Health Consultation

Evaluation of Three State-Regulated Release Sites and Brain Cancer and Childhood Cancer Incidence in Methuen, Essex County, Massachusetts

MDEP RTN 3-3661, 3-24029, 3-25262 (4 Gleason Street), 3-10447 (54 Osgood Street) and 3-20237 (254 Broadway)

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

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In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation 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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3-10447 (54 Osgood Street) and 3-20237 (254 Broadway)

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At the request of concerned residents, the Community Assessment Program (CAP) at the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of three state-regulated former industrial sites and cancer incidence for the city of Methuen. This evaluation was initiated because of community concerns about a suspected increase in the overall incidence of brain cancer and childhood cancer (individuals aged 19 and under at diagnosis) among individuals living near three former industrial sites at 4 Gleason Street, 254 Broadway, and 54 Osgood Street in central Methuen. Two of the sites, 4 Gleason Street and 254 Broadway, were industrial sites in central Methuen dating back to the mid-1800s. The third site, 54 Osgood Street, was the scene of a fire that destroyed a building housing hazardous material.

Environmental contamination associated with the three sites in central Methuen of concern to some residents was evaluated for the potential of exposure to nearby residents and subsequent health risks. The evaluation found the opportunities for exposure to contaminants to be limited. The groundwater in this area was not and is not used as a source of drinking water. No increased risks of cancer or other adverse health effects were predicted assuming a nearby resident regularly came into contact with the maximum detected concentrations of contaminants in the soil, sediment or surface water at the three sites. The three sites have been remediated, are currently vacant, and access to them is limited.

In response to community concerns, the CAP evaluated the incidence of brain cancer and childhood cancer in the city of Methuen and compared it to the incidence in the state of Massachusetts as a whole. To evaluate patterns or trends over time, cancer incidence rates were calculated over three time periods: 1982-1988, 1989-1995, and 1996-2002. Community members also expressed concern about different types of non-cancer illnesses among Methuen residents. Along with the initial written request for a review of cancer incidence, the CAP was provided with 11 surveys that had been completed by

residents of the Gleason Street neighborhood in Methuen. The CAP reviewed hospital discharge data on the number of asthma-related hospitalizations in the city of Methuen in response to concerns about respiratory impacts from the three sites. New data on pediatric asthma that have become available through an environmental public health tracking initiative at the BEH were also utilized to respond to resident concerns.

At the citywide level, the incidence of brain cancer was elevated for three time periods (1982-1988, 1989-1995 and 1996-2002). None of the elevations were statistically significant, meaning that chance or random fluctuation cannot be ruled out as a reason for the increased incidence. At the census tract (CT) level, no single CT had a consistently elevated incidence of brain cancer over the three time periods that was statistically significant. For all three time periods, the incidence of childhood cancer occurred about as expected in the city of Methuen with one exception. During 1996-2002, four children were diagnosed with cancer in CT 2523 in central Methuen when approximately one would be expected. Three of the four children were diagnosed with brain cancer. Their ages at diagnosis and cancer subtypes were consistent with what would be expected. One additional child has been diagnosed with brain cancer in this CT since 2003.

In addition to evaluating time trends, the geographic distribution of residence at diagnosis for those individuals diagnosed with brain cancer or childhood cancer in Methuen was evaluated to determine if any atypical spatial patterns existed. With one exception, the review of the geographic distribution of cancer for the years 1982–2002 did not reveal any unusual spatial patterns or concentrations of diagnoses at the neighborhood level. During the 1996-2002 time period, two of the three children diagnosed with brain cancer lived in close proximity to each other. As discussed in Section IV, the opportunity for exposure to environmental contaminants was determined to be limited. No increased risk of cancer or other adverse effects was likely even if a nearby resident was exposed regularly to the maximum detected concentrations of soil, sediment, or surface water contaminants. Analysis of risk factor information (for example, age, gender, smoking history, and occupation) for individuals diagnosed with brain cancer suggested that the trends observed in Methuen are similar to those seen in the general population. For these

reasons, it does not appear that a common factor (environmental or non-environmental) is related to these brain cancer diagnoses.

Based on criteria established by ATSDR, the 54 Osgood Street site is classified as posing no apparent public health hazard to the public in the past, present and future. Since contaminants detected in groundwater at the 4 Gleason Street and 254 Broadway sites could be drawn into potential future private wells, these sites would pose a public health hazard in the future should wells be installed in contaminated groundwater areas.

II. Introduction

At the request of concerned residents, the Community Assessment Program (CAP) at the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of three state-regulated former industrial sites and cancer incidence for the city of Methuen. This evaluation was initiated because of community concerns about a suspected increase in the overall incidence of brain cancer and childhood cancer (individuals aged 19 and under at diagnosis) among individuals living in the area of three former industrial sites at 4 Gleason Street, 254 Broadway, and 54 Osgood Street in central Methuen (see Figure 1). Two of the sites, 4 Gleason Street and 254 Broadway, were industrial sites in central Methuen dating back to the mid-1800s. The third site, 54 Osgood Street, was the scene of a fire that destroyed a building housing hazardous material. In order to address these community concerns, the MDPH reviewed available environmental data for the three sites and considered potential ways that people may come into contact with contaminants detected in groundwater, soil, surface water, and sediment located on or near the sites.

This investigation also reviewed the incidence of brain cancer and childhood cancer in the city of Methuen and compared it to the incidence in the state of Massachusetts as a whole. Additionally, available information about risk factors, including environmental factors, related to the development of these cancers was evaluated. The city of Methuen is divided into 11 smaller geographic areas, or census tracts. Cancer incidence rates were also evaluated for each census tract with a particular focus on CT 2523, where the three

former industrial sites of concern are located, and the two adjacent CTs of 2524 and 2525.02 (see Figure 2). Cancer incidence data for Methuen were obtained from the Massachusetts Cancer Registry (MCR). At the time of this evaluation, the most recent and complete cancer incidence data available from the MCR were through the year 2002. Three time periods (1982-1988, 1989-1995 and 1996-2002) were examined to assess possible trends over time.

In addition to the calculation of cancer incidence rates, a qualitative analysis of the geographic distribution of residents of Methuen diagnosed with brain cancer (both adults and children) or childhood cancer was conducted by mapping their residence at the time of diagnosis. This was done to determine if any spatial patterns existed in a particular area of the city or in relation to the three sites at 4 Gleason Street, 254 Broadway, and 54 Osgood Street.

Finally, community members also expressed concern about different types of non-cancer illnesses among Methuen residents. Along with the initial written request for a review of cancer incidence, the CAP was provided with 11 surveys that had been completed by residents of the Gleason Street neighborhood in Methuen. The survey, designed by a resident of Methuen, asked individuals to report on any illnesses they had been diagnosed with, along with other personal and residential history information. For that reason, this report also contains a qualitative review of the information contained in these surveys. In addition, the CAP also reviewed hospital discharge data on the number of asthma-related hospitalizations in the city of Methuen in response to concerns about respiratory impacts from the three sites of concern. These data are collected by the Massachusetts Division of Health Care Finance & Policy and are available through the Massachusetts Community Health Information Profile (Mass CHIP). New data that have become available through an environmental public health tracking initiative at the BEH were also utilized.

III. Objectives

The specific objectives of this investigation were as follows:

- To review the incidence of brain cancer among all ages and childhood cancer in the city of Methuen and at the smaller geographic level of the census tract, with a particular focus on central Methuen.

- To evaluate the geographic distribution of individuals diagnosed with these cancer types in Methuen, to see if there are any patterns in particular areas of the town or in the areas of potential environmental concern.

- To review descriptive information available from the MCR for individuals diagnosed with brain cancer and childhood cancer in Methuen, to see if there are any particular characteristics related to known or suspected risk factors, including environmental factors, for developing these diseases.

- To discuss possible exposures related to three former industrial sites in the context of the available scientific and medical literature on cancer and contaminants of concern to determine whether further investigation or public health action is warranted.

- To assess the number of asthma-related hospitalizations in the city of Methuen.

- To review and evaluate 11 surveys provided to the CAP by residents of Methuen who had reportedly been diagnosed with various immunological diseases.

IV. Community Environmental Concerns

Introduction

Some members of the Methuen community have expressed concerns about odors and possible contamination of the soil and groundwater in the area of three former industrial or manufacturing sites located in the center of Methuen: 4 Gleason Street, 254 Broadway, and 54 Osgood Street. In order to address these community concerns, the MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) as well as

the United States Environmental Protection Agency (EPA) to obtain and review available environmental information pertaining to these sites. Environmental sampling data were available for groundwater, soil, surface water, and sediment located on or in the vicinity of the sites. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify those substances that may need to be considered for further analysis, to determine whether they may be of potential health concern to residents. The screening analysis identified maximum concentrations of contaminants detected in various types of environmental media (i.e., soil and water) and compared these concentrations to health-based comparison values. Comparison values are set well below levels that are known or anticipated to result in adverse health effects. Contaminant concentrations that exceed comparison values will not necessarily affect one's health. If a comparison value was not available for a specific contaminant, the maximum detected concentration was compared to Risk-Based Concentrations (RBCs) developed by the United States EPA Region III (USEPA 2006a), Preliminary Remediation Goals (PRGs) developed by the United States EPA Region 9 (USEPA 2004), or the applicable groundwater and soil standards developed by MDEP (MDEP 2006a), in that order. For contaminants detected in groundwater, maximum concentrations were also compared with state and/or federal drinking water standards. An evaluation of potential exposure pathways was conducted to determine whether contamination identified at the industrial sites in the Gleason Street neighborhood could be impacting the health of nearby residents of Methuen in the past, present, or future.

For each site discussed below, background information on the history of the site is provided followed by discussion of the status of each site within MDEP's site investigation and cleanup process. An evaluation of the potential for exposure of Methuen residents to site-related contamination is provided, and finally, the available environmental data are compared to health-based screening values to determine whether there may be potential health impacts to residents.

A. 4 Gleason Street

Background

The property located at 4 Gleason Street, Methuen, is approximately 3.7 acres in size and is located on a dead end street that runs east/west between Broadway and the Spicket River in central Methuen (see Figure 1). The parcel of land currently consists of a building footprint where the building was razed, a paved area surrounding the building footprint, with the remainder of the lot being unpaved land that is adjacent to the Spicket River. The building was recently demolished and portions of the property have remained fenced off from the public since demolition (LFR 2003).

The site was used by various industries since the mid-19th century. Historical use of the property included a textile mill, wet wash laundry, semi-conductor manufacturing and manufacturing of metal cabinets for computers. Industrial activities at this site ceased in the late 1990s (LFR 2003). After being unused for several years, current plans reportedly include redevelopment of the property (along with the adjacent property at 254 Broadway) into assisted living apartments for the elderly. The site was excavated and cleaned up in preparation for construction in 2005.

MDEP 21E Releases

In 1983, the Massachusetts Legislature established a statewide hazardous waste site cleanup program (the state equivalent of the federal Superfund program) under Chapter 21E of Massachusetts General Laws (M.G.L. c21E, 310 CRM 40.0000). Under this legislation, the MDEP enforces regulations governing the investigation and cleanup of hazardous material and oil release sites, known as "21E sites," in the Commonwealth.

The 21E sites are characterized by one or more releases of oil or other hazardous material. Releases can result from a variety of sources, including trucks and other vehicles, underground storage tanks, and aboveground storage drums. Releases vary widely with respect to materials involved, the relative amount of materials released, and the geographic extent of contamination. Depending on the relative severity of the release, the deadline for reporting a release to MDEP is 2 hours, 72 hours, or 120 days.

The MDEP Bureau of Waste Site Cleanup has information on these hazardous material and oil releases, including assessment and remedial response measures, beginning in 1977 to the present; however, records prior to 1984 are known to contain significant data gaps (MDEP 2006b). MDPH obtained the most recent information regarding all hazardous material and/or oil releases located at 4 Gleason Street, Methuen. The information indicates that three known releases were reported at the site. Releases of oil were reported on July 15, 1991, and September 22, 2005. A release of oil and hazardous material was reported on July 6, 2004. Release abatement measures were conducted in response to the 1991 release and a completion report was submitted to MDEP. In 2005, during the removal of sewer lines at the 4 Gleason Street site, petroleum-impacted soils were discovered and required MDEP notification (LFR 2006a). These contaminated soils were likely linked to an underground storage tank (UST) containing heating oil that was discovered following detection of the petroleum-impacted soils (LFR 2006a). An Immediate Response Action (IRA) was taken and the UST, as well as contaminated soil around the tank, were excavated, removed from the site, and disposed of at proper facilities (LFR 2006a).

Releases in 2004 and 2005 have been classified as having a Class A-2 Response Action Outcome (RAO), indicating that remedial work was completed, a permanent solution has been achieved and, although contamination has not yet been reduced to background levels, a level of "no significant risk" of harm to health, safety, public welfare and the environment has been achieved (MDEP 1995a, MDEP 2006c).

Environmental Data and Exposure Pathway Analysis

To address community concerns about possible environmental exposures associated with the property at 4 Gleason Street, MDPH reviewed information on file with MDEP and EPA. The MDEP has recently reviewed and analyzed the data from the numerous environmental investigations that have been conducted by different Licensed Site Professionals (LSPs). Sampling data have been produced by Clean Harbors Environmental (CHE) (1986-1993), TRC (2000) and LFR Levine-Fricke (2002-present),

from their work conducted at the site (CHE 1995, TRC 2000, LFR 2005, LFR 2006a). Environmental sampling data were available for groundwater, soil, surface water, and sediment located on or in the vicinity of the site. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify those substances that may need to be considered for further analysis to determine whether they may have the potential to impact the health of residents.

Hazardous material and oil releases are *potential* sources of exposure to contamination. It is not possible to determine whether individuals residing in the 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. However, the pattern of cancer in Methuen residents was reviewed in relation to these *potential* sources of environmental exposures and is discussed in Section V.

Groundwater

According to MASSGIS, a web-based geographical information system, there are no community drinking water wells or MDEP Zone II protection areas within 1 mile of the site. A Zone II protection area is defined as the area of an aquifer which contributes water to a well under the most severe pumping recharge conditions that can be realistically anticipated (MDEP 1995a). The nearest medium yield, potentially productive aquifer is approximately ¾ of a mile northwest of the site, while the nearest high yield, potentially productive aquifer is located over 1 mile northwest of the site. The nearest Interim Wellhead Protection Area (IWPA) is also located over 1 mile from the site. Due to these conditions, it is unlikely that groundwater in the vicinity of 4 Gleason Street could be used as a source of drinking water.

There are currently many monitoring wells on the 4 Gleason Street property that have been used to collect groundwater samples since 1986. These wells are used solely to monitor groundwater near the property and are not used for any other drinking or non-

drinking water purposes. The locations of these groundwater monitoring wells are depicted in Figure 3.

The monitoring wells around the property had detected concentrations of some volatile organic compounds (VOCs) and metals. The highest levels of contaminants measured in groundwater on the entire site were compared to health-based drinking water comparison values to help determine if further evaluation was necessary. These health-based comparison values are set well below levels that are known or anticipated to result in adverse health effects. Contaminant concentrations that exceed comparison values will not necessarily affect one's health. Of the contaminants detected in monitoring wells at the 4 Gleason Street property, fourteen required further evaluation. Table 1 summarizes the maximum concentrations of each of these contaminants as well as their comparison values.

Exposure to these contaminants in groundwater is unlikely since groundwater in the vicinity of 4 Gleason Street and surrounding industrial sites of community concern is not a source of drinking water for Methuen residents. Information reviewed indicated that there are no registered or known drinking water supply wells within a 1-mile radius of the site (CHE 1994). The City of Methuen obtains its drinking water from the Merrimac River and then treats and tests this water at the Burnham Road Water Treatment Plant to ensure that it meets state and federal standards for safe drinking water (MWD 2006).

Since there is no official moratorium in place to restrict the installation of new private wells in the city of Methuen, future exposure to contaminants identified at 4 Gleason Street is possible if new private drinking water wells are installed in the path of contaminated groundwater, which flows in an easterly and southeasterly direction (CHE 1995). If nearby residents were to ingest contaminated groundwater in the future at concentrations detected in onsite monitoring wells, non-cancer and cancer health impacts could be possible due to exposure to some metals and VOCs.

Soil

Samples of soils and sediment around the 4 Gleason Street property contained detectable levels of VOCs, semi-volatile organic compounds (SVOCs), polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and metals. The highest levels measured in soils on the entire site were compared to health-based comparison values to help determine if further evaluation is necessary. As with screening values for groundwater, contaminant concentrations that exceed comparison values in soils will not necessarily affect one's health. A majority of contaminants were below health-based comparison values and are not expected to result in adverse health impacts. However, nine contaminants, including one VOC, one petroleum hydrocarbon fraction, some PAHs and some metals, required further evaluation. It is important to note that the maximum concentrations of these nine contaminants were measured during sampling events in 1992-1993 and 2000 and all are located in areas that were excavated in 2005. Table 2 summarizes the maximum concentrations of each of these chemicals as well as their comparison values.

Prior to excavation and cleanup in 2005, it is possible that an older child playing onsite at 4 Gleason Street could have been exposed through incidental ingestion of or dermal contact with contaminants such as trichloroethene, benzo(a)pyrene, arsenic, and chromium detected in onsite surface soil at levels above comparison values. However, it is important to consider that comparison values are based on a residential exposure scenario, and it is unlikely that a child playing at the site infrequently would have had contact with onsite surface soil for a comparable frequency and duration of time.

Although the area sampled is a former industrial site, it is possible that an older child could have visited the site and had infrequent contact with the soil. Assuming the child inadvertently ingested 200 milligrams of surface soil containing the maximum concentration of any of the above listed contaminants detected on the site for 2 days every week for 22 weeks (May through September, the warmer months of the year) over 12 years, the exposure would not be expected to result in an increased cancer risk. Also, under the same exposure conditions described above, the level would be below the

ATSDR Minimal Risk Level (MRL). The MRL is an estimate of daily exposure to a contaminant below which non-cancer, adverse health outcomes are unlikely to occur. Since the exposure dose for an older child playing onsite is below the MRL, non-cancer health effects would also not be expected.

Present and future exposure to contaminants in soil is unlikely since areas with the highest levels of contaminants were excavated and removed in 2005. Prior to demolition, the eastern portion of the building contained a former boiler room and elevated PAH levels in soil were suspected to be from ash left from coal burning (LFR 2006a). According to ATSDR toxicological reports, PAHs can be formed during incomplete burning of coal, oil, gas, wood, garbage, or other organic substances such as tobacco and charbroiled meat. Elevated levels of PAHs can be found in the environment as a result of forest fires, residential wood burning and exhaust from automobiles or trucks (ATSDR 1995). Following excavation, confirmatory soil sampling indicated that a majority of the elevated PAH concentrations in the area were reduced to background levels or were not detected. Chromium levels also decreased to a range of 11-33 parts per million (ppm) following excavation and were both below comparison values and within the range of typical soil background levels for chromium (1-1,000 ppm) (USGS 1984). Arsenic levels, too, were brought within the range of typical soil background levels. Areas containing high levels of trichloroethene were also excavated and levels were significantly reduced (LFR 2006a). All excavated soil was temporarily stored on the property while awaiting removal and disposal at disposal/reuse facilities (LFR 2006a).

Surface Water

The largest surface water body bordering the site is the Spicket River, which runs along the perimeter of the 4 Gleason Street property to the west and flows in a southeasterly direction until it joins the Merrimac River. Surface water from the Spicket River was sampled by CHE in 1991-1992 and some metals were detected (MDEP 2005). The highest levels measured in surface water were compared to drinking water comparison values to help determine if further evaluation was necessary. Because health-based comparison values for *surface water* do not exist, drinking water comparison values were

used as screening values. Comparison values are set well below levels that are known or anticipated to result in adverse health effects. Additionally, the use of drinking water comparison values to screen surface water is conservative (i.e., health-protective) because drinking water values assume that an individual ingests 2 liters per day of the water. Since it is unlikely that an individual would ingest 2 liters of surface water each day, exposure to contaminants found in surface water is considerably less than exposure to contaminants found in drinking water. Comparison values do not predict adverse health effects and contaminants concentrations that exceed comparison values will not necessarily affect one's health.

Of the contaminants detected in surface water near 4 Gleason Street, only cadmium and lead exceeded the drinking water comparison values. Individuals living near the 4 Gleason Street property would likely be exposed less frequently to contaminated surface water in the Spicket River through incidental ingestion and dermal contact than to drinking water. For example, while cadmium and lead were detected above the drinking water comparison value, calculations made using assumptions specific to surface water ingestion show that this concentration is not expected to produce adverse health effects in adult or child residents playing near the Spicket River.

Exposure to these contaminants for residents in the surrounding neighborhoods would be very limited. Water from the Spicket River is not incorporated into the drinking water for Methuen and residents would have limited exposure to the river. The location where the Spicket River joins the Merrimac River is approximately 3 miles downstream from where the city of Methuen obtains its drinking water (MWD 2005). Therefore, residents of Methuen would not be consuming water from the Spicket River as part of their drinking water.

Sediment

Spicket River sediment along the edge of the 4 Gleason Street property was sampled in 2000 by TRC. The highest levels measured in the sediment were compared to health-based comparison values to help determine if further evaluation is necessary. Since

comparison values for sediment do not exist, soil comparison values were used as screening values. All detected contaminants were below comparison values and within the range of typical background levels of metals in soil (USGS 1984). Therefore, exposure to sediment from the Spicket River on the 4 Gleason Street property is not expected to result in adverse health effects for residents.

Site Status

As stated above, release abatement measures were conducted in response to the 1991 release and a completion report was submitted to MDEP. In addition, releases in 2004 and 2005 have been classified as having a Class A-2 RAO, indicating that remedial work was completed, a permanent solution has been achieved and, although contamination at the property has not yet been reduced to background levels, a level of "no significant risk" of harm to health, safety, public welfare and the environment has been achieved (MDEP 1995a, MDEP 2006c).

B. 254 Broadway

Background

The property located at 254 Broadway, Methuen, is approximately a quarter acre in size and is located adjacent to the 4 Gleason Street property. A former service station and garage building which once stood on the site were demolished in 2005. Currently the lot remains undeveloped and vacant. Properties surrounding 254 Broadway consist of mixed retail and commercial office spaces.

From the 1880s until the early 1930s, this property housed textile manufacturing facilities, including a knitted fabric company, a hat manufacturer, and a worsted good manufacturer. In the mid-1930s, the property was converted to a gas station/automobile repair shop. For the next several decades the ownership changed hands numerous times, although the fundamental business as a gas station/automobile repair shop remained constant (LFR 2004). In 1986, the Methuen Fire Department asked the owner at the time to remove underground storage tanks (USTs) from the property that were no longer in

use. The USTs were removed from the ground, and from that time forward, the business was strictly an automobile repair shop (Enstrat 1998).

MDEP 21E Releases

The MDEP Bureau of Waste Site Cleanup has information on hazardous material and oil releases, including assessment and remedial response measures, for 1977 to the present; however as mentioned, records prior to 1984 are known to contain significant data gaps (MDEP 2006b). MDPH obtained the most recent information regarding all hazardous material and/or oil releases located at 254 Broadway, Methuen. The only known release reported at the site was discovered in 2000 as a result of environmental testing and assessment being conducted by TRC on behalf of a new property owner (LFR 2006b). Environmental testing conducted during UST system removal and in conjunction with testing done by TRC at the adjacent 4 Gleason Street site showed elevated levels of volatile petroleum hydrocarbons (VPHs) at the site. Under Chapter 21E of Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000), this release required reporting to the MDEP within 120 days. The 120-day reports are releases thought unlikely to result in human exposure to contaminants. The release was likely related to the UST tank system that existed during historical gas station and auto repair shop activities and information reviewed indicates that no other USTs are known to be present onsite (LFR 2006b).

Environmental Data and Exposure Pathway Analysis

To address concerns about possible environmental exposures associated with the property at 254 Broadway, MDPH reviewed information on file with MDEP and EPA. Environmental sampling data were available for water and soils located onsite. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify those substances that may need to be considered for further analysis to determine whether they may be of potential health concern to residents. As mentioned, the screening analysis identifies maximum concentrations of contaminants detected in various types of environmental media (i.e., soil and water) and compares these concentrations to health-based comparison values. Comparison values are set well below

levels that are known or anticipated to result in adverse health effects. Contaminant concentrations that exceed comparison values will not necessarily affect one's health.

The most recent groundwater monitoring data available were obtained during sampling events in 2002 (August) and 2005 (February, April, November). Groundwater from seven monitoring wells located throughout the site was analyzed for metals, VOCs, and petroleum hydrocarbon fractions. Metals were not detected onsite, however some VOCs and petroleum hydrocarbon fractions were detected at elevated levels.

Of all the contaminants detected in groundwater at 254 Broadway, four chemicals exceeded health-based comparison values and therefore required further evaluation in this report. These four chemicals are C5-C8 Aliphatics Hydrocarbons, C9-C10 Aromatic Hydrocarbons, toluene, and naphthalene. The maximum concentration of each of these four chemicals was detected at monitoring well MW-3, which is located within the area of the former UST. Table 3 summarizes the maximum concentrations of each of these chemicals as well as their comparison values.

Present exposure to these contaminants in groundwater is unlikely since groundwater in the vicinity of 254 Broadway and surrounding industrial sites of community concern is not a source of drinking water for Methuen residents. The groundwater wells sampled at 254 Broadway are for monitoring purposes only, these wells are not used for any other drinking or non-drinking water purposes. Currently, the ingestion of groundwater is not a potential pathway because there are no public or private drinking water supply wells within a 1- mile radius (CHE 1994). As previously discussed, the City of Methuen obtains its drinking water from the Merrimac River and then treats and tests this water at the Burnham Road Water Treatment Plant to ensure that it meets state and federal standards for safe drinking water (MWD 2006).

Exposure to contaminants identified at 254 Broadway could be possible if new private drinking water wells are installed in the path of contaminated groundwater, which flows south toward the Spicket River (LFR 2006b). If nearby residents were to ingest

contaminated groundwater in the future at concentrations detected in onsite monitoring wells, non-cancer health impacts are possible due to exposure to some petroleum hydrocarbon fractions and toluene.

Soil monitoring data were also available for the 254 Broadway property. Soil sampled during 2002 indicated elevated levels of some contaminants found in the area of the former USTs and gas pump island, which is expected given the background of the property and location of USTs (Enstrat 1998, TRC 2001, LFR 2006b). In August and September 2005, the contaminated soil was excavated from the former UST area (LFR 2006b). The soil was excavated to a depth of approximately 8 to 12 feet below ground surface and a total of 113 cubic yards of soil was removed. However, confirmatory sampling indicated that contamination remained in a small area. Soil was again excavated to a depth of approximately 10 to 12 feet, and an additional 44 cubic yards was removed from the site. Additional soil sampling was conducted following this excavation and showed that prior detected contamination had been removed. The limited nature of the contamination and particularly the subsurface location, a depth of over 10 feet below the ground surface, would make it unlikely for nearby residents to have been exposed to the contaminants in the past. Remediation activities conducted in 2005 eliminate present and future opportunities for nearby residents to be exposed to these contaminants. Therefore, soil on the 254 Broadway site is not expected to result in increased cancer risks nor increased non-cancer health impacts to residents in the surrounding neighborhood.

Site Status

On June 14, 2006, LFR submitted a Release Abatement Measure (RAM) Completion Report and Class A-2 RAO for the property located at 254 Broadway (LFR 2006b). A Class A-2 RAO indicates that remedial work was completed, a permanent solution has been achieved and, although contamination has not yet been reduced to background levels, a level of “no significant risk” has been achieved (MDEP 1995a, MDEP 2006c).

C. 54 Osgood Street

Background

The property located at 54 Osgood Street is near the intersection of River Street and Osgood Street in central Methuen (see Figure 1). Prior to a fire in January 1994, the site housed a metal plating business that operated for approximately 20 years (MDEP 1995b). The site is currently vacant and is bordered by commercial properties such as a restaurant and an office building.

As stated above, the MDEP Bureau of Waste Site Cleanup has information on hazardous material and oil releases, including assessment and remedial response measures, for 1977 to the present; however, records prior to 1984 are known to contain significant data gaps (MDEP 2006b). MDPH obtained the most recent information regarding all hazardous material and/or oil releases located at 54 Osgood Street, Methuen. The only known release reported at the site was reported on January 15, 1994 and coincides with a fire that destroyed the building. Prior to the fire, metal plating operations had ceased at the site, and approximately 49 55-gallon drums of hazardous materials were stored in the building (Weston 1994). The site inventory of hazardous materials present at the time of the fire is attached as Appendix A. Under Chapter 21E of Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000), this release required reporting to the MDEP within 2 hours, upon which time immediate action was taken. The Methuen Fire Department (MFD) let the fire burn until an MDEP response team could assess the possible environmental impacts of the fire and any subsequent firefighting activities. Upon arrival at the site, the response team assessed the potential environmental impacts from the fire. Two conditions were of concern: the water runoff from firefighting activities and the danger of the smoke plume emanating from the fire. The response team determined that sand dikes were needed to immediately contain the runoff. The contained runoff was then collected in vacuum trucks to prevent accumulation and the possible leaching of contaminants into the surrounding soil or water (the Spicket River is within a ¼ mile of the 54 Osgood Street property). The runoff was shipped to a licensed disposal site for appropriate disposal. Once the plans for collection of the runoff were in place, the MFD began to extinguish the fire (Weston 1994).

With the runoff water contained, the apparent immediate environmental impact from this fire was potential impacts to residents from the smoke plume created by the fire. Air monitoring was conducted initially by the Massachusetts District 6 Haverhill Hazardous Materials Team (HMT), who determined that hydrogen sulfide, hydrogen cyanide, sulfur dioxide, and benzene were not present in the smoke plume (Weston 1994). Air monitoring was then conducted by members of the MDEP response team to further assess contaminants in the smoke plume (See next section for further discussion).

Once the immediate response to the fire was complete, the MDEP sought to ensure that the site was neither a short-term nor a long-term hazard to the residents of Methuen. Immediately after the fire, an 8-foot chain-link fence was constructed to limit access to the property and any potential onsite contamination. Security was also provided to prevent site access during the night. During clean-up activities, the building debris was tested for contamination. Any contaminated material was disposed of accordingly and regular waste was removed from the site as well (MDEP 1995b).

After completing the removal of the building debris, testing was conducted on the site and surrounding properties, to determine the need for further remediation work on the site. Soil sampling performed by the MDEP did not reveal any environmental impact to the soil in the area. In early 1995, the immediate response action was considered complete and no further action was deemed necessary (MDEP 1995c).

Environmental Data and Exposure Pathway Analysis

To address concerns about possible environmental exposures associated with the property at 54 Osgood Street, MDPH reviewed and analyzed information on file with MDEP. Environmental sampling data were available for runoff water that resulted from firefighting activities as well as for air and soils located onsite. Available environmental sampling data for the runoff water and air were reviewed and evaluated qualitatively. Environmental sampling data for soil were reviewed, and a screening evaluation was

conducted to identify those substances that may need to be considered for further analysis to determine whether they may be of potential health concern to residents.

Initial air monitoring data performed by the HMT were not available, but discussion in Weston (1994) indicated that hydrogen sulfide, hydrogen cyanide, sulfur dioxide, and benzene were not detected in the smoke plume at 54 Osgood Street. Additional air monitoring data showed that levels of radiation as well as hydrogen cyanide, hydrogen sulfide, and chlorine were not detected during MDEP response team air monitoring (Weston 1994). In addition, the data show that while contaminant levels in air close to the building were elevated, levels as close as 50 feet downwind of the building were similar to background levels (Weston 1994).

Soil sampling was conducted on December 1, 1994, to determine if contaminants from the site had impacted nearby properties. The highest levels of contaminants measured in soil at each nearby property were compared to health-based comparison values for soil to help determine if further evaluation is necessary. Levels of contaminants such as lead, nickel, and zinc measured at nearby properties were all below health-based comparison values and were not expected to result in a health threat to community residents. Copper levels in the area of the Spicket River were found to be above comparison values and required further evaluation.

An evaluation of potential exposure pathways was conducted to determine whether contamination identified at the 54 Osgood Street site could be impacting nearby residents of Methuen in the past, present, or future. Fugitive or stack air emissions may have possibly occurred at the site while the facility was in operation; however, there are no data relative to past operations at the site. Since a review of available information did not find any historical record of hazardous materials being released at the site, exposure to the hazardous materials prior to the fire on the site would probably have occurred among individuals who worked in the building. During and immediately following the fire, it is possible that residents may have come in contact with contaminated water from firefighting activities as well as contaminated smoke. However, the response team sought

to minimize and prevent offsite migration of this water through the use of sand berms and vacuum pump trucks; therefore, it is unlikely that residents would have come in contact with contaminated runoff. In addition, the climate in January would have limited residents' time outdoors, thus reducing exposure opportunities to contaminated smoke. Present and future exposure to contaminants from 54 Osgood Street is unlikely since contaminated debris and remaining hazardous materials were removed from the site following the 1994 fire.

Reviewing the list of chemicals present at the time of the fire showed that the majority of chemicals onsite were most notable for their short-term (acute) effects. Most of the chemicals were some form of acid and would have caused a short-term burning sensation for individuals that came into contact with the smoke plume on the day of the fire. Two Methuen firefighters were transported to a local hospital on the day of the fire for possible chemical exposure after having been in the smoke (Weston 1994). Therefore, the chemicals in the smoke plume emanating from the fire may have presented an immediate hazard to local residents. However, the fire occurred in mid-January, and the high temperature for the day was 5º F. It is reasonable to assume that residents would have limited time outdoors on this day and likely remained indoors with the windows closed, thus limiting their exposure to the smoke plume.

Copper is not classified as a human carcinogen by the EPA and typically is considered an essential nutrient important for good health (USEPA 2006b, ATSDR 2004). However, exposure to high levels of copper could result in adverse health effects including liver and kidney damage, anemia, immunotoxicity, and developmental toxicity (ATSDR 2004). Although the area sampled on the east bank of the Spicket River is located in a commercial/industrial area, it is possible that a child resident could visit this portion of the river and could have infrequent contact with the soil. To evaluate the potential for health effects from such exposure to copper, ATSDR Minimal Risk Levels (MRLs) were compared to exposure estimates for copper at the 54 Osgood site. The MRL is an estimate of daily exposure to a contaminant below which non-cancer, adverse health outcomes are unlikely to occur. Assuming a child resident inadvertently ingested 200 mg

of soil from the bank of the Spicket River containing the maximum concentration of copper detected in the area (1,590 ppm) for 2 days each week for 22 weeks (May through September, the warmer months of the year), a child resident would not be expected to experience increased non-cancer health effects.

Site Status

In response to a fire in 1994, immediate response actions were taken to minimize and prevent offsite migration of hazardous materials from the 54 Osgood Street property. Environmental sampling following the fire demonstrated that the Spicket River, as well as the surrounding properties, had not been adversely impacted by the fire (MDEP 1995c). On January 9, 1995, the immediate response action was considered complete and no further action was deemed necessary (MDEP 1995c).

V. Methods to Evaluate Health Concerns

A. Analysis of Cancer Incidence

As part of this investigation, the CAP reviewed cancer incidence data available from the MCR for brain cancer (all ages) and childhood cancer (all cancer types in ages 0 – 19) in Methuen. The 21-year period from 1982-2002 was evaluated and constituted the time period for which the most recent and complete cancer incidence data were available at the time of this report. [Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. See Appendix B for the incidence coding definitions used in this report.] The MCR is a division in the MDPH Bureau of Health Information, Statistics, Research, and Evaluation. It is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain *in situ* (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111. s 111b). The MCR also gathers background information (e.g. gender, age, address at diagnosis) on each individual reported. This information is kept in a confidential database. Data are

collected daily and reviewed for accuracy and completeness on an annual basis. Due to the high volume of data collected 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.

To determine whether cancer incidence in a community is occurring at a higher or lower rate than expected, a statistic called a standardized incidence ratio (SIR) is calculated using data from the MCR. More specifically, the SIR is the number of observed cancer diagnoses in a town divided by the number of expected diagnoses multiplied by 100. The expected numbers are estimated using the age and gender distribution of the town and the state's corresponding cancer rates. An SIR greater than 100 indicates that more cancer diagnoses occurred than expected; an SIR less than 100 means that fewer diagnoses occurred than expected. For example, an SIR of 150 is interpreted as 50 percent more diagnoses than expected; an SIR of 90 indicates 10 percent fewer diagnoses than expected. The SIR by itself is an estimate and the true SIR is unknown. Therefore, a related statistic, called the 95% confidence interval (CI), is used to interpret the SIR and determine if the SIR is statistically significant.

Specifically, a 95% CI is the range of estimated SIR values that have a 95% probability of including the true SIR for the population. This range of numbers was calculated for each SIR to determine if the observed number of diagnoses was "significantly different" from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). If the 95% CI range does not include the value of 100, then the study population is considered 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 observed number of diagnoses.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105-130), there is a statistically significant excess in the number of cancer diagnoses. Similarly, if the confidence interval does not include 100 and the interval is

below 100 (e.g., 45-96), then statistically this means that the number of cancer diagnoses is significantly lower than expected. If the confidence interval range includes 100, then the true SIR may be 100. In this case, it cannot be determined with certainty that the difference between the observed and expected number of diagnoses reflects a real cancer increase or decrease in cancer incidence or is the result of chance.

In addition to the range of the SIR 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 (e.g., 103-115) allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval (e.g., 85-450) leaves considerable doubt about the true SIR, which could be much lower or higher than the calculated SIR. This would indicate an unstable statistic. Due to the instability of incidence rates based on a small number of diagnoses, statistical significance was not assessed when fewer than five diagnoses were observed.

Because accurate age group and gender-specific population data are required to calculate SIRs, the census tract (CT) is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, census tracts are small, relatively permanent geographic entities within counties (or the statistical equivalents of counties) defined by a committee of local data users following Census Bureau guidelines. Census tract boundaries normally follow visible features, but may follow governmental unit boundaries and other non-visible features in some instances. CTs usually contain between 2,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (See Figure 2). Over time, a city or town changes population and the number of census tracts can change with the population. This occurred in the city of Methuen. In the 1980 U.S. Census, the city of Methuen had six different CTs, in the 1990 U.S. Census it had 10 CTs, and in the 2000 U.S. Census it had 11 CTs (U.S. Census Bureau 1980, 1990 and 2000). All SIRs in this report were calculated using the 11 CTs that are currently in Methuen.

B. Review of Risk Factor Information

Despite numerous scientific and medical investigations, the causes of brain cancer are still largely unknown. However, a few risk factors have been identified. For example, the most well-established risk factor is exposure to ionizing radiation (e.g., from radiation therapy to the head and neck) (ACS 2006a). In addition, rare diagnoses of brain cancer run in some families. Some types have also been associated with certain rare genetic disorders, such as neurofibromatosis types 1 and 2, von Hippel-Lindau disease, tuberous sclerosis, and Li-Fraumeni syndrome (ACS 2006a). Environmental factors, such as exposure to vinyl chloride, have shown inconsistent results when examined by researchers. Aspartame (a sugar substitute) and electromagnetic fields have also been suggested as risk factors for brain cancer, but the evidence does not support an association between these exposures and brain cancer (ACS 2006a).

Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 25% of all reported primary brain tumors and the majority of spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the neurons of the cerebellum and are most often seen in children. Also, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS 2006a).

In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme). Incidence rates are higher in males than in

females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack 1996).

Brain and spinal cord cancers account for over 20% of malignant tumors diagnosed among children aged 0-14 (ACS 2006b). About half of all childhood brain tumors are astrocytomas and 25% are primitive neuroectodermal tumors (PNET), which spread along the spinal cord and the meninges (ACS 2006b). After a peak in childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75.

While there is little information on the causes of brain cancer, there is even less conclusive information on what is responsible for cancers that occur in children. Many of the lifestyle risk factors that are associated with some types of adult cancer (e.g., diet, smoking, and physical activity) are not relevant to childhood cancers. Some childhood cancers are the result of familial predisposition (cancer runs in families). Radiation exposures contribute to certain types of childhood cancers. However, the cause of most childhood cancers is not known (ACS 2006b).

To better understand the pattern of brain and childhood cancers in Methuen, available case information from the MCR was evaluated for individuals diagnosed from 1982 to the present[1]. The information reviewed included date of diagnosis, age at diagnosis, histology (cancer cell type), and previous cancer diagnoses. However, information about personal risk factors that may also influence the development of brain or childhood cancer (e.g., family history or heredity) are not collected by the MCR, and therefore, could not be evaluated in this investigation.

[1] The cancer incidence data in this report are drawn from data entered on MCR computer files before September 6, 2007. The numbers presented in this report may differ slightly from those published in previous or future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

C. Geographic Distribution

Address at the time of diagnosis was mapped for all childhood diagnoses of cancer and all brain cancer diagnoses in Methuen from 1982 to the present using ArcGIS software (ESRI 2005). Because the MCR is a continual surveillance system for cancer, it was possible to review case reports for more recent years (i.e., 2003-present) to qualitatively assess any possible spatial patterns of cancer diagnoses. However, because the data for the most recent years (i.e., 2003-present) are not complete, they cannot be used to calculate incidence rates. Due to community concerns related to the three sites (4 Gleason Street, 254 Broadway and 54 Osgood Street), particular attention was paid to the spatial pattern of brain cancer and childhood cancer in the census tract where these sites are located (CT 2523) as well as the two adjacent census tracts (CT 2524 & CT 2525.02) (See Figure 2).

The MDPH is bound by law not to make public the names or any other information (e.g., place of residence) that could personally identify individuals with cancer whose diagnoses have been reported to the MCR (M.G.L. c.111. s. 24A). Therefore, for confidentiality reasons, it is not possible for the MDPH to release maps showing the locations of individuals diagnosed with cancer in public reports. However, a summary of the evaluation of geographic distribution with any notable findings is presented in this report.

D. Hospital Discharge Database

The Massachusetts Division of Health Care Finance and Policy (DHCFP) collects patient-level information on all patients who are discharged from hospitals in Massachusetts. The collection of data by the DHCFP is mandated by regulation 114.1 CMR 17.00, *Requirement for the Submission of Hospital Case Mix and Charge Data*. Hospital discharge data are available through the MDPH Community Health Information Profile (MassCHIP) database. While the MCR collects address at diagnosis for each individual in its database, the hospital discharge database does not collect detailed

address information. Therefore, the CAP was able to examine aggregate data on asthma for the city of Methuen as a whole.

Each hospitalization in Massachusetts is coded using the International Classification of Disease 9th Revision codes (ICD-9). The hospital discharge database was searched using the specific ICD-9 code for asthma, a respiratory condition that can be affected by environmental conditions (including air quality and exposure to respiratory irritants) and is of community interest.

The data were examined to determine how many times individuals from Methuen were hospitalized with an asthma-related condition each year from 1989-2002. The statistic calculated and reported is the Standardized Hospitalization Ratio (SHR). The SHR is analogous to the SIR in its calculation and interpretation. However instead of counting individuals diagnosed with asthma, the SHR utilizes the number of hospitalizations for a city or town.

There are some limitations to the data on asthma-related hospitalizations. First, unlike cancer data that are available at a smaller geographic level (i.e., census tract level), the data for hospitalizations are only provided by MassCHIP at the city/town level or at the zip code level. The city of Methuen has only one zip code that covers the entire city. This makes it impossible to determine if one area of the city is affected more than another area of the city. The SHR will only determine if Methuen is experiencing a higher or lower rate of hospitalizations from asthma-related conditions as compared to the state hospitalization rate. Another important limitation would be that these numbers are counts of hospitalizations for asthma, not the number of individuals who have asthma in a town or city. For example, if the database is examined for the number of asthma-related hospitalizations in Methuen during a specified time period, it would count one individual hospitalized ten times the same number of times as it would count ten individuals hospitalized once each. It is important to keep this information in mind when interpreting the hospital discharge data presented in this report. These data, however, should allow for

an evaluation of whether the population as a whole experienced respiratory impacts at greater rates during periods of possible contamination of the air.

E. Evaluation of Neighborhood Survey

The CAP reviewed the 11 surveys that were provided by a resident of the Gleason Street neighborhood with the original written request to the MDPH for a review of cancer incidence. Each survey identified an individual in Methuen who reportedly had been diagnosed with either brain cancer, an immune-related disease, or had experienced symptoms that he or she attributed to an environmental exposure. The survey collected information on symptoms, diagnoses, and residential history on a standardized form created by the residents of Methuen.

A systematic surveillance system for the reporting of illnesses or conditions other than cancer did not exist in Massachusetts prior to 2004. Therefore, for the purposes of this report it is not possible to estimate the incidence or prevalence of diseases other than cancer. To evaluate the conditions mentioned in the surveys, the CAP researched the current medical literature for information relating to each condition as well as new data that have become available through an environmental public health tracking initiative funded by the United States Centers for Disease Control and Prevention (CDC). The CAP also looked for any specific environmental risk factors that could be associated with any of the diseases mentioned in the surveys. Also, the CAP assessed whether any commonalities appeared to exist among the diseases mentioned.

VI. Results of Evaluation of Health Concerns

A. Brain Cancer Incidence

1. 1982-1988

Table 6 summarizes the incidence of brain cancer for the city of Methuen during the seven-year time period of 1982-1988. Overall, the city experienced an increased

incidence of brain cancer. There were 25 diagnoses of brain cancer in Methuen when approximately 18 would have been expected to occur. However, this elevation in brain cancer incidence was not statistically significant (SIR=141, 95% CI=91-208). A separate evaluation by gender revealed that both males and females experienced an elevation in brain cancer incidence. However, neither males (12 observed versus 9.3 expected, SIR=129, 95% CI=66-225) nor females (13 observed versus 8.4 expected, SIR=154, 95% CI=82-263) had a statistically significant increase in the incidence of brain cancer.

With the exception of one census tract (CT 2526.01), the number of observed diagnoses of brain cancer in Methuen's census tracts was about as expected (i.e., within one of the number expected). In CT 2526.01, which is located on the eastern side of Methuen, there were seven brain cancer diagnoses observed when about two would have been expected (SIR=347, 95% CI=139-714). Both males and females residing in this CT experienced an elevated brain cancer incidence (males: 3 observed versus 1.1 expected, females: 4 observed versus 0.9 expected). In the CTs that surround the sites of concern (CT 2523, CT 2524, and CT 2525.02), the number of individuals diagnosed with brain cancer during this time period was slightly below or approximately at the rate that would have been expected (CT 2523: 2 observed versus 2.7 expected; CT 2524: 2 observed versus 1.9 expected; and, CT 2525.02: 2 observed versus 2.4 expected).

a) Risk Factor Information

The average age of individuals in Methuen diagnosed with brain cancer during this time period was 55 years with an age range of 1 to 85 years. In CT 2526.01, where a statistically significant elevation of brain cancer occurred, the average age at diagnosis was 62, with a range of 33 to 80 years of age. From 1982-1988, two of the 25 individuals (8%) diagnosed citywide were 19 years of age or younger at diagnosis when approximately two would have been expected.

During this time period, the number of brain cancer diagnoses fluctuated from year to year, with anywhere between one and eight diagnoses occurring every year. There was,

however, no trend noted of either an increasing or decreasing number of diagnoses over time.

Twenty-three of the 25 individuals (92%) were diagnosed with a glioma-type of brain cancer. This is the most common form of brain cancer. The two remaining diagnoses of brain cancer were both different subtypes of brain cancer. Among the 25 individuals diagnosed with brain cancer, one (4%) had a previous diagnosis of another cancer type.

b) Geographic Distribution

The geographic distribution of the 25 brain cancer diagnoses during 1982-1988 was consistent with the population distribution. A combined total of six brain cancer diagnoses occurred in CTs 2523, 2524, and 2525.02, the area of the three sites of concern, while seven would have been expected based on the statewide brain cancer experience. Two of the individuals lived within a ½-mile radius of the three sites at the time of their diagnosis while the remaining four did not.

2. 1989-1995

Table 7 summarizes the incidence of brain cancer in the city of Methuen during the seven-year time period of 1989-1995. Overall, the city experienced an increase in brain cancer incidence. There were 30 diagnoses in Methuen when approximately 20 would have been expected to occur. However, this elevation was not statistically significant (SIR=147, 95% CI= 99-210). During this time period, the elevation of brain cancer incidence occurred in both genders but neither males (16 observed versus 10.9 expected, SIR=146, 95% CI= 83-237) nor females (14 observed versus 9.5 expected, SIR=147, 95% CI= 80-247) had a statistically significant increase.

The observed number of diagnoses was near the expected number of diagnoses in six of the 11 CTs (i.e., within one of the number expected). In five of the 11 CTs (CTs 2521.01,

2521.02, 2522.01, 2523, and 2525.02), the number of observed diagnoses exceeded the number expected by two. In the CTs that include or surround the industrial sites of concern (CTs 2523, 2524, and 2525.02), the numbers of individuals diagnosed with brain cancer during this time period compared to what would have been expected were as follows: CT 2523: 5 observed versus 3.0 expected (SIR=169, 95% CI=54-340); CT 2524: 1 observed versus 2.0 expected; and, CT 2525.02: 5 observed versus 2.6 expected (SIR=192, 95% CI=62-449). In CT 2525.02 four females were diagnosed when one would have been expected. Among males the occurrence of brain cancer occurred at about the expected rate (1 observed versus 1.4 expected). In CTs 2523 and 2525.02, the differences were not statistically significant.

a) Risk Factor Information

The average age of the individuals in Methuen diagnosed with brain cancer during this time period was 54 with an age range of 1 to 90 years. During this time period, four of the 30 (13%) individuals diagnosed with brain cancer were 19 years of age or younger when approximately two would have been expected.

During this time period, the number of brain cancer diagnoses fluctuated from year to year, with between two and seven diagnoses occurring every year. There was, however, no trend noted of an increasing number of diagnoses over time.

Twenty-six of the 30 (87%) individuals were diagnosed with a glioma. The four remaining individuals with brain cancer had three different brain cancer subtypes. None of the 30 individuals diagnosed with brain cancer had a previous diagnosis of any cancer type reported to the MCR. Among the four children diagnosed with brain cancer during this time period, two different subtypes of brain cancer were diagnosed.

b) Geographic Distribution

The geographic distribution of the 30 brain cancer diagnoses during 1989-1995 was consistent with the population distribution. While there was a combined total of 11 brain cancer diagnoses in CTs 2523, 2524, and 2525.02, the area of the three sites of concern, four of the diagnoses lived within a ½-mile radius of the three sites at the time of their diagnosis while the remaining seven did not. Approximately eight diagnoses would have been expected in these three CTs.

3. 1996-2002

Table 8 shows the overall incidence of brain cancer in the city of Methuen during the seven-year time period of 1996-2002. Overall, Methuen experienced an increased incidence of brain cancer. There were 29 diagnoses in Methuen when approximately 23 would have been expected to occur. However, this elevation was not statistically significant (SIR=128, 95% CI= 85-183). The elevation in brain cancer incidence occurred in both genders with neither males (16 observed versus 12.0 expected, SIR=133, 95% CI= 76-215) nor females (13 observed versus 10.7 expected, SIR=122, 95% CI= 62-208) having a statistically significant increase in the incidence of brain cancer.

With the exception of two CTs (2523 and 2526.01), the number of observed diagnoses of brain cancer in Methuen's CTs occurred about as expected (i.e., within one of the number expected.) The two CTs each had six diagnoses of brain cancer when approximately three were expected in each CT. Neither SIR was statistically significantly elevated. When brain cancer incidence was examined by gender, males in CT 2523 had a statistically significant elevation in brain cancer incidence with five males diagnosed when approximately two would have been expected (SIR=312, 95% CI= 101-728). In the CTs that surround the three sites of concern (CTs 2523, 2524, and 2525.02), the number of individuals diagnosed with brain cancer during this time period versus the number expected was as follows: CT 2523: 6 observed versus 3.2 expected (SIR=186, 95% CI=68-406); CT 2524: 0 observed versus 1.7 expected; and, CT 2525.02: 4 observed versus 2.9 expected.

a) Risk Factor Information

The average age of the individuals in Methuen diagnosed with brain cancer during 1996-2002 was 58 with an age range under 1 year of age to 92 years of age. In CT 2523, which had the statistically significant elevation of brain cancer diagnoses among males (5 observed versus 1.6 expected), the average age of the individuals at diagnosis was 46, with a range of 7 to 92 years of age. Three of the 29 (10%) individuals diagnosed citywide with brain cancer were 19 years of age or younger when approximately two would have been expected. These three children were diagnosed with two different subtypes of brain cancer.

During this time period, the number of brain cancer diagnoses fluctuated from year to year. There was, however, no trend noted of either an increasing or decreasing number of diagnoses over time.

Twenty-seven of the 29 (93%) individuals were diagnosed with a glioma. The two remaining individuals with brain cancer had two different subtypes of brain cancer. Three of the 29 individuals had a previous diagnosis of another cancer type.

b) Geographic Distribution

The geographic distribution of the 29 brain cancer diagnoses during 1996-2002 was consistent with the population distribution. There was a combined total of 10 brain cancer diagnoses in CTs 2523, 2524, and 2525.02, the area of the three sites of concern, when approximately eight diagnoses would have been expected in these three CTs. The 10 brain cancer diagnoses were not distributed among all three of the CTs. Four of the 10 individuals lived within a ½-mile radius of the three sites at the time of their diagnosis. In CT 2523 six individuals were diagnosed with brain cancer when approximately three would have been expected; two of the three children diagnosed with brain cancer in this

CT lived in close proximity to one another. The remaining four individuals resided in CT 2525.02 where approximately three diagnoses would have been expected and no individuals resided in CT 2524 when approximately two would have been expected.

4. 2003-present

There have been an additional 12 diagnoses of brain cancer among residents of Methuen reported to the MCR since 2002. The age range for these diagnoses was from 4 to 82 years of age, with an average age of 51. When the spatial pattern of these current diagnoses was examined, there was no apparent clustering of the diagnoses within Methuen. Two of these diagnoses were among individuals under the age of 19 and one of the children resided in CT 2523. The other child was diagnosed while living in CT 2521.01

B. Childhood Cancer Incidence

1. 1982-1988

Table 9 summarizes the incidence of childhood cancer in the city of Methuen during the 7-year time period of 1982-1988. Children living in Methuen during this time period experienced an incidence of cancer that was about what would be expected based on the statewide childhood cancer rate. There were 11 diagnoses of childhood cancers in Methuen when 11.4 would have been expected to occur (SIR=96, 95% CI= 48-172). Neither gender had an increased incidence of childhood cancers (males: 6 observed versus 6.2 expected; females: 5 observed versus 5.3 expected). For each CT, the number of observed diagnoses was about what was expected. In the CTs that surround the three sites of concern (CTs 2523, 2524, and 2525.02), the number of children diagnosed with cancer during this time period was lower than what would have been expected (CT 2523: 0 observed versus 1.6 expected; CT 2524: 1 observed versus 1.9 expected; and, CT

2525.02: 0 observed versus 1.2 expected). Overall one child was diagnosed with cancer when approximately five would have been expected.

a) Risk Factor Information

Among the 11 children diagnosed with cancer during this time period, there were eight different types of cancer diagnosed (neuroblastoma, brain, Hodgkin's disease, leukemia, ovarian, Ewing's sarcoma and a germ cell tumor). The 11 diagnoses were spread across the 7-year time period, with each year having at least one diagnosis of a cancer in an individual aged 19 or under. The ages of the 11 children at diagnosis also varied from under 1 year of age to 18 years of age. The age at diagnosis for each individual was consistent with what would be expected for the type of cancer diagnosed.

b) Geographic Distribution

The geographic distribution of the cancer diagnoses was generally consistent with the population distribution. In the three CTs of concern (CT 2523, 2524, and 2525.02), there was one diagnosis of cancer when approximately five would have been expected. At the time of diagnosis, this child lived more than a ½-mile away from the three sites of concern in central Methuen.

2. 1989-1995

Table 10 summarizes the incidence of childhood cancer in the city of Methuen during the seven-year time period of 1989-1995. Children living in the city during this period experienced an incidence of cancer that was about what would be expected. There were 13 diagnoses of childhood cancers in Methuen when approximately 12 diagnoses would have been expected to occur (SIR=109, 95% CI= 58-186). The incidence of childhood cancers was about as expected for both genders (males: 8 observed versus 6.7 expected, females: 5 observed versus 5.2 expected). For each CT, the number of observed

diagnoses was about what was expected. In the CTs that surround the three sites of concern (CT 2523, 2524, and 2525.02), the number of children diagnosed with cancer during this time period was approximately as expected (CT 2523: 2 observed versus 1.5 expected; CT 2524: 2 observed versus 2.3 expected; and, CT 2525.02: 3 observed versus 1.1 expected).

a) Risk Factor Information

Among the 13 children diagnosed with cancer during this time period, there were eight different types of cancer diagnosed (liver, colorectal, brain, Hodgkin's disease, non-Hodgkin's lymphoma, leukemia, germ cell and neuroepithelial). The 13 diagnoses were spread across the 7-year time period, with each year in the time period having at least one diagnosis of cancer in an individual 19 years of age or under. The ages at diagnosis for the 13 children ranged from 1 year to 18 years of age. The ages of diagnosis were consistent for the types of cancer diagnosed.

b) Geographic Information

The geographic distribution of residence at time of cancer diagnosis was generally consistent with the population distribution. In the three CTs of concern (CT 2523, 2524, and 2525.02), there were seven diagnoses of cancer and one of the children lived within a ½-mile of the three sites of concern. Approximately five diagnoses were expected in the three CTs. There were no brain cancer diagnoses among children residing within a ½-mile of the three sites.

3. 1996-2002

Table 11 summarizes the incidence of childhood cancer in the city of Methuen during the 7-year time period of 1996-2002. Children experienced an incidence of cancer that was about what would be expected. There were 12 diagnoses of childhood cancers in

Methuen when approximately 13 diagnoses would have been expected to occur (SIR=90, 95% CI= 46-157). Both genders experienced an incidence of childhood cancers that was about as expected (males: 8 observed versus 7.2 expected; females: 4 observed versus 6.2 expected). In two of the three CTs that surround the three sites of concern (CTs 2524 and 2525.02), the number of children diagnosed with cancer during this time period was close to what would have been expected (CT 2524: 0 observed versus 1.8 expected and CT 2525.02: 1 observed versus 1.2 expected). CT 2523 had five observed diagnoses of cancer when approximately two would have been expected (SIR=309, 95% CI= 100-723); this difference represents a borderline statistically significant elevation in the number of children diagnosed with childhood cancer.

a) Risk Factor Information

Among the 12 children diagnosed with cancer during this time period, there were eight different types of cancer diagnosed (melanoma, ovarian, kidney, brain, thyroid, Hodgkin's disease, non-Hodgkin's lymphoma, and neuroblastoma). The 12 diagnoses were spread across the 7-year time period with 6 of the 7 years having at least one diagnosis of cancer in a child; in 1998 were there no diagnoses of childhood cancer.

b) Geographic Distribution

In the three CTs of concern (CT 2523, 2524, and 2525.02), there were six diagnoses of cancer and approximately five would have been expected. Four of the children lived within a ½-mile of the three sites of concern and three had a diagnosis of brain cancer. Their dates of diagnosis were spread out across the time period and two different histological subtypes of brain cancer were identified.

4. 2003-present

Since 2002, an additional 13 cancer diagnoses among 12 children have been reported to the MCR. These diagnoses varied in type (Hodgkin's disease, Non-Hodgkin's

Lymphoma, leukemia, retinoblastoma, neuroblastoma, and brain, skin, bone, and kidney cancers) and were not concentrated in any area of Methuen. There were six diagnoses among females and seven diagnoses among males from 2003 to the present. One of the 13 diagnoses was brain cancer; this individual's residence at the time of diagnosis was within ½ of a mile of the three sites of concern.

C. Asthma Incidence

Table 12 summarizes the number of hospitalizations for asthma-related conditions for the city of Methuen for each year from 1989 to 2002. Overall, the number of asthma-related hospitalizations was either around or below the expected value. For several of the years (1994-1996 and 1999-2002) the number of hospitalizations was statistically significantly lower than expected. When examined by gender the number of asthma-related hospitalizations was about as expected or lower each year for both males and females. There were several years (1995 and between 1999 and 2002) when asthma-related hospitalizations were statistically significantly lower than expected among both genders. Also, females experienced other years of statistically significantly lower number of asthma-related hospitalizations (1990, 1994 and 1996).

D. Survey Evaluation and Risk Factor Information

Included in the original request to MDPH for an investigation were 11 surveys completed by individuals who lived around the three sites and who reported having a variety of different diseases. The neighborhood survey was initiated and conducted by a concerned resident. All of the information was reportedly provided by the individual or a relative, when an individual was not available to complete the survey. The surveys identified a wide variety of self-reported illnesses including brain cancer, Multiple Sclerosis (MS), Myasthenia Gravis, asthma, and possible immune-mediated hearing loss.

CAP reviewed the information provided on each survey. The scientific literature was searched for relevant information about environmental exposures that could potentially be related to the reported disease. New data from MDPH's environmental public health tracking initiative was also reviewed as appropriate. The following discussion of each disease mentioned in the surveys explores the basic mechanisms of the disease, the prevalence (and/or incidence) and any known (or suspected) risk factors for each disease. It was not possible for the CAP to determine the cause of any one person's illness. Pertinent information such as an individual's medical history, family history of disease, and lifestyle information may be important in the development of an individual's disease. One or several factors acting over time can be related to the development of a disease.

The individuals who completed the surveys ranged in age from 8 to 62 years old. All of the individuals were living in Methuen at the time they completed the survey, but most had not been at their reported addresses for their entire lives. The number of years for which each individual had been living at their current address ranged from 6 to 33 years. Individuals provided a previous address to the one where they were living at the time of the survey.

1. Brain Cancer

Two of the surveys were for individuals in Methuen who had been diagnosed with brain cancer. As discussed previously in this report, there is little scientific information on the causes of brain cancer. One reason for the lack of understanding of the risk factors for brain cancer is the unusually diverse array of histologic (cell) types. Risk factors are discussed below.

Exposure to ionizing radiation is the only established environmental risk factor. Individuals can be exposed to high doses of ionizing radiation when they are treated for various types of cancer. Historically, ionizing radiation was also used to treat an assortment of non-cancerous conditions [i.e. tinea capitus (ringworm of the scalp)] (Savitz and Trichopoulos 2002). While the practice of using ionizing radiation as a

treatment for benign, non-cancerous conditions has declined to a great extent, it is still in use by doctors today for a limited number of medical conditions (Abson 2000). As mentioned previously, information collected by the MCR does not provide details about past medical treatments of individuals diagnosed with cancer. Appendix C provides further discussion of risk factor information on brain cancers.

The incidence of brain cancer in the general population has been described as having two peaks, one in early childhood prior to the age of 10 and then another peak in older individuals around the age of 70 (Savitz and Trichopoulos 2002). As with many types of cancer, the incidence of brain cancer increases with age. The age distribution of those Methuen individuals diagnosed with brain cancer is consistent with national and state patterns.

2. Immune-Related Diseases

Six of the remaining nine individuals listed a disease on their survey that involves the immune system. The immune system utilizes various mechanisms to protect the body against disease. Generally, the body is protected either by a passive defense such as a physical barrier, like the skin, or an active defense by various cells of the immune system. When the active immune system does not work properly it can cause one of the following conditions:

- an autoimmune disease (e.g., an immune response against the body)
- an allergic disease (e.g., a normal immune response to a foreign antigen that damages normal tissue)
- an immunodeficiency disease (e.g., the body cannot generate an appropriate immune response against invading microorganisms)

Each of the following four diseases fit into one of these three categories of immune-related diseases.

a) Multiple Sclerosis

Three of the individuals who completed a survey reported a diagnosis of Multiple Sclerosis (MS). MS is an autoimmune disease that damages or destroys patches of myelin (the protective coating on nerve cells) and the underlying nerve fibers in the eyes, brain, and spinal cord. This damage affects the ability of nerves to conduct electrical impulses to and from the brain. Individuals with MS generally develop problems with their muscle control.

MS is a major cause of nervous system disability among young people (i.e., individuals under the age of 45) in the United States (Lutton et al. 2004). MS is most frequently diagnosed in adults between the ages of 20 and 40, with prevalence highest in the 40 to 59 year age range (Nielsen et al. 2005; Noonan et al. 2002). Women are generally diagnosed with MS at a rate of roughly two to one compared to males. The prevalence (number of people currently living with the disease) of MS is estimated to be somewhere between 60 and 140 diagnoses per 100,000 individuals (NMSS 2002). In an MDPH study to estimate the prevalence of MS in southeastern Massachusetts for the period 1998 – 2003, a prevalence rate of 146 per 100,000 population was estimated after reviewing the medical records of neurologists serving the study population. This estimate is within the range observed in other studies conducted in the northern latitudes (north of the 37th parallel); some studies have shown a higher prevalence of MS in northern latitudes compared to southern latitudes. However, the prevalence of MS in others parts of the state may be higher or lower than this estimate. In the 2000 U.S. Census the city of Methuen had approximately 43,789 residents, meaning that roughly 60 individuals in Methuen would be expected to have MS at any one time.

The causes of MS are still unknown, but many studies have suggested that genetics and environmental exposures may play a role in the development of MS. That is, MS is thought to be a disease resulting from the complex interplay of genes and environmental factors. Hundreds of epidemiological studies have been conducted of MS and some have reported associations between MS and exposure to viral agents, organic solvents, low temperatures, trauma, and various other factors. Several viruses thought to raise an

individual's risk of developing MS are the Epstein-Barr virus (EBV) and Human Herpes virus [HHV (specifically HHV-6 and HHV-8)] (Cook 2004; Lutton et al. 2004). The National MS Society (NMSS) characterizes MS as a disease that is not directly inherited, but one for which those inflicted carry a genetic predisposition for the disease. In addition, the NMSS believes that MS may be triggered by something in the environment, such as an infectious viral or bacterial agent. It may be that no single agent will ever be shown to exert a cause-and-effect relationship, but rather that people with genetically predisposed immune systems may react to certain bacteria, viruses, or other environmental agents in a way that results in the expression of MS.

b) Myasthenia Gravis

One of the individuals who completed a survey reported a diagnosis of Myasthenia Gravis. Myasthenia Gravis is a disease in which the body's own antibodies bind to acetylcholine receptors in muscle cells, thereby impairing muscle contraction. Receptors are structures on the surface of cells that react with specific stimuli. Acetylcholine is a neurotransmitter (stimulus) that conveys impulses between the ends of nerve cells and muscle cells. An individual diagnosed with Myasthenia Gravis has fewer acetylcholine receptors available to receive the message that a nerve is trying to transmit; this leads to muscle fatigue and weakness due to a lack of proper stimulation by the nerves. The mechanism of this disease is one of the most thoroughly understood of the autoimmune diseases. It has served as a model for understanding the mechanisms of other autoimmune diseases (Drachman 1994).

The first symptoms of Myasthenia Gravis can appear at any age; however, they usually appear at an early age in women while, among males, the disease more commonly occurs at older ages. The incidence in women peaks in their 20's and 30's while in males in their 50's and 60's (Thanvi and Lo 2004). The prevalence of Myasthenia Gravis is estimated to be somewhere between 10 and 20 diagnoses per 100,000 individuals in the United States (Phillips 2004).

Similar to other autoimmune diseases, the cause of Myasthenia Gravis is thought to be a complex interaction between genetics and the environment (Drachman 1994). The thymus (a gland located in the chest behind the breastbone) is believed to play a role in Myasthenia Gravis. The removal of the thymus, a procedure called a thymectomy, relieves the symptoms for some individuals with Myasthenia Gravis (Budde et al. 2001). Other than the possible role that the thymus may play in Myasthenia Gravis, no clear picture exists as to the origin of the disease. A search of the current literature did not identify any chemical contaminants in the soil, air, or water as likely risk factors for the development of Myasthenia Gravis.

c) Asthma

One of the individuals surveyed in Methuen reported a diagnosis of asthma. Asthma is an immunological disease, but it is not an autoimmune disease like the other diseases mentioned previously. Asthma is a reversible obstructive lung disease caused by increased reaction of the airways to various stimuli. When an individual suffers from asthma, the muscles of the airways constrict causing an interruption in normal breathing.

Male children are more likely to be diagnosed with asthma than female children. This gender inequality changes once individuals reach adulthood when women become more likely than men to develop asthma (ALA 2005). Asthma that occurs in childhood will sometimes resolve itself before adulthood. Currently about 7% of the entire U.S. population suffers from asthma. Children are more likely to be diagnosed with asthma than adults, however, with approximately 12.5% of children in the US having been diagnosed with asthma (Dey and Bloom 2005). In the last few decades the prevalence of asthma has been increasing, especially among children. The reason for this increase has yet to be explained. Data available from the MDPH's environmental public health tracking program indicate that the current rate of pediatric asthma is 10% for the state as a whole. The city of Methuen's pediatric asthma rate in the 2004-2005 school year was 8.2% (95% CI 7.5% - 9.0%) (MDPH 2006).

Although there is medical agreement on agents that can trigger asthma, such as cold air and allergens like animal dander, current information in the medical literature does not provide firm conclusions about the causes or risk factors for the onset of asthma. Many researchers have sought to identify specific environmental exposures associated with the development of asthma. In a report by the Institute of Medicine for the EPA, on both exposures related to asthma onset and the worsening of already established asthma, exposure to house dust mites was reported to *cause* asthma onset while exposure to environmental tobacco smoke among preschool age children was reported to be *associated with* asthma onset (meaning the weight of the evidence for causation was less for environmental tobacco smoke than for house dust mites) (Redd 2002). It is well-documented that outdoor exposures to ozone and particulate matter can exacerbate asthma. However, whether outdoor pollution is associated with asthma onset is not known. Data available from the MDPH's environmental public health tracking program suggests that indoor exposure to moisture and/or mold may be associated with increased rates of pediatric asthma.

d) Autoimmune Inner Ear Disease

One of the individuals who completed a survey reported bilateral hearing loss and was told by their physician that it was an autoimmune-related condition. A medical condition called Autoimmune Inner Ear Disease (AIED) can cause sudden bilateral hearing loss and tinnitus (Werneck et al. 2003). Currently about one in 10 individuals in the United States suffer from some form of hearing loss and about one-third of individuals over 65 have significant hearing loss (Lalwani and Snow 2005). However, AIED is rare. Less than 1% of hearing loss incidence in the population can be accounted for by AIED (Werneck et al. 2003).

The condition of autoimmune hearing loss was first described in 1979 (McCabe 1979). Multiple potential mechanisms have been identified that can result in AIED, but there is no accepted mechanism of disease (Ruckenstein 2004). In a search of current medical

literature, no articles were found that either examined or hypothesized environmental factors as being involved in the development of AIED.

3. Other illnesses

In the remaining three surveys, the individuals did not list any specific medical conditions. Each survey listed numerous symptoms that could be associated with several different diseases. Due to a lack of information, it is not possible to discuss what may be causing these symptoms or how the symptoms may be related to the environment.

VII. Discussion

At the request of concerned residents, the Community Assessment Program (CAP) at the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of three state-regulated former industrial sites and cancer incidence for the city of Methuen. This evaluation was initiated because of community concerns about a suspected increase in the overall incidence of brain cancer and childhood cancer (individuals aged 19 and under at diagnosis) among individuals living near of three former industrial sites at 4 Gleason Street, 254 Broadway, and 54 Osgood Street in central Methuen. Two of the sites, 4 Gleason Street and 254 Broadway, were industrial sites in central Methuen dating back to the mid-1800s. The third site, 54 Osgood Street, was the scene of a fire that destroyed a building housing hazardous material.

Environmental contamination associated with the three former industrial sites in central Methuen of concern to some residents was evaluated for the potential of exposure to nearby residents and subsequent health risks. The evaluation found the opportunities for exposure to contaminants to be limited. The hazardous materials release at 54 Osgood Street was a single event in January 1994 associated with a fire that destroyed a building on the property. The release from the fire was found not to have impacted surrounding properties and exposure opportunities for residents were limited. Compared to the 1-day

event that occurred at 54 Osgood Street, the contaminants at 4 Gleason Street and 254 Broadway were potentially present for a longer period of time due to historical industrial activities at both locations. The groundwater in this area was not and is not used as a source of drinking water. No increased risks of cancer or other adverse effects were predicted assuming a nearby resident to the two sites regularly came into contact with the maximum detected concentrations of soil, surface water or sediment contaminants at the sites. The three sites have been remediated, are currently vacant, and access to them is limited.

In response to community concerns the CAP examined in this report the incidence of certain cancers for the city of Methuen. This report is descriptive in nature and therefore has certain inherent limitations. The results of a descriptive investigation cannot be used to establish a causal link between a particular risk factor (either environmental or non-environmental) and a disease outcome (Adami and Trichopoulus 2002). Neither can it determine what may have caused cancer or another disease in any one individual. However, the results can be useful in identifying areas where further public health investigations or interventions may be warranted. Despite the limitations of descriptive studies, these types of studies can help to identify patterns of risk factors that may exist, such as behaviors or opportunities for environmental exposures, in a geographic context.

Understanding that cancer is not one disease, but a group of diseases is very important. Research has shown that there are more than 100 different types of cancer, each with different causative (or risk) factors. In addition, cancers of a certain tissue type in one organ may have a number of causes. Cancer may also be caused by several factors acting over time. Tobacco use has been linked to lung, bladder, oral and pancreatic cancers. Other factors related to certain cancers may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Family history (or genetics) is an important risk factor for several cancers. In addition, some occupational exposures, such as jobs involving contact with asbestos, have been shown to increase the risk of developing cancer. Environmental contaminants have also been associated with certain types of cancers (Bang 1996; Frumkin 1995).

According to statistics from the American Cancer Society, cancer is the second leading cause of death in Massachusetts and the United States. Not only will one out of three females and one out of two males develop cancer in their lifetime, but cancer will affect three out of every four families. For this reason, cancers often appear to occur in "clusters," and it is understandable that someone may perceive that there are an unusually high number of cancer diagnoses in their surrounding neighborhoods or towns. Upon closer examination, many of these "clusters" are not unusual increases, as first thought, but are related to such factors as local population density, variations in reporting or chance fluctuations in occurrence. In other instances, the "cluster" in question includes a high concentration of individuals who possess related behaviors or risk factors for cancer. Some, however, are unusual; that is, they represent a true excess of cancer in a workplace, a community, or among a subgroup of people. A suspected cancer cluster is more likely to be a true cluster if it involves a large number of diagnoses of one type of cancer diagnosed in a relatively short time period rather than several different types diagnosed over a long period of time (i.e., 20 years), a rare type of cancer rather than common types, and a large number of diagnoses diagnosed among individuals in age groups not usually affected by that cancer. These types of clusters may warrant further public health investigation.

In general, while the city of Methuen experienced an elevated incidence in brain cancer during each of three time periods examined, the elevation was not statistically significant citywide in any of the time periods. Given that the elevations were not statistically significant, the role of chance is still a possible explanation for the higher incidence of brain cancer in Methuen. When the overall age and gender patterns of brain cancer incidence were examined in more detail, no unusual trends emerged. The brain cancer subtypes (histology distribution) for the diagnoses in each time period were also consistent with both state and national trends. There was no apparent temporal clustering of diagnoses and the number of diagnoses fluctuated from year to year, ranging from one to eight diagnoses in any year. Furthermore, no statistically significant elevations in brain cancer or childhood cancer were seen that persisted over the time periods evaluated.

At the census tract level, two census tracts experienced a statistically significant elevation in brain cancer during two different time periods. In the earliest time period, 1982-1998, in CT 2526.01 in eastern Methuen, seven diagnoses were reported when two were expected. In the next time period, this census tract had slightly fewer diagnoses than expected. In the last time period, more diagnoses occurred than expected but the difference was not statistically significant. CT 2523, in central Methuen, where the three areas of environmental concern are located, had a statistically significant elevation among males during the 1996-2002 time period. Brain cancer occurred about as expected in males during 1982-1988 and was slightly elevated in males during 1989-1995.

During each time period evaluated, the incidence of childhood cancers in Methuen was about what would have been expected based upon the rates of all cancers among children in the entire state. Between 1982 and 1996, no children living within a ½-mile of 4 Gleason Street or 254 Broadway were diagnosed with brain cancer. Between 1996 and 2002, there were three diagnoses of brain cancer among children living within a ½-mile of the two sites. There were no other diagnoses of brain cancer among children in the city of Methuen during this time period; approximately two children would have been expected to be diagnosed with brain cancer in Methuen as a whole. There was a fourth child diagnosed with brain cancer within a ½-mile of the two sites during the 2003-present time frame.

The number of asthma-related hospitalizations in the city of Methuen was consistently either at or below what would have been expected. If excessive pollutants were present in the air, especially in 1994, the year of the 54 Osgood Street fire, then they would be expected to exacerbate asthma in Methuen residents and send them to the hospital on a more frequent basis than other residents of Massachusetts. This would raise the number of hospitalizations for asthma-related conditions and the Standardized Hospitalization Ratio for asthma hospitalizations would be elevated. However, this was not seen with the data presented in this report.

Statistics on autoimmune disease incidence statewide and for most cites and towns throughout the Commonwealth were not available. Similar to other state health departments throughout the country, Massachusetts does not have a statewide registry to track diagnoses of autoimmune diseases such as MS and Myasthenia Gravis. However, through the new environmental public health tracking initiative, Massachusetts is generating surveillance estimates specific to the Commonwealth. Based upon MS prevalence estimates, Methuen would be expected to have approximately 60 individuals with MS.

No specific pattern of brain or childhood cancer emerged that was statistically significant and persisted over time to suggest that a common environmental factor is likely related to the incidence of these cancers. The spatial pattern of the diagnoses did not suggest a common environmental factor related to the diagnoses of brain or childhood cancer. In addition, no significant health risks were predicted upon review of the available environmental data for the three sites evaluated in this report.

VIII. Child Health Considerations

ATSDR and MDPH recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at a greater risk than adults from certain kinds of exposure to hazardous substances emitted from waste sites. 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 Methuen is discussed in Section VI (“Results of Evaluation of Health Concerns”) of this report. As discussed before, past exposure to onsite contamination at 4 Gleason Street and 254 Broadway could have been

possible for children prior to remediation of the sites in 2005 and 2006. However, it is unlikely that anyone would have had sufficient contact with soil and sediment at the two sites for a sufficient frequency and duration of time to result in health effects. Present and future exposures are not of concern because contaminated soil, sediment, or surface water have been removed. No other exposures were identified that would indicate that children are more likely than adults to be impacted by 4 Gleason Street and 254 Broadway. As with adults, the likelihood of health impacts to children from the fire at 54 Osgood Street was determined to be unlikely, based on an evaluation of fire conditions and consideration of the possibility of exposure of nearby residents.

IX. Limitations

Although data for air and runoff water at 54 Osgood Street were limited to those measurements obtained during and after the January 15, 1994 fire, the limited air data indicate that health impacts from the fire were unlikely. Soil data were also limited geographically to only those properties in close proximity to the 54 Osgood Street property. However, these data indicate that soil at these locations is not expected to pose a health threat to residents. In addition, because the highest concentrations of contaminants are typically closer to the source, residential properties located at increasing distances from the source would likely have even lower concentrations (if any) that would be unlikely to pose a threat to nearby residents.

This health consultation is an investigation that analyzes descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers is unusual. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further analyses are needed. Inherent limitations in this type of analysis and the available data make it difficult at best to determine causal relationships or synergistic roles that may have played a part in the development of individual cancers in these communities. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the diseases. Behavioral factors such as tobacco use, diet, and alcohol consumption are considered the most important risk factors for a number of cancers. Other factors

associated with cancer are socioeconomic status, reproductive factors, exposure to infectious agents (i.e., viruses) and heredity/genetics. 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 the CTs of Methuen.

X. Conclusions

- Environmental contamination associated with the three sites in central Methuen of concern to some residents was evaluated for the potential of exposure to nearby residents and subsequent health risks. The evaluation found the opportunities for exposure to contaminants to be limited. The groundwater in this area was not and is not used by anyone as a source of drinking water. No increased risks of cancer or other adverse health effects were predicted assuming a nearby resident regularly came into contact with the maximum detected concentrations of contaminants in the soil, sediment, or surface water at the three sites. The three sites were remediated, are currently vacant, and access to them is limited.

- At the citywide level, the incidence of brain cancer in Methuen was elevated for three time periods (1982-1988, 1989-1995 and 1996-2002). None of the elevations were statistically significant, meaning that chance or random fluctuation cannot be ruled out as a reason for the increased incidence.

- At the census tract level, no single Methuen CT had a consistently elevated incidence of brain cancer over the three time periods that was statistically significant.

- There were two CTs where the incidence of brain cancer was statistically significantly elevated for one of the three time periods. In CT 2526.01, during 1982-1988, an overall elevation of brain cancer was found with seven diagnoses observed when two would be expected. In the two subsequent time periods in CT 2526.01, brain cancer incidence was lower than expected during 1989-1995 and higher than expected during 1996-2002, although not statistically significantly

higher than expected. In CT 2523, during 1996-2002, the incidence of brain cancer among males was statistically significantly elevated with five diagnoses observed when approximately two would be expected. In the earlier two time periods in CT 2523, brain cancer incidence in males was about as expected during 1982-1988 and slightly higher than expected during 1989-1995, although not statistically significantly higher.

- During 1996-2002, four diagnoses of childhood cancer occurred in children in CT 2523 when approximately one would be expected. Three of the four children were diagnosed with brain cancer. Between 2003 and the present, one additional child has been diagnosed with brain cancer in CT 2523. The types of brain cancer varied among these four children. The age at diagnosis of the four children was consistent with what would be expected for the four subtypes of brain cancer. With one exception, the children who were diagnosed with brain cancer across 8 years did not live in close proximity to each other nor did their residences appear concentrated near any of the three sites. Two of the four children with brain cancer lived in close proximity to each other. However, based on limited opportunities for exposure and, given that no increased cancer risk was predicted assuming exposure to the highest detected concentrations of contaminants at the three sites, it does not appear that a common environmental factor is likely related to these diagnoses.

- Except for CT 2523 in 1996-2002, childhood cancers occurred about as expected citywide and in Methuen's census tracts. The types of cancers diagnosed were varied and the ages at diagnosis of these cancers were consistent with state and national trends.

- The number of asthma-related hospitalizations was consistently lower for the city of Methuen during 1989-2002, including 1994, when the fire at 54 Osgood Street took place.

- It was not possible to quantify the incidence of the other diseases that were mentioned on the surveys provided with the request letter from concerned residents. However these diseases generally varied in nature and different risk factors were identified for each disease. There did not appear to be a common environmental risk factor for these diseases.

ATSDR requires that one of five conclusion categories be used to summarize findings of a health consultation. These categories are as follows: (1) Urgent Public Health Hazard; (2) Public Health Hazard; (3) Indeterminate Public Health Hazard; (4) No Apparent Public Health Hazard; (5) No Public Health Hazard. A category is selected from site-specific conditions such as the degree of public health hazard based on the presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns. Therefore, based on MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to the cancer types evaluated in this analysis, ATSDR would classify 54 Osgood Street as posing no apparent public health hazard to the public in the past, present and future. Since contaminants detected in groundwater at the 4 Gleason Street and 254 Broadway sites could be drawn into potential future private wells, these sites would pose a public health hazard in the future should wells be installed in contaminated groundwater areas.

XI. Recommendations

- No specific pattern of brain or childhood cancer emerged that was statistically significant and persisted over time to suggest that a common environmental factor is likely related to the incidence of these cancers. In addition, no significant health risks were predicted upon review of the available environmental data for the three sites evaluated in this report. The MDPH recommends no further investigation of brain or childhood cancer in Methuen at this time. The BEH will, however, continue to monitor the incidence of brain cancer in Methuen and its census tracts using data routinely collected by the Massachusetts Cancer Registry.

- Upon request, the MDPH will be available to assist the Methuen Health Department in reviewing and/or modifying the testing and approval process currently in place for new well construction in the area of 4 Gleason Street and 254 Broadway to ensure that contaminated groundwater from beneath the area is not consumed in the future as drinking water by nearby residents.

XII. Public Health Action Plan

The purpose of the Public Health Action Plan is to ensure that this health consultation not only identifies potential public health hazards, but also provides a plan of action designed to mitigate and prevent adverse health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of ATSDR/MDPH to follow up on this plan to ensure that it is implemented. The public health action to be implemented by ATSDR/MDPH is as follows: MDPH will continue to monitor the incidence of brain cancer in Methuen and its census tracts using data collected routinely by the Massachusetts Cancer Registry.

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PREPARER

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

CERTIFICATION

The Health Consultation, *Evaluation of Three State-Regulated Release Sites and Brain Cancer and Childhood Cancer Incidence in Methuen, Essex County, Massachusetts, MDEP RTN 3-3661, 3-24029, 3-25262 (4 Gleason Street), 3-10447 (54 OsgoodStreet), and 3-20237 (254 Broadway),* 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 Health Consultation was initiated. Editorial review was completed by the cooperative agreement partner.

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Division of Health Assessment and Consultation
Agency for Toxic Substances & Disease Registry

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

Alan W. Yarbrough, M.S.
Team Lead, CAPEB
Division of Health Assessment and Consultation
Agency for Toxic Substances & Disease Registry

Figures

Figure 1
Areas of Environmental Concern
Methuen, Massachusetts

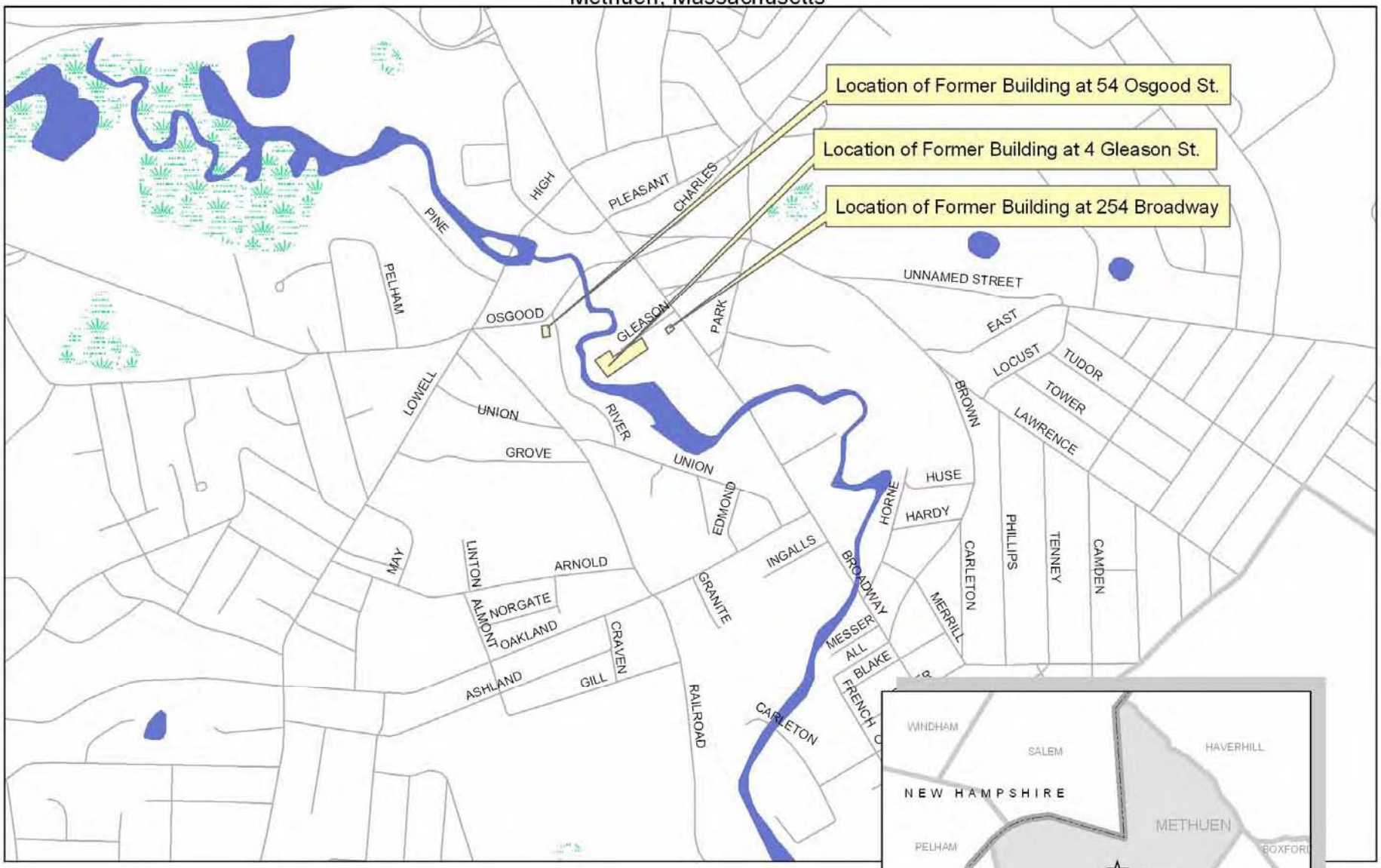
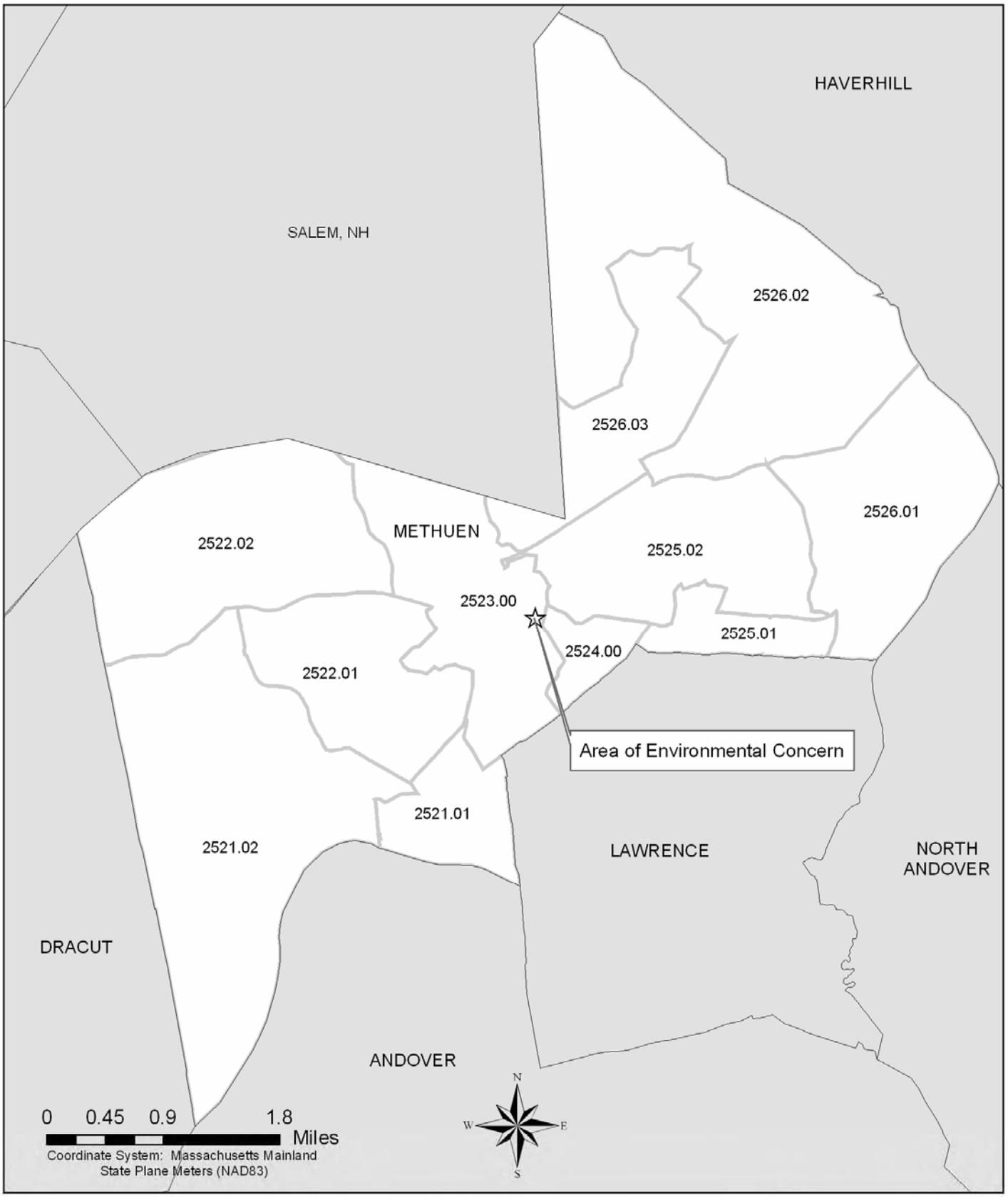


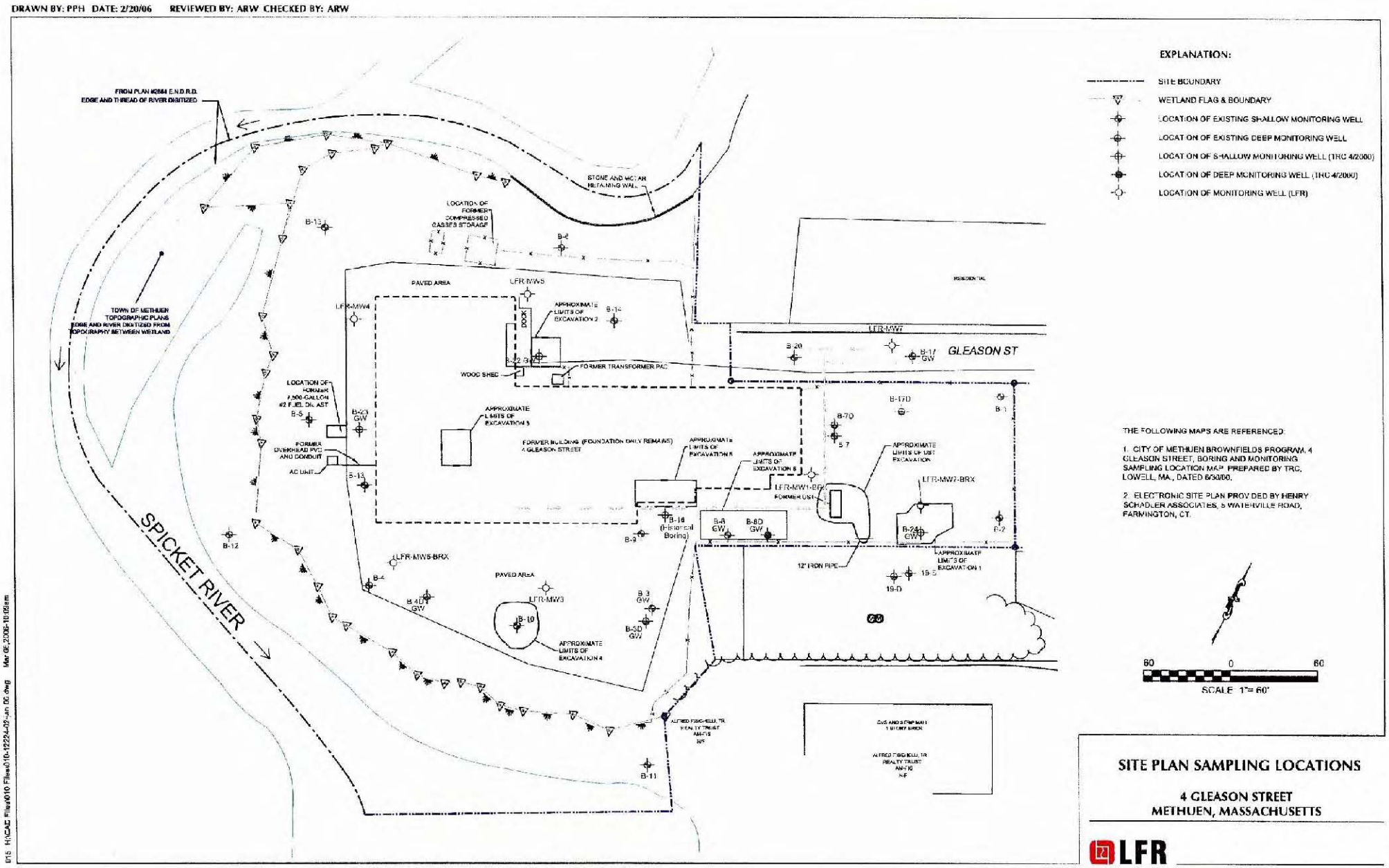
Figure 2
Location of Census Tracts in Methuen, Massachusetts



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.

Figure 3
Location of Groundwater Monitoring Wells at 4 Gleason Street
Methuen, Massachusetts



Tables

Table 1
Maximum concentrations of contaminants detected in groundwater samples at 4 Gleason Street
(samples taken from 1986 - 2005)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
1,2-Dichloroethane	Mar-93	B-19D	2	CREG = 0.4 Intermediate EMEG (child) = 2,000 Intermediate EMEG (adult) = 7000 U.S. EPA MCL & MDEP MMCL = 5
1,2-Dichloroethylene, cis-	Apr-00	B-3	87	Intermediate EMEG (child) = 3,000 Intermediate EMEG (adult) = 10,000 EPA LTHA = 70 U.S. EPA MCL & MDEP MMCL = 70
1,2-Dichloroethylene, trans-	Mar-93	B-3	110	Intermediate EMEG (child) = 2,000 Intermediate EMEG (adult) = 7,000 RMEG (child) = 200 RMEG (adult) = 700 U.S. EPA MCL & MDEP MMCL = 100
Methylene chloride	Nov-86	B-2 and B-3	16	Chronic EMEG (child) = 600 Chronic EMEG (adult) = 2,000 U.S. EPA MCL & CREG = 5
Tetrachloroethene	Mar-93	B-13	56	RMEG (child) = 100 RMEG (adult) = 400 EPA LTHA = 10 U.S. EPA MCL & MDEP MMCL = 5
Trichloroeththene	Mar-93	B-17D	670	EPA PRG = 0.028 EPA RBC = 0.026 EPA MCLG = 0 U.S. EPA MCL & MDEP MMCL = 5
Vinyl chloride	Nov-86	B-3	11	Chronic EMEG (child) = 30 Chronic EMEG (adult) = 100 CREG = 0.03 U.S. EPA MCL & MDEP MMCL = 2
Antimony	Oct-92	B-2 and B-3	5	RMEG (child) = 4 RMEG (adult) = 10 LTHA; U.S. EPA MCL & MCLG = 6
Arsenic	Apr-00	B-3	35	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3 Chronic EMEG (Adult), RMEG (adult) = 10 U.S. EPA MCL & MDEP MMCL = 10
Beryllium	Oct-92	B-12	7	Chronic EMEG (child); RMEG (child) = 20 Chronic EMEG (Adult), RMEG (adult) = 70 U.S. EPA MCL = 4

Table 1 (cont.)				
Contaminant	**Date of sample**	**Descriptive location of sample**	**Maximum concentration (ppb)**	**Drinking water comparison value (ppb)**
Cadmium	Oct-92	B-12	50	Chronic EMEG (child) = 2 Chronic EMEG (adult) = 7 RMEG (child) & US EPA MCL = 5 RMEG (adult) = 20
Chromium	Oct-92	B-12	60	Hexavalent Chromium: RMEG (child) = 30 Hexavalent Chromium: RMEG (adult) = 100 U.S. EPA MCL & MDEP MMCL = 100
Lead	May-91	B-2	96	MCLG = 0 U.S. EPA MCL & MDEP MMCL** = 15
Mercury	Aug-06	B-5	6.82	EPA PRG = 11 U.S. EPA MCL & MDEP MMCL = 2

ppb=parts per billion

Data sources:

CHE 1994, CHE 1995, TRC 2000, LFR 2003, LFR 2006

Comparison values (source organization, reference):

CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR 2006a)

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2006a)

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR 2006a)

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 2006a)

EPA PRG = EPA Region 9 Preliminary Remediation Goal for tap water (U.S. EPA 2004)
EPA RBC = EPA Region 3 Risk Based Concentration for tap water (U.S. EPA 2006a)

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 2006a)

MCLG = Maximum Contaminant Level Goal for drinking water (ATSDR 2006a)
MDEP MMCL = Massachusetts Maxiumum Contaminant Level for drinking water (MDEP 2006a)
LTHA = Lifetime Health Advisory

** This is an action level. Action must be taken if more than 10% of tap water samples exceed this value.

Table 2
Maximum concentrations of contaminants detected in soil samples at 4 Gleason Street
(samples taken from 1992 - 2005)

Contaminant	Date of sample	Descriptive location of sample (depth in feet)	Maximum concentration (ppm)	Soil Background (ppm)	Soil comparison value (ppm)
Trichloroethylene	Apr-00	B-8D (1-2)	88 (post-excavation = range 3.8 - 30)	---	EPA RBC (residential soil) = 1.6 EPA PRG (residential soil) 0.053 MDEP S-1 Soil & GW-1 = 0.3
Benzo(a)anthracene	Apr-00	B-24 (3-5)	41 (post-excavation = 2.1)	0.005 - 0.02 (rural soil) 0.169 - 59 (urban soil)	EPA RBC (residential soil) = 0.87
Benzo(b)fluoranthene	Apr-00	B-24 (3-5)	46(post-excavation = 1.6)	0.02 - 0.03 (rural soil) 15 - 62 (urban soil)	EPA RBC (residential soil) = 0.87
Benzo(a)pyrene	Apr-00	B-24 (3-5)	24(post-excavation = 1.9)	0.002 - 1.3 (rural soil) 0.165 - 0.22 (urban soil)	CREG = 0.1 EPA RBC (residential soil) = 0.087
Dibenzo(a,h)anthracene	Apr-00	B-24 (3-5)	1.7(post-excavation = ND)	---	EPA RBC (residential soil) = 0.087
Indeno(1,2,3-cd)pyrene	Apr-00	B-24 (3-5)	11 (post-excavation = 1.5)	0.01 - 0.015 (rural soil) 8-61 (urban soil)	EPA RBC (residential soil) = 0.87
C11-C22 Aromatics	Apr-00	B-24 (3-5)	660 (post-excavation = 56)	---	S-1 Soil & GW-1 = 200
Arsenic	Sept-92 and Apr-00	B-10 (5-7) and B-8D (1-2)	40 (post excavation = range 8.3 - 27)	7.4 (range: <0.1 - 73)	Chronic EMEG; RMEG (child) = 20 Chronic EMEG; RMEG (adult) = 200 CREG = 0.5
Chromium (total)	Mar-93	P-8 (2-3)	2200 (post-excavation = 11 -33)	52 (range: 1 - 1,000)	RMEG (child) (Cr VI) = 200 RMEG (adult) (Cr VI) = 2,000 EPA RBC (residential soil) (Cr VI) = 230

Note: Post-excavation soil measurements include those areas surrounding the location of the maximum concentration measured.

ppm=parts per million

Data sources:

CHE 1994, CHE 1995, TRC 2000, LFR 2003, LFR 2006

Comparison values (source organization, reference):

CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR 2006b)

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2006b)

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR 2006b)

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 2006b)
EPA PRG = EPA Region 9 Preliminary Remediation Goal for residential soil (U.S. EPA 2004)

EPA RBC = EPA Region 3 Risk Based Concentration for residential soil (U.S. EPA, U.S. EPA 2006a)

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 2006b)

S-1 Soil & GW-1 = MCP Method 1 soil category S-1 standards applicable to residential exposure scenarios. (MDEP 2006d)

Sources of background values:
Estimated arithmetic mean for the Eastern United States (east of 96th meridian). (USGS 1984)
Range of background Soil Concentrations. ATSDR Toxicological Profiles 2000 (on CD-ROM), Table 5-3. ATSDR 2005.

Table 3
Maximum concentrations of contaminants detected in groundwater samples at 254 Broadway
(samples taken 2002 & 2005)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
C5-C8 Aliphatics	Apr-05	MW-3	2,300	MDEP GW-1 = 400
C9-C10 Aromatics	Apr-05	MW-3	5,300	MDEP GW-1 = 200
Toluene	Apr-05	MW-3	770	Intermediate EMEG (child) = 200 Intermediate EMEG (adult) = 700 RMEG (child) = 800 RMEG (adult) = 3,000 EPA LTHA = 1,000 MDEP MMCL = 1,000
Naphthalene	Apr-05	MW-3	140	Intermediate EMEG (child) = 6,000 Intermediate EMEG (adult) = 20,000 RMEG (child) = 200 RMEG (adult) = 700 EPA LTHA = 100

ppb=parts per billion

Data sources:

LFR 2006

Comparison values (source organization, reference):

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR 2006a)

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 2006a)

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 2006a)

MDEP MMCL = Massachusetts Department of Environmental Protection Massachusetts Maxiumum Contaminant Level for drinking water (MDEP 2006a)
MDEP GW-1 = Massachusetts Department of Environmental Protection Massachusetts standard for either potential current or future sources of drinking water (MDEP 2006d)
LTHA = Lifetime Health Advisory

Table 4
Maximum concentrations of contaminants detected in soil samples at 254 Broadway
(samples taken from Pre-(2002) and Post- (2005) Excavation)

Contaminant	Date of sample	Descriptive location of sample (Sample depth)	Maximum concentration (ppm)	Soil Background (ppm)	Soil comparison value (ppm)
1,2,4-Trimethylbenzene*	Aug-05	LFR-7-N (10.5')	310 (post)	---	EPA PRG (residential soil) = 52
1,3,5-Trimethylbenzene*	Aug-05	LFR-7-N (10.5')	110 (post)	---	EPA PRG (residential soil) = 21
p-isopropyltoluene*	Aug-05	LFR-7-N (10.5')	4.5 (post)	---	Comparison values not available
Benzo(a)pyrene	Aug-02	LFR B10 S-4 (12-16')	0.52 (pre)	0.002 - 1.3 (rural soil) 0.165 - 0.22 (urban soil)	CREG = 0.1 EPA RBC (residential soil) = 0.087
Arsenic	Aug-02	LFR B10 S-4 (12-16')	3.9 (pre)	7.4 (range: <0.1 - 73)	Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200 RMEG (child) = 20 RMEG (adult) = 200 CREG = 0.5
C5-C8 Aliphatics †	Aug-05	LFR-7-N (10.5')	190 (post)		S-1 Soil & GW-1 = 100
C9-C10 Aromatics †	Aug-02 & Aug-05	LFR B9 S-3 (8-10') & LFR-7-N (10.5')	0.0444 (pre); 3,000 (post)		S-1 Soil & GW-1 = 100

ppm=parts per million

*Additional excavation was conducted to remove contamination at LFR-7-N in Sept. 2005 and although these contaminants were not reanalyzed, it appears that contamination was removed.

†Additional excavation was conducted to remove elevated concentrations of C5-C8 Aliphatics and C9-C10 Aromatics. Sampling during Sept. 2005 confirmed that these contaminants were not detected following this second excavation.

Data sources:

LFR, 2006b. Release Abatement Measurement (RAM) Completion Report and Class A-2 Response Action Outcome (RAO), 254 Broadway, Methuen, Massachusetts

Comparison values (source organization, reference):

CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR 2006b)

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2006b)

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 2006b)

EPA PRG = EPA Region 9 Preliminary Remediation Goal for residential soil (U.S. EPA, U.S. EPA 2004)

EPA RBC = EPA Region 3 Risk Based Concentration for tap water (U.S. EPA, U.S. EPA 2006a)

S-1 Soil & GW-1 = MCP Method 1 soil category S-1 standards applicable to residential exposure scenarios. (MDEP 2006d)

Sources of background values:
Estimated arithmetic mean for the Eastern United States (east of 96th meridian). (USGS 1984)
Range of background Soil Concentrations. ATSDR Toxicological Profiles 2000 (on CD-ROM), Table 5-3. ATSDR 2005.

Table 5
Maximum concentrations of contaminants detected in soil samples around the 54 Osgood St. Property
(samples taken during response action activities 1994)

Contaminant	Date of sample	Unpaved lot south of Site (ppm)	Restaurant parking lot west of Site (ppm)	Spicket River bank southeast of site (ppm)	Background Samples taken west of restaurant building (ppm)	Soil comparison value (ppm)
Copper	Dec-94	90.4	19.8	1590	27	Intermediate EMEG (child) = 500 Intermediate EMEG (adult) = 7,000
Cyanide	Dec-94	ND	ND	ND	ND	RMEG (child) = 1,000 RMEG (adult) = 10,000
Lead	Dec-94	39.2	97.6	166	620	EPA PRG (residential soil) = 400
Nickel	Dec-94	70.4	17.5	134	27	RMEG (child) = 1,000 RMEG (adult) = 10,000
Zinc	Dec-94	35.7	54.5	340	142	Chronic & Intermediate EMEG; RMEG (child) = 20,000 Chronic & Intermediate EMEG; RMEG (adult) = 200,000

ppm=parts per million

Data sources:

Masssachusetts Department of Environmental Protection (MDEP). 1995b. Memorandum to the File, RTN 3-10447, METHUEN-Bullseye Facility. Dated January 15, 1995.

Comparison values (source organization, reference):

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2006b)

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR 2006b)

Intermediate EMEG (child) = Environmental Media Evaluation Guide for children (i.e., for exposures between 14 days and 1 year and considers vulnerabilities of children to environmental exposures). (ATSDR 2006b)

EPA PRG = EPA Region 9 Preliminary Remediation Goal for residential soil (U.S. EPA, U.S. EPA 2004)

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 2006b)

TABLE 6
Brain Cancer Incidence
Methuen, Massachusetts
1982-1988

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	3	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.8	NC	NC -- NC
2521.02	2	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
2522.01	1	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
2522.02	1	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
2523	2	2.7	NC	NC -- NC	1	1.3	NC	NC -- NC	1	1.4	NC	NC -- NC
2524	2	1.9	NC	NC -- NC	1	0.9	NC	NC -- NC	1	1.0	NC	NC -- NC
2525.01	2	1.9	NC	NC -- NC	1	1.0	NC	NC -- NC	1	0.9	NC	NC -- NC
2525.02	2	2.4	NC	NC -- NC	1	1.3	NC	NC -- NC	1	1.1	NC	NC -- NC
2526.01	7	2.0	347	* 139 -- 714	3	1.1	NC	NC -- NC	4	0.9	NC	NC -- NC
2526.02	2	1.0	NC	NC -- NC	2	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
2526.03	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
City Total †	25	17.8	141	91 -- 208	12	9.3	129	66 -- 225	13	8.4	154	82 -- 263

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

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

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

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

TABLE 7
Brain Cancer Incidence
Methuen, Massachusetts
1989-1995

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	4	1.9	NC	NC -- NC	2	1.0	NC	NC -- NC	2	0.9	NC	NC -- NC
2521.02	4	1.5	NC	NC -- NC	3	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
2522.01	4	1.5	NC	NC -- NC	4	0.8	NC	NC -- NC	0	0.7	NC	NC -- NC
2522.02	1	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC
2523	5	3.0	169	54 -- 340	3	1.5	NC	NC -- NC	2	1.5	NC	NC -- NC
2524	1	2.0	NC	NC -- NC	0	1.0	NC	NC -- NC	1	1.0	NC	NC -- NC
2525.01	3	2.1	NC	NC -- NC	3	1.1	NC	NC -- NC	0	1.0	NC	NC -- NC
2525.02	5	2.6	192	62 -- 449	1	1.4	NC	NC -- NC	4	1.2	NC	NC -- NC
2526.01	2	2.7	NC	NC -- NC	0	1.5	NC	NC -- NC	2	1.3	NC	NC -- NC
2526.02	0	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
2526.03	1	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC
City Total	30	20.4	147	99 -- 210	16	10.9	146	83 -- 237	14	9.5	147	80 -- 247

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

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

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

TABLE 8
Brain Cancer Incidence
Methuen, Massachusetts
1996-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	1	2.0	NC	NC -- NC	0	1.1	NC	NC -- NC	1	1.0	NC	NC -- NC
2521.02	3	1.8	NC	NC -- NC	2	1.0	NC	NC -- NC	1	0.8	NC	NC -- NC
2522.01	3	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.8	NC	NC -- NC
2522.02	1	1.4	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
2523	6	3.2	186	68 -- 406	5	1.6	312	* 101 -- 728	1	1.6	NC	NC -- NC
2524	0	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
2525.01	2	2.0	NC	NC -- NC	1	1.0	NC	NC -- NC	1	1.0	NC	NC -- NC
2525.02	4	2.9	NC	NC -- NC	2	1.5	NC	NC -- NC	2	1.4	NC	NC -- NC
2526.01	6	3.3	182	67 -- 397	3	1.8	NC	NC -- NC	3	1.5	NC	NC -- NC
2526.02	3	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.7	NC	NC -- NC
2526.03	0	1.3	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
City Total	29	22.7	128	85 -- 183	16	12.0	133	76 -- 215	13	10.7	122	65 -- 208

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

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

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

TABLE 9
Childhood Cancer Incidence
Methuen, Massachusetts
1982-1988

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2521.02	1	0.9	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2522.01	3	1.0	NC	NC -- NC	1	0.5	NC	NC -- NC	2	0.5	NC	NC -- NC
2522.02	2	0.8	NC	NC -- NC	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC
2523	0	1.6	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
2524	1	1.9	NC	NC -- NC	0	1.0	NC	NC -- NC	1	0.9	NC	NC -- NC
2525.01	2	0.9	NC	NC -- NC	2	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2525.02	0	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
2526.01	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
2526.02	2	0.8	NC	NC -- NC	1	0.4	NC	NC -- NC	1	0.4	NC	NC -- NC
2526.03	0	0.6	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.3	NC	NC -- NC
City Total †	11	11.4	96	48 -- 172	6	6.2	97	35 -- 211	5	5.3	95	31 -- 222

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

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

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

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

TABLE 10
Childhood Cancer Incidence
Methuen, Massachusetts
1989-1995

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	1	0.8	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC
2521.02	1	1.0	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC
2522.01	3	0.9	NC	NC -- NC	3	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
2522.02	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.3	NC	NC -- NC
2523	2	1.5	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
2524	2	2.3	NC	NC -- NC	1	1.3	NC	NC -- NC	1	1.0	NC	NC -- NC
2525.01	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2525.02	3	1.1	NC	NC -- NC	1	0.6	NC	NC -- NC	2	0.5	NC	NC -- NC
2526.01	0	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
2526.02	1	1.0	NC	NC -- NC	0	0.5	NC	NC -- NC	1	0.5	NC	NC -- NC
2526.03	0	0.7	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC
City Total †	13	11.9	109	58 -- 186	8	6.7	119	51 -- 234	5	5.2	96	31 -- 224

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

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

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

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

TABLE 11
Childhood Cancer Incidence
Methuen, Massachusetts
1996-2002

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
2521.01	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2521.02	3	1.2	NC	NC -- NC	2	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC
2522.01	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
2522.02	1	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC
2523	5	1.6	309	100 -- 723	4	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC
2524	0	1.8	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.8	NC	NC -- NC
2525.01	1	1.0	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.5	NC	NC -- NC
2525.02	1	1.2	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC
2526.01	0	1.7	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.8	NC	NC -- NC
2526.02	1	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.6	NC	NC -- NC
2526.03	0	0.9	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC
City Total †	12	13.4	90	46 -- 157	8	7.2	111	48 -- 219	4	6.2	NC	NC -- NC

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

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

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

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

TABLE 12
Asthma Hospitalizations by Year
Methuen, Massachusetts

Year	Total						Males						Females					
	Obs	Exp	SHR		95% CI		Obs	Exp	SHR		95% CI		Obs	Exp	SHR		95% CI	
1989	164	174.7	94		80 --	109	72	67.4	107		84 --	134	92	107.6	86		69 --	105
1990	165	187.9	88		75 --	102	70	71.4	98		76 --	124	95	117.0	81	*	66 --	99
1991	213	209.7	102		88 --	116	83	80.3	103		82 --	128	130	129.9	100		84 --	119
1992	196	217.3	90		78 --	104	79	81.3	97		77 --	121	117	136.2	86		71 --	103
1993	233	238.1	98		86 --	111	94	88.3	107		86 --	130	139	150.0	93		78 --	109
1994	176	212.5	83	*	71 --	96	74	75.1	98		77 --	124	102	137.3	74	*	61 --	90
1995	163	212.5	77	*	65 --	89	54	73.5	74	*	55 --	96	109	138.9	78	*	64 --	95
1996	163	217.1	75	*	64 --	88	70	73.3	96		74 --	121	93	143.7	65	*	52 --	79
1997	195	221.2	88		76 --	101	61	74.7	82		62 --	105	134	146.2	92		77 --	109
1998	222	227.8	97		85 --	111	83	74.7	111		89 --	138	139	152.8	91		76 --	107
1999	179	244.6	73	*	63 --	85	50	79.2	63	*	47 --	83	129	165.5	78	*	65 --	93
2000	190	268.6	71	*	61 --	82	42	86.1	49	*	35 --	66	148	183.1	81	*	68 --	95
2001	217	288.4	75	*	66 --	86	71	92.3	77	*	60 --	97	146	196.9	74	*	63 --	87
2002	233	310.5	75	*	66 --	85	77	101.7	76	*	60 --	95	156	209.5	74	*	63 --	87

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

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

Data Source: Massachusetts Division of Health Care Finance and Policy, Inpatient Hospital Discharge Database.

Appendices

Appendix A

Site Inventory List
Precise Circuit/Bullseye Facility
Methuen, Massachusetts

Site Inventory List
Precise Circuit/Bullseye Facility
Methuen, Massachusetts

Number of Drums	Contents of Drums
2	Waste Ammonium Hydroxide & Water
1	Waste Sulfuric Acid & Water
2	Waste Potassium Hydroxide
6	Non-Hazardous Wastewater
15	Metal Hydroxide Solids
9	Fluoboric Acid/Nitric Acid
3	Hydrochloric Acid
5	Waste Oil
4	Hazardous Liquid
1	Nitric Acid
1	Speedy Dry with Ammonia

Source: Emergency Response Documentation Report, Precise Circuit/Bullseye Facility Methuen, MA, 15-19 January 1994. Prepared by Roy F. Weston, Inc.

Appendix B

Cancer Incidence Coding Definitions

APPENDIX B

ICD CODES USED FOR THIS REPORT

Cancer Site / Type	ICD-O-3[2]	
	Primary Site Codes	**Histology Type Codes[3]**
Brain & Other Nervous System	C70.0 - C72.9	all except 9590 - 9989
Breast	C50.0 - C50.9	all except 9590 - 9989
Bronchus & Lung	C34.0 - C34.9	all except 9590 - 9989
Cervix Uteri	C53.0 - C53.9	all except 9590 - 9989
Colon/Rectum	C18.0 - C18.9, C19.9, C20.9, C26.0	all except 9590 - 9989
Corpus Uteri & Uterus, NOS	C54.0 - C54.9, C55.9	all except 9590 - 9989
Esophagus	C15.0 - C15.9	all except 9590 - 9989
Hodgkin's Disease (Hodgkin Lymphoma)	C00.0 - C80.9	includes 9650 – 9667
Kidney & Renal Pelvis	C64.9, C65.9	all except 9590 - 9989
Larynx	C32.0 - C32.9	all except 9590 - 9989
Leukemia	C00.0 - C80.9	includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964
	C42.0, C42.1, C42.4	includes 9823, 9827
Liver and Intrahepatic Bile Ducts	C22.0, C22.1	all except 9590 - 9989
Melanoma of Skin	C44.0 - C44.9	includes 8720 - 8790
Multiple Myeloma	C00.0 - C80.9	includes 9731, 9732, 9734
Non-Hodgkin('s) Lymphoma	C00.0 - C80.9	includes 9590 - 9595, 9670 – 9729
	all sites except C42.0, C42.1, C42.4	includes 9823, 9827

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

[3] Only invasive cancers (those with invasive behaviors) are included in this report.

Cancer Site / Type	ICD-O-3	
	Primary Site Codes	**Histologic Type Codes**
Multiple Myeloma	C00.0 - C80.9	includes 9731, 9732, 9734
Non-Hodgkin('s) Lymphoma	C00.0 - C80.9	includes 9590 - 9595, 9670 – 9729
	all sites except C42.0, C42.1, C42.4	includes 9823, 9827
Oral Cavity & Pharynx	C00.0 - C14.8	all except 9590 - 9989
Ovary	C56.9	all except 9590 - 9989
Pancreas	C25.0 - C25.9	all except 9590 - 9989
Prostate	C61.9	all except 9590 - 9989
Stomach	C16.0 - C16.9	all except 9590 - 9989
Testis	C62.0 - C62.9	all except 9590 - 9989
Thyroid	C73.9	all except 9590 - 9989
Urinary Bladder	C67.0 - C67.9	all except 9590 - 9989

Appendix C

Risk Factor Information for Brain and Central Nervous System Cancers

Brain and central nervous system (CNS) tumors can be either malignant (cancerous) or benign (non-cancerous). Primary brain tumors (i.e., brain cancer) comprise two main types: gliomas and malignant meningiomas. Gliomas are a general classification of malignant tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. Meningiomas arise from the meninges, which are tissues that surround the outer part of the spinal cord and brain. Although meningiomas are not technically brain tumors, as they occur outside of the brain, they account for about 25% of all reported primary brain tumors and the majority of spinal cord tumors. The majority of meningiomas (about 85%) are benign and can be cured by surgery. In addition to these main types, there are a number of rare brain tumors, including medulloblastomas, which develop from the neurons of the cerebellum and are most often seen in children. Also, the brain is a site where both primary and secondary malignant tumors can arise; secondary brain tumors generally originate elsewhere in the body and then metastasize, or spread, to the brain (ACS 2006a). The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with primary brain cancer (including cancers of the central nervous system, or spinal cord) and approximately 12,820 people (7,260 men and 5,560 women) will die from this disease in 2006 (ACS 2006).

Brain and spinal cord cancers account for over 20% of malignant tumors diagnosed among children aged 0-14 (ACS 2006b). About half of all childhood brain tumors are astrocytomas and 25% are primitive neuroectodermal tumors (PNET), which spread along the spinal cord and the meninges (ACS 2006b). After a peak in incidence during childhood (generally under 10 years of age), the risk of brain cancer increases with age from age 25 to age 75. In adults, the most frequent types of brain tumors are astrocytic tumors (mainly astrocytomas and glioblastoma multiforme). Incidence rates are higher in males than in females for all types. In general, the highest rates of brain and nervous system cancer tend to occur in whites. However, this varies somewhat by type; the incidence of gliomas is lower among black men and women than whites, but for meningiomas, the reverse is true (Preston-Martin and Mack 1996).

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

The most established risk factor (and only established environmental risk factor) for brain tumors (either cancerous or non-cancerous) is high-dose exposure to ionizing radiation (i.e., x-rays and gamma rays). Most radiation-induced brain tumors are caused by radiation to the head from the treatment of other cancers (ACS 2006a). Meningiomas are the most common type of tumors that occur from this type of exposure, but gliomas may also occur (Preston-Martin and Mack 1996). Among adults, the risk of developing meningiomas has been associated with full-mouth dental x-rays taken decades ago when radiation doses were higher than today. Although the relationship between low-dose radiation exposure and increased risk of brain tumors has been debated in several studies,

prenatal exposure from diagnostic x-rays has been related to an increase in childhood brain tumors (Preston-Martin and Mack 1996).

In recent years, there has been increasing public concern and scientific interest regarding the relationship of electromagnetic fields (EMF) to brain cancer. However, results from recent epidemiological investigations provide little or no evidence of an association between residential EMF exposure (e.g., from power lines and home appliances) and brain tumors (Kheifets 2001). Studies also suggest that the use of handheld cellular telephones is not associated with an increased risk of primary brain cancer (Muscat et al. 2000). However, given the relatively recent use of cellular phones, evidence is preliminary and few studies have been conducted.

Other environmental factors such as exposure to vinyl chloride (used in the manufacturing of some plastics) and aspartame (a sugar substitute) have been suggested as possible risk factors for brain cancer but no conclusive evidence exists implicating these factors (ACS 2006a). Although some occupational studies have suggested that electrical and electric utility workers may be at a slightly increased risk of brain cancer, these studies have important limitations, such as exposure misclassifications and a lack of dose-response relationships (Kheifets 2001). Some researchers have also reported an increased risk of brain tumors in adults among veterinarians and farmers. Exposures to farm animals and pets have been considered as possible risk factors because of their association with bacteria, pesticides, solvents, and certain animal oncogenic (cancer-related) viruses (Yeni-Komshian and Holly 2000). However, the relationship between farm life and brain cancer remains controversial.

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

The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A study concluded that an increased risk of pediatric brain tumor may

be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin 2001). However, the role of nitrites and cured meats in the development of brain cancer remains controversial (Blot et al. 1999; Bunin 2000). Because most people have continuous, low level exposure to N-nitroso compounds throughout their lives, further studies, especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin 1996).

Injury to the head has been suggested as a possible risk factor for later development of brain tumors but most researchers agree that there is no conclusive evidence for an association (ACS 2006a). Head trauma is most strongly associated with the development of meningiomas compared with other types of brain tumor. Several studies have found an increased risk in women with histories of head trauma; in men who boxed; and in men with a previous history of head injuries. Gliomas are the most common type of childhood brain tumor and have been positively associated with trauma at birth (e.g., Cesarean section, prolonged labor, and forceps delivery). However, other studies have found no association (Preston-Martin and Mack 1996).

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-Martin and Mack 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS 2006b). Of those afflicted with the disease, about 5-10% will develop a central nervous system tumor (Preston-Martin and Mack 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS 2006b). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack 1996).

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Appendix D

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

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

General Terms

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

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

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

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

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

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

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

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

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

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

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

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

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

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

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

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

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

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

CAP [see Community Assistance Panel.]

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

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

Carcinogen
A substance that causes cancer.

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

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

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

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

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

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

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

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

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

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

Completed exposure pathway [see exposure pathway].

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

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

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

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

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

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

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

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

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

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

DOD
United States Department of Defense.

DOE
United States Department of Energy.

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

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

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

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

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

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

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

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

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), non-cancerous 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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