINE (22.8 INCH), GASOLINE (9 GAL)	TANKER	RAO
Mapped	1-0010638	MUNICIPAL, WATSUP FAC	WEST PARISH FILTERS	GRANVILLE RD	WF	11/30/1994	FUEL OIL #2 (60 GAL)	PIPE, UST	RAO
Mapped	1-0010640	RESIDNTIAL	OAKS MOBILE HOME PARK LOT #21	404 SOUTHWICK RD	WF	12/2/1994	FUEL OIL #2, FUEL OIL #2 (200 GAL)	AST	RAO
Mapped	1-0010663	INDUSTRIAL	NEW ENGLAND CONCRETE PIPE	69 NECK RD	WF	12/19/1994	FUEL OIL #6, FUEL OIL #6 (1.5 INCH)	UST	RAONR
Not Mapped	1-0010776	COMMERCIAL	PRIDE GAS STATION	97 SOUTH MAIN ST	WF	3/20/1995	GASOLINE (846952 PPMV), GASOLINE (952 PPMV)	UST	RAONR
Mapped	1-0010788	COMMERCIAL	BAYBANK	30 ELM ST	WF	3/28/1995	FUEL OIL #2, FUEL OIL #2 (1000 PPMV)	UST	REMOPS
Mapped	1-0010794	GASSTATION	MERIT STATION KMART PLAZA	310 EAST MAIN ST	WF	3/30/1995	BENZENE, 1,2-DIMETHYL, BENZENE, ETHYL- (38 MG/L), BTEX (2.6 PPM), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (7.8 PPM), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (96 MG/L)	FMR REMOV, UST	TIERII
Mapped	1-0011028	STATE	WESTFIELD DEPOT FACILITY	EAST MAIN ST	WF	9/1/1995	TOTAL PETROLEUM HYDROCARBONS (TPH) (947 PPM)	UNKNOWN	RAO
Mapped	1-0011040	COMMERCIAL	GENESIS SPIRITUAL CENTER	53 MILL ST	WF	10/5/1995	FUEL OIL #2 (100 PPM)	UST	RAO
Mapped	1-0011055	COMMERCIAL, RESIDNTIAL	BAKER RESIDENCE	342 WEST RD	WF	9/22/1995	PETROLEUM BASED OIL (125 PPM)	UST	RAO
Mapped	1-0011126	RESIDNTIAL	C & S WHOLESALE GROCERS INC	BENNETT RD	WF	11/8/1995	FUEL OIL #2	AST	RAO
Mapped	1-0011165	STATE	WESTERN MASS HOSPITAL	91 EAST MOUNTAIN RD	WF	12/5/1995	FUEL OIL #2 (1000 PPMV)	UST	RAO
Mapped	1-0011354	COMMERCIAL	FOOD & FUEL STATION	1400 RUSSELL RD	WF	5/2/1996	GASOLINE	UST	RAO
Mapped	1-0011449	INDUSTRIAL	COLUMBIA MFG CO	CYCLE ST	WF	7/19/1996	FUEL OIL #4	UNKNOWN	ADQREG
Mapped	1-0011503	RESIDNTIAL	JOYNER PROPERTY	78 WESTERN AVE	WF	9/3/1996	FUEL OIL #2 (100 GAL)	UST	RAO
Mapped	1-0011520	COMMERCIAL	COMMERCIAL DISTRUBUTING CO	46 SOUTH BROAD ST	WF	9/16/1996	GASOLINE	UST	RAO
Mapped	1-0011687	RESIDNTIAL	MOBILE HOME	6 CLIFTON ST	WF	1/29/1997	FUEL OIL #2 (275 GAL)		TIER1D
Not Mapped	1-0011698	COMMERCIAL	REAR OF WAREHOUSE	SERVISTAR INDUSTRIAL WAY	WF	2/4/1997	FUEL OIL #2 (100 GAL)	PIPE, UST	RAO
Mapped	1-0011881	RESIDNTIAL	TERRANCE FLAHIVE RESIDENCE	34 TEKOA TER	WF	6/16/1997	FUEL OIL #2 (580 PPMV)	UST	RAO
Mapped	1-0011950	RESIDNTIAL	RESIDENTIAL FORECLOSURE	56 WESTERN AVE	WF	8/9/1997	FUEL OIL #2 (1000 PPMV)	UST	RAO
Mapped	1-0012001	COMMERCIAL	US LINE CO	14 UNION ST	WF	9/12/1997	FUEL OIL #2	UST	RAO
Mapped	1-0012037	COMMERCIAL	PRIDE GAS STATION	322 EAST MAIN ST	WF	10/8/1997	GASOLINE	PIPE	RAONR
Mapped	1-0012267	RESIDNTIAL	SAMEL RESIDENCE	179 SACKETT RD	WF	3/30/1998	FUEL OIL #2 (-80 GAL)	AST	TIER1D

Table 1 (Continued)

MAPPED	RTN	LOCATION AID	SITE NAME	ADDRESS	TOWN	DATE	MATERIALS	SOURCES	STATUS
Mapped	1-0012277	MUNICIPAL	FIRE STATION	34 BROAD ST	WF	4/6/1998	PETROLEUM BASED OIL	UST	TIERII
Mapped	1-0012292	RESIDNTIAL	SERENDIPITY GIFTS	1375 SOUTHAMPTON RD	WF	4/16/1998	GASOLINE (100 PPM)	UST	RAO
Mapped	1-0012355	RESIDNTIAL	NO LOCATION AID	5 TO 7 HIGH ST	WF	5/21/1998	FUEL OIL #2	UST	RAO
Mapped	1-0012385		POWDERMILL VILLAGE	126 UNION ST	WF	6/5/1998	FUEL OIL #2	UST	RAO
Mapped	1-0012414	OPENSPACE, RESIDNTIAL	LOT BETWEEN #11 AND #17	CROWN ST	WF	6/23/1998	ARSENIC (108 MG/KG)	UNKNOWN	TIER1D
Mapped	1-0012417	COMMERCIAL	NO LOCATION AID	110 AIRPORT RD	WF	6/25/1998	UNKNOWN CHEMICAL OF UNKNOWN TYPE (100 PPM)	UST	RAO
Not Mapped	1-0012565	ROADWAY	INTERCHANGE 3	RTE 90	WF	9/8/1998	PETROLEUM BASED OIL (-50 GAL)	VEHICLE	RAO
Mapped	1-0012619	ROADWAY	C & S FACILITY	53 SUMMIT LOCK RD	WF	10/1/1998	DIESEL FUEL (20 GAL)	VEHICLE	RAO
Mapped	1-0012768	COMMERCIAL	BEMBEN NURSERIES	17 BROOKLINE AVE	WF	12/29/1998	FUEL OIL #2 (125 PPM)	UST	RAO
Mapped	1-0012779	INDUSTRIAL	CALDOR DISTRIBUTION CENTER	1111 SOUTHAMPTON RD	WF	1/10/1999	FUEL OIL #4 (2000 GAL)	PIPE	RAO
Mapped	1-0012870	MUNICIPAL	WORKS	1515 GRANVILLE RD	WF	3/22/1999	GASOLINE (188 PPM)	UST	RAO
Mapped	1-0012983	STATE	WMASS HOSPITAL	91 EAST MOUNTAIN RD	WF	6/21/1999	GASOLINE (759 PPM)	UST	TIER1C
Mapped	1-0013160	RESIDNTIAL	DRIVEWAY	47 PINE RIDGE DR	WF	10/20/1999	FUEL OIL #2 (15 GAL)	PIPE	RAO
Mapped	1-0013182	RESIDNTIAL	RESIDENCE	148 NORTHWEST RD	WF	11/12/1999	FUEL OIL #2 (100 PPM)	UST	RAO
Not Mapped	1-0013224	ROADWAY	MILE MARKER 43.5	MASSACHUSETTS TPKE	WF	12/2/1999	DIESEL FUEL (20 GAL), WASTE OIL	TANKER	RAO
Mapped	1-0013341	RESIDNTIAL	ARBOR TRAILOR PARK	16 KLONDIKE AVE	WF	3/10/2000	KEROSENE (200 GAL)	AST	RAO
Mapped	1-0013417	SCHOOL	CROSS STREET PLAYGROUND	22 ASHLEY ST	WF	4/29/2000	DIESEL FUEL (10 GAL)	UNKNOWN	RAO
Mapped	1-0013432	INDUSTRIAL	COLUMBIA MANUFACTURING	SOUTH MEADOW RD	WF	5/10/2000	UNKNOWN CHEMICAL OF TYPE - OIL (100 GAL)	TRANSFORM	ADQREG
Mapped	1-0013436	RESIDNTIAL	RESIDENCE	1259 WESTERN AVE	WF	5/11/2000	FUEL OIL #2 (100 PPM)	UST	RAO
Mapped	1-0013528	COMMERCIAL, RESIDNTIAL	BUILDING 5	126 UNION ST	WF	7/11/2000	NAPHTHALENE (5.9 PPM), UNKNOWN CHEMICAL OF UNKNOWN TYPE (4500 PPM)	UST	RAO
Mapped	1-0013531	COMMERCIAL, RESIDNTIAL	BUILDING 4	126 UNION ST	WF	7/12/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (1800 PPM)	UST	RAO
Mapped	1-0013538	COMMERCIAL, RESIDNTIAL	BUILDING 6	126 UNION ST	WF	7/13/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (4700 PPM)	UST	RAO
Mapped	1-0013542	COMMERCIAL, RESIDNTIAL	BUILDING 3	126 UNION ST	WF	7/14/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (970 PPM)	UST	RAO
Mapped	1-0013550	COMMERCIAL, RESIDNTIAL	BUILDING 7	126 UNION ST	WF	7/21/2000	NAPHTHALENE (15.2 PPM), UNKNOWN CHEMICAL OF UNKNOWN TYPE (67000 PPM)	UST	RAO

Source: Massachusetts Department of Environmental Protection, Bureau of Waste Site Cleanup, 4/25/2005. Downloadable Site Lists.
http://www.state.ma.us/dep/bwsc/sites/sdown.htm

Notes:
RTN - Release Tracking Number. Unique ID assigned to releases not remediated by October 1993 and to those occuring October 1993-present.
Location Aid - Place name of release
Address - Street location of release
Town - EH (Easthampton), HO (Holyoke), SH (Southampton), WF (Westfield)
Date - Official release notification date of release
Materials - Chemical(s) in release
Sources - Origin(s) of release contamination
Current 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.
UST - Underground Storage Tank
AST - Aboveground Storage Tank
GAL - Gallon
PPMV - Parts per million by volume
PPM - Parts per million
PPB - Parts per billion
LBS - Pounds
MG/KG - Milligrams per kilogram

Table 2
Maximum concentration of trichloroethylene (TCE) detected in groundwater samples from the Pequot Well
Holyoke, Massachusetts
(samples taken from 1980–1992)

Location	Frequency of detection	Maximum concentration (ppb)	Date of sample	Drinking water comparison value (ppb)	Number of samples above comparison value
Pequot Wells	7 / 8	15	October 1984	MassDEP MMCL = 5	6

ppb = parts per billion

Data sources:
Holyoke Water Works. Data sheets concerning tests of Pequot Well and Coronet Homes Well. Holyoke, Massachusetts. 2002.
Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 1988.

Comparison value (source organization, reference):
MassDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MassDEP, MassDEP 2004c)

Table 3
Maximum concentration of trichloroethylene (TCE) detected in groundwater samples from Coronet Homes Well
Holyoke, Massachusetts
(samples taken from 1980–1988)

Location	Frequency of detection	Maximum concentration (ppb)	Date of sample	Drinking water comparison value (ppb)	Number of samples above comparision value
Coronet Homes Wells	4 / 6	1.7	October 1984	MassDEP MMCL = 5	0

ppb = parts per billion

Data sources:
Holyoke Water Works. Data sheets concerning tests of Pequot Well and Coronet Homes Well. Holyoke, Massachusetts. 2002.
Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 1988.

Comparison value (source organization, reference):
MassDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MassDEP, MassDEP 2004c)

Table 4
Maximum concentration of trichloroethylene (TCE) detected in groundwater samples from Easthampton Water Works wells
Easthampton, Massachusetts
(samples taken from 1980–2003)

Location	Frequency of detection	Maximum concentration (ppb)	Date of sample	Drinking water comparison value (ppb)	Number of samples above comparison value
Hendrick Street Wellfield	105 / 127	12.0	December 1991	MassDEP MMCL = 5	89
Hendrick Street/Pines Well Treatment Plant effluent	1 / 22	0.5	March 2000		0
Pines Well	70 / 72	9.5	March 2001		50

ppb = parts per billion

Data sources:

Easthampton Water Works. Data sheets concerning TCE testing of municipal water. Easthampton, Massachusetts. 2002.

Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 1988.

Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 2004.

Comparison value (source organization, reference):

MassDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MassDEP, MassDEP 2004c)

Table 5

Maximum concentration of trichloroethylene (TCE) detected in groundwater samples from Westfield Water Department wells that draw from the Barnes Aquifer (samples taken from 1986–2004)

Westfield, Massachusetts

Location	Frequency of detection	Maximum concentration (ppb)	Date of sample	Drinking water comparison value (ppb)	Number of samples above comparison value
Group Well #1	0 / 9	--	--	MassDEP MMCL = 5	0
Group Well #2	0 / 45	--	--		0
Group Well #7	0 / 50	--	--		0
Group Well #8	2 / 47	2.9	November 1993		0

ppb = parts per billion

Data sources:

Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 1988.

Massachusetts Department of Environmental Protection (MassDEP). Drinking Water Program, Water Quality Assurance Program. Statewide Purgeable Organic Testing Program data. Boston, Massachusetts. 2004.

Comparison value (source organization, reference):

MassDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MassDEP, MassDEP 2004c)

Table 6
Maximum concentrations of contaminants detected in 541 groundwater samples, 1997-2005, from 452 private wells in Easthampton, Holyoke, Southampton, and Westfield, Massachusetts

Contaminant	Frequency of Detection[a]	Maximum concentration (ppb)	Drinking water comparison value (ppb)	Number of samples above comparison values
Chloroform	13 / 541	24	Chronic EMEG (child) = 100 Chronic EMEG (adult) = 400 MassDEP MMCL = 70	0 0 0
1,4-Dichlorobenzene	1 / 541	3.1	Int. EMEG (child) = 700 Int. EMEG (adult) = 2,000 MassDEP MMCL = 5	0 0 0
1,1-Dichloroethene	1 / 541	0.018	Chronic EMEG (child) = 90 Chronic EMEG (adult) = 300 RMEG (child) = 500 RMEG (adult) = 2,000 MassDEP MMCL = 7	0 0 0 0 0
1,2-Dichloroethene, cis-	4 / 541	1.9	Int. EMEG (child) = 3,000 Int. EMEG (adult) = 10,000 MassDEP MMCL = 70	0 0 0
1,2-Dichloroethene, trans-	2 / 541	0.13	Int. EMEG (child) = 2,000 Int. EMEG (adult) = 7,000 RMEG (child) = 200 RMEG (adult) = 700 MassDEP MMCL = 100	0 0 0 0 0
Methyl-tert-butyl-ether (MTBE)	17 / 541	74	Int. EMEG (child) = 3,000 Int. EMEG (adult) = 10,000 MassDEP MMCL = 70	0 0 1
Naphthalene	2 / 541	5.4	Int. EMEG (child) = 6,000 Int. EMEG (adult) = 20,000 RMEG (child) = 200 RMEG (adult) = 700 MassDEP MMCL = 140	0 0 0 0 0
Tetrachloroethylene (PCE)	29 / 541	4.3	RMEG (child) = 100 RMEG (adult) = 400 MassDEP MMCL = 5	0 0 0

Table 6 (continued)

Contaminant	Frequency of Detection[a]	Maximum concentration (ppb)	Drinking water comparison value (ppb)	Number of samples above comparison values
Toluene	1 / 541	0.66	Int. EMEG (child) = 200 Int. EMEG (adult) = 700 RMEG (child) = 2,000 RMEG (adult) = 7,000 MassDEP MMCL = 1,000	0 0 0 0 0
1,1,1-Trichloroethane	3 / 541	2.2	Int. EMEG (child) = 200,000 Int. EMEG (adult) = 700,000 MassDEP MMCL = 200	0 0 0
Trichloroethylene (TCE)	107 / 541	34.2	MassDEP MMCL = 5	19

[a]Effluent samples not included

ppb = parts per billion

Data sources:

Massachusetts Department of Environmental Protection (MassDEP). Bureau of Waste Site Cleanup, Site Discovery Program. Private well data, 1997-2000, from Hendrick St. Wellfield/Barnes Aquifer TCE investigation. Springfield, Massachusetts. 2002.

Massachusetts Department of Environmental Protection (MassDEP). Bureau of Waste Site Cleanup, Site Discovery Program. Data sheets concerning private well data, 2001-2005, from Hendrick St. Wellfield/Barnes Aquifer TCE investigation. Springfield, Massachusetts. 2005b.

Comparison value (source organization, reference):

CREG = Cancer Risk Evaluation Guide for 1 x 10^{-6} excess cancer risk (ATSDR, ATSDR 2005a)

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2005a)

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, ATSDR 2005a)

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 2004)

MassDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MassDEP, MassDEP 2004c)

Table 7
Summary of Possible Exposure Pathways Related to Barnes Aquifer Contamination
Easthampton, Holyoke, Southampton, and Westfield, Massachusetts

Environmental Medium	Exposure Pathway	Contaminant(s)	Point of Exposure	Route of Exposure	Receptor Populations	Time Frame	Type of Pathway	Notes
Groundwater	Groundwater contamination	Trichloroethylene (TCE) and other volatile organic compounds (VOCs)	Offsite municipal water	Ingestion/ Inhalation/ Dermal contact	Residents	Past	Completed	Historical concentrations are unknown.
	Groundwater contamination	TCE and other VOCs	Offsite private well water	Ingestion/ Inhalation/ Dermal contact	Residents	Past	Completed	Exposure eliminated in present/future for residents who connected to municipal water or use whole house charcoal filters.
	Groundwater contamination	TCE and other VOCs	Offsite private well water	Ingestion/ Inhalation/ Dermal contact	Residents	Past, Present, Future	Completed/ Potential	Small number of residents who refused testing, might not properly maintain filters, or use unfiltered water.
Soil	Soil/dust	Polychlorinated biphenyls (PCBs)	Apremont Highway site, Dupuis Road site, nearby residences	Incidental Ingestion	Residents	Past	Potential	Sites have been remediated. Estimated past exposures to surface soil unlikely to result in adverse health effects.
Ambient Air	Combustion of PCBs	PCBs, dioxins, polychlorinated dibenzofurans	Dupuis Road site and nearby residences	Inhalation	Residents	Past	Potential	Historical ambient air concentrations are unknown.

TABLE 8a
Cancer Incidence
Easthampton, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	47	52.8	89	65 -- 118	35	38.7	91	63 -- 126	12	14.1	85	44 -- 149
Esophageal	15	16.3	92	51 -- 151	13	12.0	108	58 -- 185	2	4.3	NC	NC -- NC
Hodgkin's Disease	13	11.1	118	62 -- 201	7	6.1	115	46 -- 237	6	5.0	120	44 -- 262
Kidney	36	32.1	112	79 -- 155	22	19.8	111	70 -- 169	14	12.3	114	62 -- 191
Leukemia	23	27.8	83	52 -- 124	16	15.8	101	58 -- 165	7	12.0	58	23 -- 120
Liver	7	7.9	89	36 -- 183	7	5.8	121	49 -- 250	0	2.1	NC	NC -- NC
NHL	45	49.3	91	67 -- 122	25	26.1	96	62 -- 141	20	23.0	86	53 -- 133
Pancreatic	34	29.7	115	79 -- 160	16	14.4	111	64 -- 181	18	15.3	118	70 -- 186

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

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

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

TABLE 8b
Cancer Incidence
Easthampton, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	15	17.0	88	49 -- 146	9	12.5	72	33 -- 137	6	4.5	133	49 -- 289
Esophageal	4	4.4	NC	NC -- NC	4	3.2	NC	NC -- NC	0	1.3	NC	NC -- NC
Hodgkin's Disease	5	3.5	144	46 -- 337	2	1.9	NC	NC -- NC	3	1.6	NC	NC -- NC
Kidney	11	7.6	145	72 -- 260	5	4.5	110	36 -- 258	6	3.0	197	72 -- 429
Leukemia	6	7.7	78	29 -- 170	4	4.3	NC	NC -- NC	2	3.3	NC	NC -- NC
Liver	0	1.6	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.5	NC	NC -- NC
NHL	13	12.0	108	58 -- 185	9	6.2	144	66 -- 274	4	5.8	NC	NC -- NC
Pancreatic	8	8.5	94	40 -- 185	3	4.1	NC	NC -- NC	5	4.4	114	37 -- 266

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

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

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

TABLE 8c
Cancer Incidence
Easthampton, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	14	16.5	85	46 -- 143	11	12.1	91	45 -- 162	3	4.3	NC	NC -- NC
Esophageal	3	4.9	NC	NC -- NC	2	3.5	NC	NC -- NC	1	1.4	NC	NC -- NC
Hodgkin's Disease	4	3.7	NC	NC -- NC	3	2.0	NC	NC -- NC	1	1.7	NC	NC -- NC
Kidney	11	10.5	104	52 -- 187	8	6.6	121	52 -- 239	3	3.9	NC	NC -- NC
Leukemia	7	7.9	89	36 -- 183	6	4.6	131	48 -- 285	1	3.3	NC	NC -- NC
Liver	4	2.2	NC	NC -- NC	4	1.6	NC	NC -- NC	0	0.6	NC	NC -- NC
NHL	9	15.3	59	27 -- 111	2	8.2	NC	NC -- NC	7	7.1	98	39 -- 202
Pancreatic	5	8.8	57	18 -- 133	2	4.3	NC	NC -- NC	3	4.5	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

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

TABLE 8d
Cancer Incidence
Easthampton, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	18	19.5	92	55 -- 146	15	14.0	107	60 -- 177	3	5.5	NC	NC -- NC
Esophageal	8	7.0	114	49 -- 225	7	5.3	133	53 -- 275	1	1.8	NC	NC -- NC
Hodgkin's Disease	4	4.0	NC	NC -- NC	2	2.2	NC	NC -- NC	2	1.8	NC	NC -- NC
Kidney	14	14.1	99	54 -- 166	9	8.6	104	48 -- 198	5	5.5	91	29 -- 212
Leukemia	10	12.5	80	38 -- 148	6	6.8	88	32 -- 191	4	5.6	NC	NC -- NC
Liver	3	4.0	NC	NC -- NC	3	3.0	NC	NC -- NC	0	1.1	NC	NC -- NC
NHL	23	22.4	103	65 -- 154	14	11.7	120	65 -- 201	9	10.7	84	38 -- 160
Pancreatic	21	12.6	167*	103 -- 255	11	5.9	186	93 -- 332	10	6.6	151	72 -- 277

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

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

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

TABLE 9a
Cancer Incidence
Census Tract 8223
Easthampton, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	22	22.9	96	60 -- 146	18	16.5	109	65 -- 173	4	6.4	NC	NC -- NC
Esophageal	10	6.9	145	69 -- 266	9	4.9	183	83 -- 347	1	2.0	NC	NC -- NC
Hodgkin's Disease	4	3.7	NC	NC -- NC	4	2.0	NC	NC -- NC	0	1.7	NC	NC -- NC
Kidney	10	13.2	76	36 -- 140	6	7.9	76	28 -- 165	4	5.3	NC	NC -- NC
Leukemia	5	11.2	45	14 -- 104	3	6.2	NC	NC -- NC	2	5.0	NC	NC -- NC
Liver	1	3.3	NC	NC -- NC	1	2.3	NC	NC -- NC	0	0.9	NC	NC -- NC
NHL	22	20.1	109	68 -- 165	14	10.1	138	75 -- 232	8	10.0	80	34 -- 158
Pancreatic	12	13.0	92	48 -- 161	4	6.0	NC	NC -- NC	8	7.0	114	49 -- 225

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

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

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

TABLE 9b
Cancer Incidence
Census Tract 8223
Easthampton, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	7.7	117	53 -- 222	7	5.6	126	51 -- 260	2	2.1	NC	NC -- NC
Esophageal	2	2.0	NC	NC -- NC	2	1.4	NC	NC -- NC	0	0.6	NC	NC -- NC
Hodgkin's Disease	2	1.2	NC	NC -- NC	2	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Kidney	3	3.3	NC	NC -- NC	1	1.9	NC	NC -- NC	2	1.4	NC	NC -- NC
Leukemia	1	3.2	NC	NC -- NC	1	1.8	NC	NC -- NC	0	1.5	NC	NC -- NC
Liver	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
NHL	4	5.2	NC	NC -- NC	3	2.6	NC	NC -- NC	1	2.6	NC	NC -- NC
Pancreatic	3	3.9	NC	NC -- NC	2	1.8	NC	NC -- NC	1	2.1	NC	NC -- NC

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

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

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

TABLE 9c
Cancer Incidence
Census Tract 8223
Easthampton, 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	7.2	83	30 -- 181	5	5.2	95	31 -- 222	1	2.0	NC	NC -- NC
Esophageal	2	2.1	NC	NC -- NC	2	1.5	NC	NC -- NC	0	0.6	NC	NC -- NC
Hodgkin's Disease	1	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Kidney	4	4.4	NC	NC -- NC	3	2.7	NC	NC -- NC	1	1.7	NC	NC -- NC
Leukemia	2	3.2	NC	NC -- NC	1	1.8	NC	NC -- NC	1	1.4	NC	NC -- NC
Liver	0	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.3	NC	NC -- NC
NHL	4	6.3	NC	NC -- NC	1	3.2	NC	NC -- NC	3	3.1	NC	NC -- NC
Pancreatic	0	3.9	NC	NC -- NC	0	1.8	NC	NC -- NC	0	2.1	NC	NC -- NC

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

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

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

TABLE 9d
Cancer Incidence
Census Tract 8223
Easthampton, 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	7.9	89	36 -- 183	6	5.6	108	39 -- 235	1	2.3	NC	NC -- NC
Esophageal	6	2.7	221	81 -- 481	5	2.0	254	82 -- 592	1	0.7	NC	NC -- NC
Hodgkin's Disease	1	1.4	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Kidney	3	5.4	NC	NC -- NC	2	3.2	NC	NC -- NC	1	2.2	NC	NC -- NC
Leukemia	2	4.8	NC	NC -- NC	1	2.5	NC	NC -- NC	1	2.2	NC	NC -- NC
Liver	1	1.5	NC	NC -- NC	1	1.1	NC	NC -- NC	0	0.4	NC	NC -- NC
NHL	14	8.6	163	89 -- 274	10	4.3	233*	111 -- 428	4	4.3	NC	NC -- NC
Pancreatic	9	5.1	177	81 -- 336	2	2.3	NC	NC -- NC	7	2.8	249	100 -- 513

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

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

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

TABLE 10a
Cancer Incidence
Census Tract 8224
Easthampton, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	23	30.0	77	49 -- 115	16	22.3	72	41 -- 117	7	7.7	91	36 -- 187
Esophageal	5	9.5	53	17 -- 123	4	7.1	NC	NC -- NC	1	2.4	NC	NC -- NC
Hodgkin's Disease	8	7.3	109	47 -- 215	3	4.1	NC	NC -- NC	5	3.3	153	49 -- 357
Kidney	25	18.9	132	85 -- 195	15	11.9	126	71 -- 208	10	7.1	142	68 -- 261
Leukemia	18	16.6	108	64 -- 171	13	9.6	135	72 -- 231	5	7.0	71	23 -- 166
Liver	6	4.6	130	47 -- 282	6	3.4	175	64 -- 380	0	1.2	NC	NC -- NC
NHL	21	29.2	72	44 -- 110	10	16.0	63	30 -- 115	11	13.2	83	41 -- 149
Pancreatic	22	16.7	132	83 -- 199	12	8.4	142	74 -- 249	10	8.3	121	58 -- 222

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

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

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

TABLE 10b
Cancer Incidence
Census Tract 8224
Easthampton, 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	9.3	65	24 -- 141	2	6.9	NC	NC -- NC	4	2.4	NC	NC -- NC
Esophageal	2	2.4	NC	NC -- NC	2	1.8	NC	NC -- NC	0	0.7	NC	NC -- NC
Hodgkin's Disease	2	2.3	NC	NC -- NC	0	1.2	NC	NC -- NC	2	1.0	NC	NC -- NC
Kidney	8	4.2	189	81 -- 372	4	2.6	NC	NC -- NC	4	1.7	NC	NC -- NC
Leukemia	5	4.4	113	36 -- 263	3	2.6	NC	NC -- NC	2	1.9	NC	NC -- NC
Liver	0	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC
NHL	8	6.8	118	51 -- 233	5	3.6	138	44 -- 321	3	3.1	NC	NC -- NC
Pancreatic	5	4.6	110	35 -- 256	1	2.3	NC	NC -- NC	4	2.3	NC	NC -- NC

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

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

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

TABLE 10c
Cancer Incidence
Census Tract 8224
Easthampton, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	7	9.2	76	30 -- 156	5	6.9	73	23 -- 170	2	2.4	NC	NC -- NC
Esophageal	1	2.8	NC	NC -- NC	0	2.1	NC	NC -- NC	1	0.7	NC	NC -- NC
Hodgkin's Disease	3	2.4	NC	NC -- NC	2	1.3	NC	NC -- NC	1	1.1	NC	NC -- NC
Kidney	6	6.1	98	36 -- 213	4	3.9	NC	NC -- NC	2	2.2	NC	NC -- NC
Leukemia	5	4.7	106	34 -- 248	5	2.8	180	58 -- 421	0	1.9	NC	NC -- NC
Liver	4	1.3	NC	NC -- NC	4	1.0	NC	NC -- NC	0	0.3	NC	NC -- NC
NHL	5	9.0	55	18 -- 129	1	5.0	NC	NC -- NC	4	4.0	NC	NC -- NC
Pancreatic	5	4.9	102	33 -- 238	2	2.5	NC	NC -- NC	3	2.4	NC	NC -- NC

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

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

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

TABLE 10d
Cancer Incidence
Census Tract 8224
Easthampton, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	10	11.6	86	41 -- 158	9	8.5	106	48 -- 202	1	3.2	NC	NC -- NC
Esophageal	2	4.3	NC	NC -- NC	2	3.3	NC	NC -- NC	0	1.0	NC	NC -- NC
Hodgkin's Disease	3	2.7	NC	NC -- NC	1	1.5	NC	NC -- NC	2	1.2	NC	NC -- NC
Kidney	11	8.8	125	62 -- 224	7	5.5	128	51 -- 264	4	3.3	NC	NC -- NC
Leukemia	8	7.7	104	45 -- 205	5	4.3	117	38 -- 272	3	3.4	NC	NC -- NC
Liver	2	2.5	NC	NC -- NC	2	1.9	NC	NC -- NC	0	0.6	NC	NC -- NC
NHL	8	13.8	58	25 -- 114	4	7.4	NC	NC -- NC	4	6.4	NC	NC -- NC
Pancreatic	12	7.5	160	83 -- 280	9	3.7	246*	112 -- 466	3	3.8	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

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

TABLE 11a
Cancer Incidence
Holyoke, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	149	163.7	91	77 -- 107	99	113.2	87	71 -- 106	50	50.5	99	74 -- 131
Esophageal	58	49.0	118	90 -- 153	37	33.5	111	78 -- 152	21	15.5	135	84 -- 207
Hodgkin's Disease	24	28.9	83	53 -- 124	13	14.8	88	47 -- 151	11	14.1	78	39 -- 139
Kidney	82	93.5	88	70 -- 109	45	54.0	83	61 -- 112	37	39.5	94	66 -- 129
Leukemia	97	86.7	112	91 -- 136	51	45.2	113	84 -- 148	46	41.5	111	81 -- 148
Liver	19	23.1	82	49 -- 128	14	15.9	88	48 -- 148	5	7.3	69	22 -- 161
NHL	140	147.4	95	80 -- 112	67	70.3	95	74 -- 121	73	77.1	95	74 -- 119
Pancreatic	99	95.1	104	85 -- 127	41	40.9	100	72 -- 136	58	54.2	107	81 -- 138

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

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

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

TABLE 11b
Cancer Incidence
Holyoke, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	61	60.8	100	77 -- 129	40	42.1	95	68 -- 130	21	18.7	112	69 -- 171
Esophageal	15	15.3	98	55 -- 162	9	10.2	88	40 -- 168	6	5.1	117	43 -- 256
Hodgkin's Disease	7	9.4	74	30 -- 153	5	4.9	103	33 -- 240	2	4.6	NC	NC -- NC
Kidney	22	25.5	86	54 -- 130	10	14.2	71	34 -- 130	12	11.4	106	55 -- 185
Leukemia	29	26.8	108	72 -- 155	15	14.0	107	60 -- 176	14	12.8	109	60 -- 183
Liver	5	5.6	89	29 -- 207	3	3.7	NC	NC -- NC	2	2.0	NC	NC -- NC
NHL	37	41.2	90	63 -- 124	19	19.2	99	60 -- 155	18	22.0	82	48 -- 129
Pancreatic	33	31.6	104	72 -- 147	13	13.6	96	51 -- 164	20	18.0	111	68 -- 171

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

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

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

TABLE 11c
Cancer Incidence
Holyoke, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	33	51.6	64*	44 -- 90	21	36.0	58*	36 -- 89	12	15.7	77	40 -- 134
Esophageal	18	15.1	119	71 -- 188	11	10.0	110	55 -- 197	7	5.1	137	55 -- 282
Hodgkin's Disease	9	9.7	93	42 -- 176	4	4.9	NC	NC -- NC	5	4.8	104	34 -- 243
Kidney	29	31.2	93	62 -- 134	17	18.4	93	54 -- 148	12	12.8	94	48 -- 164
Leukemia	36	24.8	145*	102 -- 201	16	13.3	121	69 -- 196	20	11.5	173*	106 -- 268
Liver	7	6.6	106	42 -- 218	6	4.6	130	47 -- 283	1	2.0	NC	NC -- NC
NHL	44	46.5	95	69 -- 127	24	22.1	109	70 -- 162	20	24.4	82	50 -- 127
Pancreatic	27	28.5	95	62 -- 138	11	12.3	89	45 -- 160	16	16.2	99	56 -- 160

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

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

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

TABLE 11d
Cancer Incidence
Holyoke, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	55	53.8	102	77 -- 133	38	37.4	102	72 -- 140	17	16.4	103	60 -- 166
Esophageal	25	18.6	134	87 -- 198	17	13.3	128	74 -- 204	8	5.3	151	65 -- 298
Hodgkin's Disease	8	9.7	82	35 -- 162	4	5.0	NC	NC -- NC	4	4.7	NC	NC -- NC
Kidney	31	36.3	85	58 -- 121	18	21.3	85	50 -- 134	13	15.0	87	46 -- 148
Leukemia	32	34.2	93	64 -- 132	20	17.7	113	69 -- 174	12	16.5	73	38 -- 127
Liver	7	10.4	67	27 -- 138	5	7.3	69	22 -- 160	2	3.1	NC	NC -- NC
NHL	59	58.6	101	77 -- 130	24	28.8	83	53 -- 124	35	29.8	117	82 -- 163
Pancreatic	39	35.1	111	79 -- 152	17	15.3	111	65 -- 178	22	19.8	111	70 -- 168

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

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

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

TABLE 12a
Cancer Incidence
Census Tract 8121
Holyoke, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	57	68.0	84	64 -- 109	41	48.4	85	61 -- 115	16	19.5	82	47 -- 133
Esophageal	22	20.0	110	69 -- 166	11	14.0	78	39 -- 140	11	6.0	183	91 -- 328
Hodgkin's Disease	7	9.7	72	29 -- 149	3	5.2	NC	NC -- NC	4	4.5	NC	NC -- NC
Kidney	34	37.1	92	64 -- 128	18	22.2	81	48 -- 128	16	14.9	107	61 -- 175
Leukemia	39	32.7	119	85 -- 163	24	17.7	136	87 -- 202	15	15.0	100	56 -- 165
Liver	11	9.3	119	59 -- 212	9	6.5	138	63 -- 261	2	2.7	NC	NC -- NC
NHL	56	57.6	97	73 -- 126	26	28.5	91	59 -- 133	30	29.1	103	70 -- 147
Pancreatic	43	38.2	113	82 -- 152	14	17.3	81	44 -- 136	29	20.9	139	93 -- 199

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

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

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

TABLE 12b
Cancer Incidence
Census Tract 8121
Holyoke, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	16	22.0	73	42 -- 118	14	15.7	89	49 -- 150	2	6.3	NC	NC -- NC
Esophageal	4	5.4	NC	NC -- NC	1	3.8	NC	NC -- NC	3	1.7	NC	NC -- NC
Hodgkin's Disease	1	3.0	NC	NC -- NC	1	1.6	NC	NC -- NC	0	1.4	NC	NC -- NC
Kidney	6	8.9	68	25 -- 147	1	5.2	NC	NC -- NC	5	3.7	135	43 -- 315
Leukemia	10	9.1	110	53 -- 203	6	4.9	122	44 -- 265	4	4.1	NC	NC -- NC
Liver	4	2.0	NC	NC -- NC	2	1.3	NC	NC -- NC	2	0.7	NC	NC -- NC
NHL	21	14.2	148	91 -- 226	11	7.0	158	79 -- 282	10	7.3	138	66 -- 254
Pancreatic	12	11.0	109	56 -- 191	5	5.0	99	32 -- 232	7	6.0	118	47 -- 242

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

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

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

TABLE 12c
Cancer Incidence
Census Tract 8121
Holyoke, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	15	21.2	71	40 -- 117	8	15.2	53	23 -- 104	7	6.0	117	47 -- 241
Esophageal	8	6.1	131	56 -- 257	4	4.2	NC	NC -- NC	4	2.0	NC	NC -- NC
Hodgkin's Disease	3	3.2	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.5	NC	NC -- NC
Kidney	9	12.3	73	33 -- 139	5	7.5	67	22 -- 156	4	4.8	NC	NC -- NC
Leukemia	10	9.2	108	52 -- 200	4	5.1	NC	NC -- NC	6	4.1	146	53 -- 317
Liver	5	2.6	NC	NC -- NC	5	1.9	265	85 -- 617	0	0.7	NC	NC -- NC
NHL	14	18.0	78	43 -- 131	6	8.8	68	25 -- 148	8	9.2	87	38 -- 172
Pancreatic	17	11.3	150	87 -- 241	5	5.2	97	31 -- 226	12	6.2	195*	101 -- 340

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

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

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

TABLE 12d
Cancer Incidence
Census Tract 8121
Holyoke, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	26	23.9	109	71 -- 159	19	17.2	111	67 -- 173	7	6.7	104	42 -- 215
Esophageal	10	8.0	125	60 -- 230	6	5.8	103	38 -- 224	4	2.2	NC	NC -- NC
Hodgkin's Disease	3	3.4	NC	NC -- NC	0	1.8	NC	NC -- NC	3	1.6	NC	NC -- NC
Kidney	19	15.1	126	76 -- 197	12	9.1	131	68 -- 229	7	5.9	118	47 -- 243
Leukemia	19	13.7	139	83 -- 216	14	7.4	190*	104 -- 318	5	6.3	79	25 -- 184
Liver	2	4.4	NC	NC -- NC	2	3.1	NC	NC -- NC	0	1.2	NC	NC -- NC
NHL	21	24.2	87	54 -- 133	9	12.4	73	33 -- 138	12	11.8	102	52 -- 177
Pancreatic	14	15.0	94	51 -- 157	4	6.8	NC	NC -- NC	10	8.1	123	59 -- 227

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

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

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

TABLE 13a
Cancer Incidence
Southampton, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	20	12.8	156	95 -- 241	17	9.8	173*	101 -- 277	3	3.0	NC	NC -- NC
Esophageal	2	4.1	NC	NC -- NC	1	3.2	NC	NC -- NC	1	0.9	NC	NC -- NC
Hodgkin's Disease	3	3.1	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.3	NC	NC -- NC
Kidney	7	8.3	84	34 -- 173	1	5.4	NC	NC -- NC	6	2.9	206	75 -- 448
Leukemia	7	7.1	98	39 -- 202	4	4.3	NC	NC -- NC	3	2.9	NC	NC -- NC
Liver	0	2.0	NC	NC -- NC	0	1.6	NC	NC -- NC	0	0.5	NC	NC -- NC
NHL	7	12.5	56	22 -- 115	3	7.2	NC	NC -- NC	4	5.3	NC	NC -- NC
Pancreatic	7	7.0	101	40 -- 207	2	3.8	NC	NC -- NC	5	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

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

TABLE 13b
Cancer Incidence
Southampton, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	8	3.6	220	95 -- 433	6	2.8	216	79 -- 470	2	0.9	NC	NC -- NC
Esophageal	0	1.0	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
Hodgkin's Disease	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
Kidney	1	1.7	NC	NC -- NC	1	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC
Leukemia	2	1.7	NC	NC -- NC	0	1.0	NC	NC -- NC	2	0.7	NC	NC -- NC
Liver	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
NHL	1	2.7	NC	NC -- NC	0	1.5	NC	NC -- NC	1	1.2	NC	NC -- NC
Pancreatic	1	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC

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

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

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

TABLE 13c
Cancer Incidence
Southampton, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	3.9	230*	105 -- 436	8	3.0	266*	114 -- 524	1	0.9	NC	NC -- NC
Esophageal	1	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.3	NC	NC -- NC
Hodgkin's Disease	1	1.0	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC
Kidney	2	2.7	NC	NC -- NC	0	1.8	NC	NC -- NC	2	0.9	NC	NC -- NC
Leukemia	2	2.0	NC	NC -- NC	1	1.2	NC	NC -- NC	1	0.8	NC	NC -- NC
Liver	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
NHL	3	3.8	NC	NC -- NC	2	2.2	NC	NC -- NC	1	1.6	NC	NC -- NC
Pancreatic	5	2.0	248	80 -- 579	1	1.1	NC	NC -- NC	4	0.9	NC	NC -- NC

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

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

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

TABLE 13d
Cancer Incidence
Southampton, 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	5.2	NC	NC -- NC	3	3.8	NC	NC -- NC	0	1.3	NC	NC -- NC
Esophageal	1	2.0	NC	NC -- NC	1	1.6	NC	NC -- NC	0	0.4	NC	NC -- NC
Hodgkin's Disease	2	1.2	NC	NC -- NC	2	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
Kidney	4	4.1	NC	NC -- NC	0	2.6	NC	NC -- NC	4	1.5	NC	NC -- NC
Leukemia	3	3.5	NC	NC -- NC	3	2.0	NC	NC -- NC	0	1.5	NC	NC -- NC
Liver	0	1.2	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.3	NC	NC -- NC
NHL	3	6.3	NC	NC -- NC	1	3.5	NC	NC -- NC	2	2.8	NC	NC -- NC
Pancreatic	1	3.3	NC	NC -- NC	0	1.7	NC	NC -- NC	1	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

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

TABLE 14a
Cancer Incidence
Westfield, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	147	128.9	114	96 -- 134	110	92.5	119	98 -- 143	37	36.3	102	72 -- 140
Esophageal	37	39.8	93	65 -- 128	29	28.6	101	68 -- 145	8	11.2	72	31 -- 141
Hodgkin's Disease	21	27.2	77	48 -- 118	8	14.4	56	24 -- 110	13	12.8	102	54 -- 174
Kidney	77	77.7	99	78 -- 124	43	46.8	92	66 -- 124	34	30.8	110	76 -- 154
Leukemia	65	68.8	95	73 -- 120	40	37.8	106	76 -- 144	25	31.0	81	52 -- 119
Liver	21	19.1	110	68 -- 168	16	13.7	117	67 -- 190	5	5.4	93	30 -- 217
NHL	94	120.2	78*	63 -- 96	51	61.6	83	62 -- 109	43	58.6	73*	53 -- 99
Pancreatic	66	73.4	90	70 -- 114	39	34.3	114	81 -- 155	27	39.1	69	45 -- 100

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

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

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

TABLE 14b
Cancer Incidence
Westfield, Massachsuetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	45	40.7	111	81 -- 148	33	29.4	113	77 -- 158	12	11.3	106	55 -- 185
Esophageal	8	10.6	76	33 -- 149	5	7.5	67	22 -- 156	3	3.1	NC	NC -- NC
Hodgkin's Disease	6	8.2	73	27 -- 158	2	4.4	NC	NC -- NC	4	3.9	NC	NC -- NC
Kidney	21	18.0	117	72 -- 178	11	10.6	104	52 -- 186	10	7.4	135	65 -- 249
Leukemia	17	18.5	92	54 -- 148	10	10.2	98	47 -- 179	7	8.2	85	34 -- 176
Liver	1	3.9	NC	NC -- NC	1	2.7	NC	NC -- NC	0	1.2	NC	NC -- NC
NHL	19	28.6	66	40 -- 104	10	14.5	69	33 -- 126	9	14.1	64	29 -- 121
Pancreatic	14	20.6	68	37 -- 114	9	9.7	93	42 -- 176	5	10.9	46	15 -- 107

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

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

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

TABLE 14c
Cancer Incidence
Westfield, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	49	40.2	122	90 -- 161	37	29.0	128	90 -- 176	12	11.2	107	55 -- 187
Esophageal	11	12.0	91	46 -- 164	8	8.4	95	41 -- 187	3	3.6	NC	NC -- NC
Hodgkin's Disease	6	9.0	67	24 -- 146	4	4.7	NC	NC -- NC	2	4.3	NC	NC -- NC
Kidney	20	25.5	78	48 -- 121	12	15.6	77	40 -- 134	8	9.9	81	35 -- 160
Leukemia	19	19.5	97	59 -- 152	12	10.9	110	57 -- 192	7	8.6	82	33 -- 168
Liver	7	5.4	131	52 -- 269	5	3.9	129	42 -- 202	2	1.5	NC	NC -- NC
NHL	27	37.4	72	48 -- 105	13	19.2	68	36 -- 116	14	18.2	77	42 -- 129
Pancreatic	23	21.7	106	67 -- 159	14	10.2	137	75 -- 230	9	11.5	78	36 -- 148

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

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

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

TABLE 14d
Cancer Incidence
Westfield, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	53	47.1	113	84 -- 147	40	34.0	118	85 -- 161	13	13.3	97	52 -- 167
Esophageal	18	16.9	106	63 -- 168	16	12.6	127	72 -- 206	2	4.3	NC	NC -- NC
Hodgkin's Disease	9	9.9	91	42 -- 173	2	5.3	NC	NC -- NC	7	4.6	153	61 -- 316
Kidney	36	33.7	107	75 -- 148	20	20.5	98	60 -- 151	16	13.2	121	69 -- 196
Leukemia	29	30.3	96	64 -- 137	18	16.5	109	65 -- 173	11	13.8	79	40 -- 142
Liver	13	9.7	134	71 -- 230	10	7.1	142	68 -- 261	3	2.6	NC	NC -- NC
NHL	48	53.4	90	66 -- 119	28	27.8	101	67 -- 146	20	25.7	78	48 -- 120
Pancreatic	29	30.4	95	64 -- 137	16	14.2	112	64 -- 182	13	16.2	80	43 -- 137

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

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

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

TABLE 15a
Cancer Incidence
Census Tract 8125
Westfield, Massachusetts
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	28	18.1	155*	103 -- 223	22	13.8	160	100 -- 242	6	4.4	137	50 -- 299
Esophageal	6	5.8	103	38 -- 224	5	4.5	111	36 -- 260	1	1.3	NC	NC -- NC
Hodgkin's Disease	8	4.6	174	75 -- 342	2	2.5	NC	NC -- NC	6	2.1	287*	105 -- 624
Kidney	9	11.7	77	35 -- 146	6	7.5	80	29 -- 175	3	4.3	NC	NC -- NC
Leukemia	15	10.3	146	82 -- 241	9	6.0	150	68 -- 284	6	4.2	141	52 -- 308
Liver	2	2.9	NC	NC -- NC	1	2.2	NC	NC -- NC	1	0.7	NC	NC -- NC
NHL	12	17.6	68	35 -- 119	3	9.9	NC	NC -- NC	9	7.8	116	53 -- 220
Pancreatic	9	9.9	91	41 -- 172	8	5.2	152	66 -- 300	1	4.7	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

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

TABLE 15b
Cancer Incidence
Census Tract 8125
Westfield, Massachusetts
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	10	5.6	179	86 -- 329	8	4.3	187	81 -- 369	2	1.3	NC	NC -- NC
Esophageal	0	1.5	NC	NC -- NC	0	1.1	NC	NC -- NC	0	0.4	NC	NC -- NC
Hodgkin's Disease	3	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	3	0.7	NC	NC -- NC
Kidney	2	2.6	NC	NC -- NC	1	1.6	NC	NC -- NC	1	1.0	NC	NC -- NC
Leukemia	4	2.7	NC	NC -- NC	2	1.6	NC	NC -- NC	2	1.1	NC	NC -- NC
Liver	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
NHL	5	4.1	123	40 -- 287	1	2.3	NC	NC -- NC	4	1.8	NC	NC -- NC
Pancreatic	0	2.7	NC	NC -- NC	0	1.4	NC	NC -- NC	0	1.2	NC	NC -- NC

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

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

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

TABLE 15c
Cancer Incidence
Census Tract 8125
Westfield, Massachusetts
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	5.6	161	73 -- 305	7	4.3	164	66 -- 339	2	1.3	NC	NC -- NC
Esophageal	3	1.7	NC	NC -- NC	2	1.3	NC	NC -- NC	1	0.4	NC	NC -- NC
Hodgkin's Disease	2	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
Kidney	2	3.8	NC	NC -- NC	1	2.5	NC	NC -- NC	1	1.4	NC	NC -- NC
Leukemia	2	2.9	NC	NC -- NC	1	1.7	NC	NC -- NC	1	1.2	NC	NC -- NC
Liver	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
NHL	3	5.4	NC	NC -- NC	1	3.1	NC	NC -- NC	2	2.4	NC	NC -- NC
Pancreatic	5	2.9	172	55 -- 400	4	1.6	NC	NC -- NC	1	1.4	NC	NC -- NC

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

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

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

TABLE 15d
Cancer Incidence
Census Tract 8125
Westfield, Massachusetts
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	7.0	128	59 -- 243	7	5.2	133	53 -- 275	2	1.8	NC	NC -- NC
Esophageal	3	2.7	NC	NC -- NC	3	2.1	NC	NC -- NC	0	0.6	NC	NC -- NC
Hodgkin's Disease	3	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.8	NC	NC -- NC
Kidney	5	5.4	92	30 -- 214	4	3.4	NC	NC -- NC	1	2.0	NC	NC -- NC
Leukemia	9	4.8	186	85 -- 353	6	2.7	218	80 -- 475	3	2.1	NC	NC -- NC
Liver	2	1.6	NC	NC -- NC	1	1.2	NC	NC -- NC	1	0.4	NC	NC -- NC
NHL	4	8.4	NC	NC -- NC	1	4.6	NC	NC -- NC	3	3.8	NC	NC -- NC
Pancreatic	4	4.5	NC	NC -- NC	4	2.3	NC	NC -- NC	0	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

Data Source: Massachusetts Cancer Registry, Center for Health Information, 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
Esophagus	150.0-150.9	except 9590-9980	C15.0-C15.9	except 9590-9989	C15.0-C15.9	except 9590-9989
Hodgkin's Disease	140.0-199.9	includes O9650-O9667, P9653-P9683, B9653-B9658	C00.00-C80.9	includes 9650-9667	C00.00-C80.9	includes 9650-9667
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
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
Pancreas	157.0-157.9	except 9590-9980	C25.0-C25.9	except 9590-9989	C25.0-C25.9	except 9590-9989

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

ICD-O = International Classification of Diseases for Oncology

Appendix B

Risk Factor Information for Selected Cancer Types

Bladder Cancer

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

The greatest risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS 2006a). The risk of developing bladder cancer increases with the number of packs smoked per day and with duration of smoking. Further, the risk of bladder cancer may be higher in women than in men who smoke comparable numbers of cigarettes (Castelao et al. 2001). Approximately 25-60% of all bladder cancers can be attributed to tobacco use (Johansson and Cohen 1997). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30% to 60% (Silverman et al. 1996).

Studies have also revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the U.S. (Johansson and Cohen 1997). Occupational exposure to aromatic amines, such as benzidine and beta-naphthylamine, increases the risk of bladder cancer (ACS 2006b). These chemicals were common in the dye industry in the past. A higher risk of bladder cancer has also been observed among aromatic amine manufacturing workers as well as among workers in the rubber, leather, textiles, printing, and paint products industries (ACS 2006a, Silverman et al. 1996). The development of new chemicals, changed worker exposures, and the elimination of many known bladder carcinogens in the workplace have caused shifts in those occupations considered to be high risk. For example, risks among dye, rubber, and leather workers have declined over time, while other occupations such as motor vehicle operation (e.g., drivers of trucks, buses, and taxis) and the aluminum industry have emerged as potential high-risk occupations (Silverman et al. 1996). However, specific occupational exposures in these occupations have not been confirmed and study findings are not consistent. Further, the risk of bladder cancer from occupational exposures may be increased among smokers (ACS 2006b).

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

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS 2006b, 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).

References

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

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

Cantor KP, Lynch CF, Hildesheim ME, et al. 1998. Drinking water source and chlorination by-products I. Risk of bladder cancer. Epidemiology 9(1):21-28.

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

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

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

Hodgkin's disease

Hodgkin's disease (or Hodgkin's lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin's lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,800 new cases of this disease in the U.S. in 2006, accounting for less than 1% of all cancer types, and approximately 1,490 deaths (ACS 2006). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS 2006).

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women and more common among whites than blacks. People of Jewish descent appear to be at higher risk of Hodgkin's disease compared to people of non-Jewish descent (Mueller 1996). Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 to 40 (typically ages 25-30) and the second peak occurs in adults aged 55 years and above (ACS 2006).

Scientists have identified a few risk factors that may make a person more likely to develop Hodgkin's disease. The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. A four times higher rate of Hodgkin's disease has been observed in individuals who have had infectious mononucleosis, an infection that is caused by the Epstein-Barr virus (EBV). The virus is present in the lymph nodes of approximately half of the individuals diagnosed with Hodgkin's disease the other half have no evidence of EBV in their Hodgkin cells (ACS 2006). The absence of EBV infection in about half the cases and the high prevalence of EBV in the general population suggest that EBV may be only one of several factors in the development of this cancer. Although cytomegalovirus (CMV) and the more recently identified human herpesvirus type 6 have been considered as possible factors in the development of Hodgkin's disease, results of antibody studies are inconsistent; these viruses do not appear to be related to the risk of Hodgkin's disease (Mueller 1996).

Slightly higher rates of Hodgkin's disease occur among people with reduced immunity, such as those with AIDS, people with congenital immune deficiencies, and individuals on immunosuppressant medication following organ transplants. However, Hodgkin's disease occurs at a much lower rate than non-Hodgkin's lymphomas among this group of individuals (ACS 2006).

Hodgkin's disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin's disease is associated with an infectious agent (Mueller 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller 1996). Based on an examination of medical and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin's disease does not seem to be caused by lifestyle (e.g., dietary), or environmental factors (ACS 2006).

References

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

Mueller, Nancy E. 1996. Hodgkin's Disease. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press: 1996.

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 2006a). The American Cancer Society estimates that there will be approximately 38,890 cases of kidney and upper urinary tract cancer, resulting in more than 12,840 deaths in 2006 (ACS 2006a). Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in individuals between 55 and 84 years of age (ACS 2006a). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

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

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

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

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al. 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS 2006a, 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 2006a, Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS 2006a). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not

identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al. 1997; McLaughlin et al. 1996).

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

References

American Cancer Society. 2006a. Kidney Cancer (Adult) – Renal Cell Carcinoma. Available at: http://www.cancer.org.

American Cancer Society. 2006b. Wilms' Tumor. Available at: http://www.cancer.org.

American Cancer Society. 2000. High Blood Pressure, Obesity Linked to Rise in Kidney Cancers. Available at: http://www.cancer.org.

Chow WH, Devesa SS, Waren JL, Fraumeni JF Jr. 1999. Rising incidence of renal cell cancer in the United States. JAMA 281(17):1628-31.

Linehan WM, Shipley WU, Parkinson DR. 1997. Cancer of the Kidney and Ureter. In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia. P. 1271-1297.

McLaughlin JK, Blot WJ, Devesa SS, Fraumeni JF. 1996. Renal Cancer. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press. P. 1142-1155.

Yuan JM, Castelao JE, Gago-Dominguez M, Yu MC, Ross RK. 1998. Tobacco use in relation to renal cell carcinoma. Cancer Epidemiol Biomarkers Prev 1998; 7: 429-433.

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 1997, Kinlen and Balkwill 2001, Rosenbaum et al. 2000).

Although AML can occur in children (usually during the first 2 years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS 2000a, 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, ACS 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, 2000b). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al. 2001).

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

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

References

American Cancer Society. 2005. Cancer Facts & Figures 2005. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2000a. Leukemia – Adult Acute. Available at: http://www3.cancer.org/cancerinfo/.

American Cancer Society. 2000b. Leukemia – Children's. Available at: http://www3.cancer.org/cancerinfo/.

American Cancer Society. 1999. Leukemia – Adult Chronic. Available at: http://www3.cancer.org/cancerinfo/.

Deisseroth AB, Kantarjian H, Andreeff M, Talpaz M, Keating MJ, Khouri I, Champlin RB. 1997. Chronic leukemias. In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia. P. 1271-1297.

Greaves MF. 1997. Aetiology of acute leukaemia. Lancet 349:344-9.

Infante-Rivard C, Labuda D, Krajinovic M, Sinnett D. 1999. Risk of childhood leukemia: associated with exposure to pesticides and with gene polymorphisms. Epidemiology 10:481-7.

Kinlen LJ, Balkwill A. 2001. Infective cause of childhood leukaemia and wartime population mixing in Orkney and Shetland, UK. Lancet 357:858.

Kleinerman RA, Kaune WT, Hatch EE, Wacholder S, Linet MS, Robison LL, Niwa S, Tarone RE. 2000. Are children living near high-voltage power lines at increased risk of acute lymphoblastic leukemia? Am J Epidemiol 151(5):512-5.

Linet MS, Cartwright RA. 1996. The Leukemias. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press: 1996.

Minder CE, Pfluger DH. 2001. Leukemia, brain tumors, and exposure to extremely low frequency electromagnetic fields in Swiss railway employees. Am J Epidemiol 153(9):825-35.

Rosenbaum PF, Buck GM, Brecher ML. 2000. Early child-care and preschool experiences and the risk of childhood acute lymphoblastic leukemia. Am J Epidemiol 152(12):1136-44.

Scheinberg DA, Maslak P, Weiss M. Acute leukemias. 1997.In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia. P. 1271-1297.

Schuz J, Grigat JP, Brinkmann K, Michaelis J. 2001. Residential magnetic fields as a risk factor for childhood acute leukaemia: results from a German population-based case-control study. Int J Cancer 91(5):728-35.

Smith MT, Wang Y, Kane E, Rollinson S, Wiemels JL, Roman E, Roddam P, Cartwright R, Morgan G. 2001. Low NAD(P)H:quinone oxidoreductase 1 activity is associated with increased risk of acute leukemia in adults. Blood 97(5):1422-6.

Weinstein HJ, Tarbell NJ. 1997. Leukemias and lymphomas of childhood. In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia. P. 1271-1297.

Liver Cancer

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

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

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

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

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that is the result of scar tissue formation on the liver, which can lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al. 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS 2006b). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS 2006b). 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 2006b, London and McGlynn 1996). These chemicals may also increase the risk of cholangiocarcinoma and HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS 2006b). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS 2006b, 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 2006b, London and McGlynn 1996). Long-term anabolic steroid use may slightly increase the risk of HCC (ACS 2006b). 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).

References

Agency for Toxic Substance and Disease Registry. 2001. ToxFAQs: Arsenic. U.S. Department of Health and Human Services, CAS #7440-38-2.

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

American Cancer Society. 2006b. Detailed Guide: Liver Cancer. Available at: http://www.cancer.org. Cited April 18, 2006.

El-Serag HB. 2001. Epidemiology of hepatocellular carcinoma. Clin Live Dis 5(1):87-107.

El-Serag HB, Mason AC. 2000. Risk factors for the rising rates of primary liver cancer in the United States. Arch Intern Med 160(21):3227-30.

Fracanzani AL, Conte D, Fraquelli M, Taioli E, Mattioli M, Losco A, Fargion S. 2001. Increased cancer risk in a cohort of 230 patients with hereditary hemochromatosis in comparison to matched control patients with non-iron-related chronic liver disease. Hepatology 33(3):647-51.

Garr BI, Flickinger JC, Lotze MT. 1997. Cancer of the Liver. In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia P. 1271-1297.

Ince N, Wands JR. 1999. The increasing incidence of hepatocellular carcinoma. NEJM 340(10):789-9.

London WT, McGlynn KA. 1996. Liver cancer. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press: 1996.

Mizoue T, Tokui N, Nishisaka K, Nishisaka S, Ogimoto I, Ikeda M, Yoshimura T. 2000. Prospective study on the relation of cigarette smoking with cancer of the liver and stomach in an endemic region. Int J Epidemiol 29(2):232-7.

Wogan GN. 2000. Impacts of chemicals on liver cancer risk. Semin Cancer Biol. 10(3):201-10.

Yu MC, Yuan JM, Govindarajan S, Ross RK. 2000. Epidemiology of hepatocellular carcinoma. Can J Gastroenterol 14(8):703-9.

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 U.S. among women and the sixth most common cancer among men, excluding non-melanoma skin cancers (ACS 2005).

Overall, between 1973 and 1997, the incidence of NHL in the U.S. grew 81% (Garber 2001), although during the 1990s, the rate of increase appears to have stabilized (ACS 2005). In Massachusetts, the incidence of NHL increased 50% during 1982-1997 from 10.5 cases per 100,000 to 15.7 cases per 100,000 (MCR 1997, 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, 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 five cohort studies and 14 case-control studies concludes that results of epidemiological studies have been inconsistent and that smoking has not been determined to be a definitive risk factor in the development of NHL (Peach and Barnett 2000).

A 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).

References

American Cancer Society. 2005. Cancer Facts & Figures 2005. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2003. Non-Hodgkin's Lymphoma. Available at: http://www3.cancer.org/cancerinfo/.

Dalton SO, Johansen C, Mellemkjaer L, Sorensen HT, McLaughlin JK, Olsen J, and Olsen JH. 2000. Antidepressant medications and risk for cancer. Epidemiology 11(2):171-6.

Devesa SS and Fears T. 1992. Non-Hodgkin's lymphoma time trends: United States and international data. Cancer Res 52(19 Suppl.):5492s-549s.

Garber K. 2001. Lymphoma rate rise continues to baffle researchers. J Natl Cancer Inst 93(7):494-6.

Holly EA, Lele C, Bracci PM. 1998. Hair-color products and risk for non-Hodgkin's lymphoma: a population-based study in the San Francisco Bay area. Am J Public Health 88(12):1767-73.

Linet MS, McLaughlin JK, Hsing AW, Wacholder S, Co Chien HT, Schuman LM, et al. 1992. Is cigarette smoking a risk factor for non-Hodgkin's lymphoma or multiple myeloma? Results from the Lutheran Brotherhood cohort study. Leuk Res 16(6-7):621-624.

Massachusetts Cancer Registry. 1997. Cancer Incidence and Mortality in Massachusetts 1987-1994: Statewide Report. August 1997. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

Massachusetts Cancer Registry. 2000. Cancer Incidence and Mortality in Massachusetts 1993-1997: Statewide Report. March 2000. Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation, Massachusetts Cancer Registry. Boston, MA.

Peach HG and Barnett NE. 2001. Critical review of epidemiological studies of the association between smoking and non-Hodgkin's lymphoma. Hematol Oncol 19(2):67-80.

Scherr PA and Mueller NE. Non-Hodgkin's Lymphomas. 1996. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

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

Ward MH, Mark SD, Cantor KP, Weisenburger DD, Correa-Villasenor A, Zahm SH. 1996. Drinking water nitrate and the risk of non-Hodgkin's lymphoma. Epidemiology 7(6):465-71.

Wigle DT, Semenciw RM, Wilkins K, Riedel D, Ritter L, Morrison HI, et al. 1990. Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma mortality and agricultural practices in Saskatchewan. J Natl Cancer Inst 82(7):575-82.

Wingo PA, Ries LAG, Rosenberg HM, Miller DS, and Edwards BK. 1998. Cancer incidence and mortality, 1973-1995: A report card for the U.S. Cancer 82(6):1197-1207.

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

Zahm SH, Weisenburger DD, Babbit PA, Saal RC, Vaught JB, Blair A. 1992. Use of hair coloring products and the risk of lymphoma, multiple myeloma, and chronic lymphocytic leukemia. Am J Public Health 82:990-97.

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

Zahm SH, Weisenburger DD, Saal RC, Vaught JB, Babbitt PA, Blair A. 1993. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women. Archives of Environmental Health 48(5):353-8.

Pancreatic Cancer

The American Cancer Society estimates that approximately 32,180 people in the U.S. (16,100 men and 16,080 women) will develop pancreatic cancer in 2005. This disease accounts for approximately 2% of all new cases of cancer in both men and women, but between 5% and 6% of all cancer deaths (ACS 2005). This discrepancy has been attributed to detection of pancreatic cancer at an advanced stage and the short median survival time for this cancer of approximately three months. Between 1920 and 1965, mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people. These increases are believed to be due, in part, to improved diagnosis during this time period (Anderson et al. 1996). However, over the past 25 years, incidence rates have declined slowly but consistently in men and a slight decline in rates among women has been observed since the mid-1980s. Further, since about 1975, men have experienced a slight decrease in mortality from pancreatic cancer, although rates among women have not dropped (ACS 2005). The risk of developing pancreatic cancer increases with age and the majority of cases occur between age 60 and 80. Men are approximately 30% more likely to develop pancreatic cancer than are women (ACS 2000).

Very little is known about what causes pancreatic cancer and how to prevent it. However, a number of risk factors have been identified. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS 2000). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al. 1996).

Certain medical conditions, such as chronic pancreatitis, diabetes mellitus, and cirrhosis, have been associated with pancreatic cancer, but the reasons for these associations are largely unknown (ACS 2000). More recently, a possible role for the bacteria Helicobacter pylori, which causes ulcers and some gastric cancers, has been suggested in the development of pancreatic cancer (Stolzenberg-Solomon et al. 2001).

There is also some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer may be associated with animal protein and fat consumption as evidenced by higher rates of this cancer in countries whose populations eat a diet high in fat (ACS 2005). Decreased risks for the disease are usually associated with fruit and vegetable consumption (ACS 2000). Obesity is also a risk factor for pancreatic cancer (ACS 2000). Although older studies suggested that coffee and alcohol consumption may be risk factors, more recent studies do not support this association (Michaud et al. 2001).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS 2000, Ji et al. 2001, Porta et al. 1999). Exposure to certain dyes and certain chemicals related to gasoline, in addition to asbestos and ionizing radiation, have also been associated with the development of pancreatic cancer in some studies; however, other studies have found no link between these agents and pancreatic cancer (ACS 2000, Anderson et al. 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to corroborate this claim (Ojajarvi et al. 2000). Although occupational exposures may have played a role in the incidence of this cancer in the past, currently most newly diagnosed patients with pancreatic cancer do not have evidence of a specific chemical exposure or relevant occupational history (Evans et al. 1997).

Finally, pancreatic cancer seems to run in some families. According to the American Cancer Society, an inherited tendency to develop pancreatic cancer may account for approximately 5% to 10% of cases (ACS

2000). Pancreatic cancer has been observed in both familial clusterings among siblings as well as in individuals of consecutive generations (Anderson et al. 1996).

References

American Cancer Society. 2005. Cancer Facts & Figures 2005. Atlanta: American Cancer Society, Inc.

American Cancer Society. 2000. Pancreas Cancer. Available at: http://www3.cancer.org/cancerinfo/.

Anderson D, Potter J, Mack T. 1996. Pancreatic Cancer. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

Evans DB, Abbruzzese JL, Rich TA. 1997. Cancer of the Pancreas. In: Cancer: Principles and Practice of Oncology, Fifth Edition, edited by Devita V, Hellman S, Rosenberg S. Lippincott-Raven Publishers, Philadelphia. P. 1271-1297.

Ji BT, Silverman DT, Stewart PA, Blair A, Swanson GM, Baris D, Greenberg RS, Hayes RB, Brown LM, Lillemoe KD, Schoenberg JB, Pottern LM, Schwartz AG, Hoover RN. 2001. Occupational exposure to pesticides and pancreatic cancer. Am J Ind Med 39(1):92-9.

Michaud DS, Giovannucci E, Willett WC, Colditz GA, Fuchs CS. 2001. Coffee and alcohol consumption and the risk of pancreatic cancer in two prospective United States cohorts. Cancer Epidemiol Biomarkers Prev 10(5):429-37.

Ojajarvi IA, Partanen TJ, Ahlbom A, Boffetta P, Hakulinen T, Jourenkova N, Kauppinen TP, Kogevinas M, Porta M, Vainio HU, Weiderpass E, Wesseling CH. 2000. Occupational exposures and pancreatic cancer: a meta-analysis. Occup Environ Med 57(5):316-24.

Porta M, Malats N, Jariod M, Grimalt JO, Rifa J, Carrato A, Guarner L, Salas A, Santiago-Silva M, Corominas JM, Andreu M, Real FX. 1999. Serum concentrations of organochlorine compounds and K-ras mutations in exocrine pancreatic cancer. Lancet 354:2125-29.

Stolzenberg-Solomon RZ, Blaser MJ, Limburg PJ, Perez-Perez G, Taylor PR, Virtamo J, Albanes D. 2001. Helicobacter pylori seropositivity as a risk factor for pancreatic cancer. J Natl Cancer Inst 93(12):937-41.

Appendix C

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

General Terms

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

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

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

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

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

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

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

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

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

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

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

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

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

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

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

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

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

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

CAP [see Community Assistance Panel.]

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

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

Carcinogen
A substance that causes cancer.

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

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

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

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

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

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

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

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

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

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

Completed exposure pathway [see exposure pathway].

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

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

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

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

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

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

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

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

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

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

DOD
United States Department of Defense.

DOE
United States Department of Energy.

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

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

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

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

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

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

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

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

Migration
Moving from one location to another.

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

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

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

Mutagen
A substance that causes mutations (genetic damage).

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

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

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

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

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

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

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

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

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

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

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

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

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

ppb
Parts per billion.

ppm
Parts per million.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Radionuclide
Any radioactive isotope (form) of any element.

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

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

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

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

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

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

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

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

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

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

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

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

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

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

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

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

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

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

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

Substance
A chemical.

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

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

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

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

Surveillance [see public health surveillance]

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

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

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

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

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

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

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

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

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

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

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

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

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

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