ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Evaluation of Environmental Concerns and Cancer Incidence, 2000-2003,
Related to the Woburn Landfill in Woburn, Middlesex County, Massachusetts

WOBURN SANITARY LANDFILL
FACILITY ID NUMBER: SL0347.001
EPA FACILITY ID: MAD980504153

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

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

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

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PUBLIC HEALTH ASSESSMENT

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Prepared by:

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List of Abbreviations

ACS	American Cancer Society
AML	Acute Myeloid Leukemia
ALL	Acute Lymphoid Leukemia
ATSDR	Agency for Toxic Substances and Disease Registry
BEH	Bureau of Environmental Health
CAP	Community Assessment Program
CERCLA	Comprehensive Environmental Response, Compensation, & Liability Act
CI	Confidence Interval
CLL	Chronic Lymphoid Leukemia
CML	Chronic Myeloid Leukemia
CNS	Central Nervous System
CREG	Cancer Risk Evaluation Guide
CT	Census Tract
EMEG	Environmental Media Evaluation Guide
EPH	Extractable Petroleum Hydrocarbons
GIS	Geographic Information System
IARC	International Agency for Research on Cancer
ICD-O	International Classification of Diseases for Oncology
MBTA	Massachusetts Bay Transportation Authority
MG/KG	Milligrams per Kilogram
MTBE	Methyl Tert Butyl Ether
MCR	Massachusetts Cancer Registry
MDEP	Massachusetts Department of Environmental Protection
MDPH	Massachusetts Department of Public Health
MRL	Minimum Risk Level
MWRA	Massachusetts Water Resources Authority
NDMA	Nitrosodimethylamine
NPDWSA	Non-Potential Drinking Water Source Area
NPL	National Priorities List
PCB	Polychlorinated Biphenyls
PAH	Polycyclic Aromatic Hydrocarbons
PPB	Parts Per Billion
PPM	Parts Per Million
PRG	Preliminary Remediation Goal
RBC	Risk-Based Concentration
RMEG	Reference Dose Media Evaluation Guide
SIR	Standardized Incidence Ratio
SVOC	Semi-Volatile Organic Compound
TCE	Trichloroethylene
TPH	Total Petroleum Hydrocarbons
UG/M	Micrograms per Meter
USEPA	United States Environmental Protection Agency
USGS	United States Geological Survey
VOC	Volatile Organic Compound

I. SUMMARY

At the request of the Woburn Neighborhood Association, concerned residents, and State Representative James R. Miceli, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of possible environmental exposures and a review of cancer incidence in relation to the Woburn Landfill in Woburn, Massachusetts. The landfill is located off Merrimac Street and west of New Boston Street in North Woburn. The landfill began accepting waste for disposal around 1966 and officially ceased operations in 1986. Residential, commercial and industrial waste, including construction and demolition debris and by-products of gelatin manufacturing, were accepted for disposal. Prior to waste disposal activities, a gravel pit was operated on the Woburn Landfill property. Closure activities began in 1999 and were completed in late 2003, followed by the development of a post-closure monitoring and maintenance plan for the Woburn Landfill. MDEP accepted the Final Closure Report on the landfill in 2005. This evaluation was initiated based on community concerns about possible environmental exposures and potential adverse health effects for residents living near the landfill. This project was conducted under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct site-specific activities in Massachusetts.

The investigation reviewed available environmental data for Woburn Landfill and considered potential ways that people may come into contact with contaminants detected in groundwater, surface water, air, and wetland sediment. The evaluation also looked at the pattern of cancer in Woburn census tract (CT) 3336, where the Woburn Landfill is located, and focused in particular on residential neighborhoods in Woburn closest to the landfill. The cancer incidence analysis is an update to *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999* (MDPH 2004).

Five cancer types were evaluated in this investigation: bladder cancer, brain and central nervous system (CNS) cancer, kidney cancer, leukemia, and lung and bronchus cancer. The cancer types were chosen for analysis based on potential associations with

contaminants of concern identified at the Woburn Landfill and community concerns in neighborhoods surrounding the Woburn Landfill. Using data from the Massachusetts Cancer Registry (MCR), cancer incidence rates from 2000 to 2003 were calculated for Woburn CT 3336. Available information about risk factors, including environmental factors, related to the development of cancer was also considered.

In general, four of the five cancer types evaluated for Woburn CT 3336 occurred near or below expected rates from 2000 to 2003, the period for which the most recent and complete cancer incidence data were available from the MCR at the time of analysis. Bladder cancer was statistically significantly elevated among males and females evaluated together. A review of available risk factor information revealed that seven of the nine individuals diagnosed with bladder cancer were current or former smokers, which is a major risk factor for bladder cancer.

While three diagnoses of leukemia among individuals 0-19 years of age were observed, and less than one was expected, it is important to note that no diagnoses were observed in the previous 5-year time period, 1995–1999. Also, a review of the geographic distribution of these three cases of leukemia indicated that the cases were approximately 0.5 miles from each other and approximately 1 mile from the Woburn Landfill.

A review of the geographic distribution of individuals diagnosed with the five cancer types in Woburn CT 3336 revealed no apparent spatial patterns at the neighborhood level. Further review of the geographic pattern of bladder cancer did not indicate a concentration or an atypical distribution of diagnoses. Further, no unusual geographic patterns emerged as a result of evaluating the residential information for individuals diagnosed with any of the five cancer types in the vicinity of the Woburn Landfill or in any other area of Woburn CT 3336.

In addition to a review of cancer incidence data, available environmental data were reviewed and potential ways that people may come into contact with contaminants detected in groundwater, surface water, air, and wetland sediment related to the Woburn Landfill were explored. Past and present exposures to groundwater contaminants via drinking water were determined to be unlikely because nearby residents consume

drinking water from sources in southern Woburn and the Quabbin Reservoir in western Massachusetts. In addition, there are no known private wells used for drinking water in the area. It is unlikely that groundwater contaminants detected at the Woburn Landfill would reach the Zone II groundwater protection area for Woburn's municipal wells because it is located over 2 miles south of the landfill. For these reasons, exposure to contaminated groundwater in drinking water would not be expected in the past, present, or future. However, future exposures to polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs) and metals detected in onsite groundwater could be possible if new offsite private drinking water wells are installed in the direction of groundwater flow from the landfill. Regarding the potential for indoor air exposures to VOCs, based on the levels detected in onsite groundwater, conservative (i.e. health-protective) indoor air concentrations predicted by the Johnson-Ettinger model (USEPA 2004a, 2004b), groundwater flow direction, and distance to nearby homes, it is unlikely that groundwater contaminants would present an exposure concern for homes down-gradient of the site.

Intermittent exposures to contaminants in onsite surface water and wetland sediment may be possible for individuals trespassing on the Woburn Landfill or visiting nearby wetlands in the past, present, and future. However, based on the concentrations detected and the frequency and duration of contact expected, it is unlikely that intermittent exposures would result in adverse health effects.

Based on criteria established by ATSDR, the Woburn Landfill would be classified as posing no apparent public health hazard in the past or present and as posing a public health hazard in the future should private wells be installed in areas of contaminated groundwater. Based on the information reviewed in this evaluation, including available environmental data for the Woburn Landfill and risk factor information for individuals diagnosed with cancer, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in Woburn CT 3336 from 2000 to 2003.

II. INTRODUCTION

At the request of the Woburn Neighborhood Association, concerned residents, and State Representative James R. Miceli, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of possible environmental exposures and a review of cancer incidence in relation to the Woburn Landfill. This evaluation was initiated based on community concerns about possible environmental exposures and potential adverse health effects for residents of nearby neighborhoods from contaminants associated with the landfill. The Woburn Landfill, which operated from 1966 to 1986, and surrounding neighborhoods are located immediately south of the Woburn–Wilmington town line (see Figures 1 and 2). This project was conducted under a cooperative agreement with the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct public health assessments in Massachusetts.

In addition to a review of available environmental data and an evaluation of potential environmental exposures, an analysis of cancer incidence was conducted. The cancer incidence analysis is an update to *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999* in which MDPH concluded that the majority of the cancer types evaluated in Woburn CT 3336 occurred approximately at or below expected rates during the 5-year time period 1995–1999 (MDPH 2004). That report demonstrated that incidence ratios for two cancer types (i.e., bladder cancer and lung and bronchus cancer) were statistically significantly elevated above expected rates for 1995–1999. Data regarding smoking history for individuals diagnosed with these cancer types suggested that smoking likely played a role in the incidence of bladder and lung and bronchus cancer among residents in CT 3336 (MDPH 2004).

In order to update the 2004 report, cancer incidence rates for five cancer types were calculated for Woburn census tract (CT) 3336 during the years 2000–2003, the time period for which the most recent and complete cancer incidence data were available from the Massachusetts Cancer Registry (MCR) at the time of analysis. The five cancer types (bladder cancer, brain and central nervous system (CNS) cancer, kidney cancer,

leukemia, and lung and bronchus cancer) were chosen for evaluation based on potential associations with contaminants of concern identified at the Woburn Landfill and based on community concern about bladder cancer and leukemia.

The City of Woburn is divided into seven smaller geographic areas or census tracts (CTs): CT 3331, CT 3332, CT 3333, CT 3334, CT 3335.01, CT 3335.02, and CT 3336. The Woburn Landfill and the nearby residential areas are all located in CT 3336 in northern Woburn (Figure 1). A census tract is a smaller geographic subdivision of a city or town that is designated by the United States Census Bureau. CTs usually contain between 2,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (U.S. DOC. 1990, 2000). Because age-group and gender-specific population information is necessary to calculate cancer incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated.

The City of Woburn is located approximately 12 miles northwest of Boston and is bordered by the towns of Wilmington to the north, Burlington and Lexington to the west, Winchester to the south, and Reading and Stoneham to the east. Woburn is primarily a suburban community and comprises an area of 12.7 square miles with an average of 2,940 residents per square mile (U.S. DOC 2002). The 2000 United States Census reports a total of 37,258 residents in the City of Woburn (U.S. DOC 2002). Census tract locations and boundaries in Woburn are shown in Figure 1.

III. OBJECTIVES

The specific objectives of this investigation were as follows:

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

- To evaluate the incidence rates of five cancer types (bladder cancer, brain and CNS cancer, kidney cancer, leukemia, and lung and bronchus cancer) in CT 3336, where the Woburn Landfill is located, to determine if cancer is occurring more or less often than expected;

- To evaluate the geographic distribution of individuals diagnosed with cancer in Woburn CT 3336 to determine if there are any unusual patterns in relation to areas of potential environmental concern;

- To review available descriptive information from the MCR for individuals diagnosed with cancer in Woburn CT 3336 to see if there are any particular characteristics related to known or suspected risk factors, including environmental factors, for developing these diseases; and

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

- To make recommendations for mitigating possible exposure pathways related to the Woburn Landfill.

IV. BACKGROUND AND COMMUNITY ENVIRONMENTAL CONCERNS

The Woburn Neighborhood Association, concerned residents, and State Representative James R. Miceli expressed concerns about odors and gas emissions from the Woburn Landfill as well as concerns about soil, groundwater, and surface water contamination from landfill waste. Residents have expressed particular concern about the possibility of contamination from the Woburn Landfill impacting private wells. In order to address these community concerns, the MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) to obtain and review available environmental information pertaining to the Woburn Landfill. In addition, information regarding other potential environmental sources located in the area and listed with MDEP as a hazardous release or spill location was reviewed.

This public health assessment titled "*Evaluation of Environmental Concerns and Cancer Incidence, 2000 – 2003, Related to the Woburn Landfill in Woburn, Middlesex County,*

Massachusetts" was released on November 26, 2007, for a public comment period ending on January 8, 2008. Public comments were received by the MDPH and are addressed in Appendix D.

A. Woburn Landfill

The Woburn Landfill is located at 202 Merrimac Street in Woburn, Middlesex County, Massachusetts and abuts the Woburn–Wilmington town line to the north (Figure 2). The property consists of approximately 61 acres, of which about 37 acres contain waste. The landfill is currently surrounded by a 6-foot chain link fence topped with barbed wire that restricts access to the property. The fence is currently in good condition and the post-closure monitoring calls for bi-annual visual inspection to ensure its continued integrity. To the north, the landfill is bordered by the Olin Chemical facility at 51 Eames Street in Wilmington, Massachusetts. To the east, the landfill is bordered by the Massachusetts Bay Transit Authority (MBTA) Right of Way for the Lowell-Boston Commuter Rail. Beyond the commuter rail line, to the east, are general commercial/industrial areas that include the Industri-Plex property at the intersection of Commerce Way and Atlantic Avenue. To the west and southwest, the landfill is bordered by wetland areas that formerly contained landfill waste and are located outside of the chain-link security fence. Beyond the wetlands are residential and commercial areas. To the south, the landfill is bordered by undeveloped land zoned for commercial or light industrial uses, wetlands, and the Blox Chemical property at 100 Ashburton Avenue in Woburn. The landfill is traversed by two Boston Edison electric power line easements, both on the southern portion of the site; one traverses in a south to northwesterly direction, while the other traverses the landfill in a south to northeasterly direction toward New Boston Street.

Prior to waste disposal activities, the Woburn Landfill property was operated as a gravel pit. The landfill began accepting waste for disposal around 1966, beginning with the relocation of tens of thousands of cubic yards of refuse material from a former dump used by the City of Woburn to allow for the development of the Woburn Industrial Park in northeast Woburn (MDEP 2002). Residential, commercial and industrial waste, including construction and demolition debris and by-products of gelatin manufacturing, were accepted for disposal. A review of the available information indicated that the

southeastern portion of the landfill appears to have been owned and used by the Merrimac Chemical Company, which was listed as a former owner of the Industri-Plex property (MDEP 2002, USEPA 2006a). The City of Woburn assumed ownership of the property in 1975 and the landfill officially ceased operations on June 30, 1986. Available information indicates that some illegal waste disposal occurred past this date. In 1999, final closure procedures at the Woburn Landfill began and continued until completion in the fall of 2003. During the closure process a landfill gas collection and destruction system and a landfill cap (approximately 3 feet thick) were installed. A post-closure monitoring and maintenance plan was also developed to include sampling of groundwater, surface water, sediment, and landfill gas as well as inspections and repair to the vegetative cover and to the security systems (Maguire 2005). MDEP accepted the Final Closure Report on February 7, 2005. (MDEP 2002; Maguire 2005)

B. Other Potential Environmental Sources (including Massachusetts Department of Environmental Protection 21E Sites)

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), commonly known as Superfund, was enacted by the U.S. Congress in 1980. As part of the Superfund clean-up process, certain sites requiring long-term remedial response actions are placed on United States Environmental Protection Agency's (USEPA) National Priorities List (NPL). The clean-up process aims to permanently and significantly reduce the dangers associated with releases or threats of releases of hazardous substances (USEPA 2006b). The Woburn Landfill is in close proximity to one Superfund site (i.e., Blox Chemical) and two properties that are listed on the NPL (i.e., Industri-plex and Olin Chemical).

The Blox Chemical property, part of the Superfund program, is an approximately 8-acre site located 700 feet south of the Woburn Landfill at 100 Ashburton Avenue in Woburn. Historically, the property housed tannery operations, a poultry slaughterhouse, and miscellaneous other commercial/industrial operations. Previous investigations of the property indicated the presence of waste materials from former tannery operations as well as the presence of volatile organic compounds (VOCs), metals (including arsenic, cadmium, chromium, and lead), cyanide, and total petroleum hydrocarbons (TPHs).

Groundwater is reported to flow in a northeasterly direction and may discharge to a wetland. No known impacts to drinking water in the area have been reported. (USEPA 2002) The U.S. Environmental Protection Agency (USEPA) conducted sampling and released a report on the Blox Chemical property in the early 1990s and the site is currently inactive (N. Smith, U.S. EPA Region 1, personal communication, 2006).

The Industri-Plex site is a 245-acre property located immediately east of the Woburn Landfill at Commerce and Atlantic Way and is listed on the NPL. Historically, the property housed chemicals manufacturing and miscellaneous other commercial/industrial operations. Manufactured products included many types of acids, tin crystals, oxy-muriate of antimony, and arsenical pesticides. In and around the Industri-Plex site, the groundwater is contaminated with VOCs such as benzene and toluene, as well as arsenic and chromium. The soil is contaminated with heavy metals, including arsenic, chromium, and lead. Also, a pervasive "rotten egg" odor was due to hydrogen sulfide gas generated by the decay of buried animal hides from glue manufacturing wastes. Many investigations have been completed at the Industri-Plex site to determine the approach to the remediation process. Currently, long-term remedial actions are focusing on site stabilization and cleanup of groundwater contamination. (USEPA 2006c)

The Olin Chemical facility is a 53-acre property located immediately north of the Woburn Landfill at 51 Eames Street in Wilmington and is also listed on the NPL. In and around the Olin Chemical site, the groundwater is contaminated with ammonia, chloride, sodium, sulfate, chromium and N-nitrosodimethylamine (NDMA). Contaminated groundwater has migrated about 0.75 miles west of the site. Onsite soil is contaminated with trimethylpentenes, acetone, bis(2-ethylhexyl)phthalate, NDMA, pesticides, aluminum, calcium, chromium, iron, sodium, chloride, ammonia, sulfate and aromatic extractable petroleum hydrocarbon (EPH) fractions. Surface water and sediment on or near the site are contaminated with trimethylpentenes, bis(2-ethylhexyl)phthalate, chromium and petroleum hydrocarbon compounds. The Olin Chemical site was added to the NPL in 2006 and extensive work to control and contain waste and to investigate the site has occurred. No efforts have been initiated to address the groundwater contamination plume. (USEPA 2006d, 2006e)

In 1983, the Massachusetts Legislature established a statewide hazardous waste site cleanup program (the state Superfund program) under Chapter 21E of Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000). Under this legislation, MDEP administers investigation and clean-up of hazardous material and oil release sites, known as "21E sites", in the Commonwealth. The MDPH reviewed available information regarding these releases to determine whether potential environmental exposures could have played a role in the incidence of cancer in Woburn CT 3336, where the Woburn Landfill and surrounding neighborhoods are located.

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 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 in Woburn CT 3336. The large number of releases in the census tract precluded individual examination of each release in relation to patterns of cancer incidence. Therefore, MDPH focused the analysis on those releases categorized by 2-hour or 72-hour reporting categories. Releases categorized as 120-day reporting notifications and releases where reporting category information was unavailable were excluded. The 120-day reports are releases thought to be unlikely to result in human exposure to contaminants.

Hazardous material and oil releases are *potential* sources of human exposure to contamination. It is not possible to determine whether individuals residing in Woburn CT 3336 were actually exposed to contaminants without detailed information about contaminant movement through the environment, the population at risk of exposure, a

location of actual human contact with the contaminant, and evidence that the contaminant actually entered the body of persons at risk of exposure through ingestion, dermal absorption, or inhalation.

Using a geographic information system, MDPH mapped the approximate location of 2-hour and 72-hour releases for which sufficient address information was available (ESRI 2005). Of all releases in the city of Woburn from 1980-2003, approximately 18% could not be mapped due to insufficient address information. Some of these unmapped 21E sites may have addresses within CT 3336. According to the most current information, from 1980 to 2003, 120 2-hour and 72-hour releases were reported in Woburn CT 3336 (MDEP 2006b). Approximately half of the releases (56%) involved petroleum-based oil (e.g., gasoline, fuel oil, waste oil) or some combination of oil and another material (either known or unknown). The type of material was unknown for 16 (13%) of the releases. There were no releases reported at the Woburn Landfill. The full list of releases recorded as "21E sites" in Woburn CT 3336 is shown in Table 1 and are mapped on Figure 3.

The pattern of cancer in Woburn CT 3336 was reviewed in relation to these potential sources of environmental exposures and is discussed in Section VII.

V. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with the Woburn Landfill, MDPH reviewed information on file with MDEP. Environmental sampling data were available for groundwater, surface water, landfill gas, and wetland sediment 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. The screening analysis identifies maximum concentrations of contaminants detected in various types of environmental media (i.e., air, soil, water) and compares these concentrations to health-based comparison values established by the Agency for Toxic Substances and Disease Registry (ATSDR) (ATSDR 2006a, 2006b). If an ATSDR comparison value was not available for a specific chemical, the maximum detected concentration of that chemical was compared to Preliminary Remediation Goals (PRGs)

developed by the USEPA Region IX (USEPA 2004c), Risk-Based Concentrations (RBCs) developed by the USEPA Region III (USEPA 2006f), or the applicable groundwater and soil standards developed by MDEP (1997; 2006a), in that order. For compounds detected in groundwater, maximum concentrations were also compared with state and/or federal drinking water standards.

The ATSDR comparison values are specific concentrations of a chemical for air, soil, or water that are used by health assessors to identify environmental contaminants that require further evaluation. These comparison values are developed based on health guidelines and assumed exposure situations that represent conservative estimates of human exposure. Comparison values are set well below levels that are known or anticipated to result in adverse health effects. Chemical concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, chemical concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a chemical to impact one's health, it must not only be present in the environmental media, but one must also come in contact with the chemical. Therefore, if a concentration of a chemical is greater than the appropriate comparison value, the potential for exposure to the chemical should be further evaluated to determine whether exposure is occurring and whether health effects might be possible as a result of that exposure. The factors related to exposure which are unique to the specific situation under investigation need to be considered to determine if an adverse health effect from this chemical could occur.

ATSDR has compiled levels of metals and polycyclic aromatic hydrocarbons (PAHs) that are considered normal for soil of urban and suburban communities (ATSDR 1995). The United States Geological Society (USGS) has identified levels of metals that are considered typical for soil in the eastern United States (Shacklette and Boerngen 1984). These levels are "background" and are used along with comparison values for both metals and PAHs in this analysis.

A. Groundwater

Depth to groundwater at the Woburn Landfill property is between 2.75 and 12.80 feet below grade, according to groundwater monitoring wells sampled in 1997, and between 5 and 20 feet below grade, according to groundwater monitoring wells sampled in 2004. Groundwater flow is in a southeasterly direction (MDEP 2002, Maguire 2005). Groundwater at the Woburn Landfill is not used as a source of drinking water, and there are no known private drinking water wells in nearby Woburn neighborhoods (Maguire 2005, D. Dulong, Woburn Water Department, personal communication, 2006).

According to Mass GIS, Massachusetts's Office of Geographic and Environmental Information, the southern portion of the Woburn Landfill is situated over a medium yield aquifer and is abutted by a Non-Potential Drinking Water Supply Area (NPDWSA). A medium or high yield aquifer is a designation given by the U.S. Geological Survey (USGS) and means that it is a Potentially Productive Aquifer, but not necessarily a Potential Drinking Water Source Area (310 CMR 40.0000). A municipality or private party may petition MDEP to have an area designated as a NPDWSA for a number of reasons, including overlying land use, population density, sufficient water from other sources, and/or contamination (310 CMR 40.0000). A majority of the nearest high yield aquifer, which is approximately 500 feet southeast of the landfill, has been designated as an NPDWSA. This aquifer and the nearest MDEP Zone II protection area lie approximately 0.25 miles to the northwest. A Zone II protection area is defined as the area of an aquifer which contributes water to a drinking water well under the most severe pumping and recharge conditions that can be realistically anticipated (MDEP 1995). The nearest public drinking water wells are located in Wilmington, approximately 1 mile northwest of the landfill. These public wells formerly served the Town of Wilmington and are currently inactive due to contamination from the Olin Chemical site in Wilmington (USEPA 2006e). Since groundwater from the Woburn Landfill flows in a southeasterly direction, areas northwest of the landfill, including wells in Wilmington, would not be impacted by Woburn Landfill groundwater contamination.

Twelve groundwater monitoring wells currently exist onsite at the Woburn Landfill and were sampled in 1997 and 2004. The approximate locations of the 12 existing wells are

depicted in Figure 4. Groundwater sampling was conducted on four occasions in 1997 and on one occasion in 2004. There were also seven former wells that were used to monitor groundwater in 1988. All older wells were either destroyed during closure activities or replaced by new monitoring wells (ENSR 1998; Maguire 2005). Seven samples were collected in 1988, 23 samples were collected in 1997, and seven samples were collected in 2004. Thirty-three samples were analyzed for most volatile organic compounds (VOCs) and metals, 30 samples were analyzed for landfill indicator parameters (i.e. pH, temperature, alkalinity, total dissolved solids, dissolved oxygen, chemical oxygen demand, sulfate, chloride, iron, manganese, specific conductance), 26 samples were analyzed for most semi-volatile organic compounds (SVOCs), 20 samples were analyzed for polychlorinated biphenyls (PCBs) and pesticides, and 16 samples were analyzed for cyanide. All of the aforementioned wells were used solely to monitor groundwater near the landfill and are not used for any other drinking or non-drinking water purposes.

Samples from the onsite groundwater monitoring wells had detectable concentrations of some VOCs, SVOCs, and metals. The maximum concentrations of contaminants measured in groundwater at the Woburn Landfill were compared to drinking water comparison values to help determine if further evaluation was necessary.

Of the contaminants detected in groundwater monitoring wells at the Woburn Landfill, 20 exceeded comparison values and, therefore, required further evaluation in this report. Table 2 summarizes the maximum concentrations of each of these contaminants as well as their comparison values. Two contaminants detected in groundwater did not have comparison values and are also discussed in this report.

Of the five VOCs detected in groundwater at the Woburn Landfill, trichloroethylene (TCE), 1,4-dichlorobenzene, and methyl-tert-butyl ether (MTBE) were each detected once in samples analyzed for VOCs and all fall with the range of comparison values (see Table 2). MBTE was the only VOC of concern detected at monitoring wells located closest to residential areas to the west and southwest of the landfill (Figure 4). MTBE is a gasoline additive introduced in the 1980s and used to reduce air pollution. MTBE

could enter groundwater from a number of sources including spills or leaks from storage containers, especially near manufacturing sites, pipelines, and shipping facilities, or leakage from underground storage tanks, such as tanks at gasoline filling stations (ATSDR 1996).

Two other VOCs, benzene and chloroethane, were detected above comparison values at monitoring wells located on the southeastern and eastern edges of the landfill (Figure 4). The maximum concentrations of benzene and chloroethane were both detected in 1997 at MW-14 (Table 2).

Several SVOCs detected in groundwater monitoring wells at the Woburn Landfill were retained for further evaluation. The maximum detected concentration of N-nitrosodiphenylamine (23 ppb) was found in 1997 in MW-14, which is located at the southeast corner of the landfill.

Three SVOCs, carbazole, dibenzofuran, and 4,6-dinitro-2-methylphenol, were detected only at monitoring well MW-13 on the northeast boundary of the Woburn Landfill and exceeded their respective comparison values (see Table 2).

4-Nitrosodiphenylamine was detected once in 1997 at monitoring well MW-14 in the southeast corner of the Woburn Landfill and was not detected in 2004. 4-Chloro-3-methylphenol and pentachlorophenol were both detected at MW-13 and MW-14 during 1997. Comparison values are not available for 4-nitrosodiphenylamine or 4-chloro-3-methylphenol. Pentachlorophenol (50 ppb) was detected above the CREG of 0.3 ppb. Historically, pentachlorophenol was one of the most widely used biocides in the United States, while more recently it has been used industrially as a wood preservative for power line poles, cross arms, and fence posts (ATSDR 2001a). The SVOC dibenzofuran, detected along with pentachlorophenol at well location MW-13, is a known impurity present in pentachlorophenol (ATSDR 2001a). These compounds were not detected in monitoring wells located nearest to residential areas to the west and southwest of the landfill.

The maximum concentration of bis(2-ethylhexyl)phthalate (38 ppb) was detected in MW-14 on the southeast end of the landfill in 1997 and was within the range of comparison values (see Table 2) . Of SVOCs that were detected above comparison values in groundwater, only bis(2-ethylhexyl)phthalate was detected on the western side of the Woburn Landfill.

Maximum concentrations of PAHs that exceed comparison values were detected in 1997 in shallow groundwater at monitoring well MW-13 on the northeast side of the landfill (Figure 4). PAHs [including benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, and naphthalene] were not detected above comparison values in groundwater on the west or southwestern portions of the landfill nearest residential areas. Table 2 provides the maximum concentrations of each of the PAHs that exceeded comparison values as well as their comparison values.

Three metals, arsenic, lead, and manganese, exceeded their corresponding comparison values in groundwater at the Woburn Landfill (Table 2). Arsenic was detected in the eastern portion of the landfill in 2004. The maximum concentrations of both lead and manganese were detected in the southern portion of the landfill at monitoring well MG-2 in 1988. Lead was not detected in groundwater sampling in 1997, and was detected at concentrations near or below the MCL in 2004. Manganese was not detected in subsequent sampling rounds in 1997 and 2004. Manganese is an essential trace element and is necessary for good health (ATSDR 2000a).

According to the post-closure plan, groundwater sampling will be conducted bi-annually for 2 years to monitor conditions at the Woburn Landfill (Maguire 2005).

B. Wetland Sediment

Wetland sediments are located in the southern and northwestern portions of the Woburn Landfill as well as in a small brook flowing west to east along the southern boundary of the landfill (Figure 2). A review of available information from MDEP indicates that as of September 2001, approximately 100,000 cubic yards of waste were removed from these wetland areas adjacent to the landfill and relocated to within the footprint of the landfill as part of closure activities.

Wetland sediment sampling was conducted in 1997, 1998, and 2004. Ten samples were collected in 1997, five samples were collected in 1998, and eight samples were collected in 2004. Twenty samples were analyzed for most volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs), 23 samples were analyzed for most metals, 15 samples were analyzed for polychlorinated biphenyls (PCBs), and 10 samples were analyzed for pesticides. Wetland sediment around the Woburn Landfill property had levels of SVOCs, PAHs, polychlorinated biphenyls (PCBs), and metals that were above soil comparison values (Table 3). The highest levels measured in wetland sediment on the entire site were compared to ATSDR comparison values to help determine if further evaluation was necessary. Since ATSDR comparison values for wetland sediment do not exist, soil comparison values were used as screening values.

Of the wetland sediment sampled at the Woburn Landfill, three samples (SED-3, SED-103, and SED-203) were located within the security fence that restricts access to the property (Figure 5). The remaining samples were taken in the wetlands outside of the fence.

Initial samples collected in March 1997 indicated that contamination was predominantly located in the southeastern corner of the landfill (SED-2) (Figure 5). The remainder of the sampling during 1997 and 1998 was conducted to delineate the nature and extent of contamination in this southeastern corner. During 1997, samples were taken below the initial depth (unknown) of SED-2 (samples identified as SED2 D-1 and SED2 D-2), as well as at 30-foot intervals east and west of SED-2 (samples identified as S2-1 through S2-4). Like SED-2, these additional samples were analyzed for VOCs, SVOCs, metals, and pesticides. During 1998, samples were again taken at 30-foot intervals east and west of SED-2 (samples identified as S2-5 through S2-10). These samples were analyzed for all of the above parameters except PCBs and pesticides. Additional sampling was conducted in 2004 to further evaluate conditions in the wetlands. The locations of 2004 sampling approximated those areas sampled during 1997. (Maguire 2005)

As seen in groundwater monitoring at the Woburn Landfill, contaminants at levels above comparison values were generally concentrated in the eastern or southeastern portion of

the landfill, away from residential areas to the west and southwest. Of the contaminants detected in wetland sediment at the Woburn Landfill, nine exceeded comparison values and required further evaluation in this report. Table 3 summarizes the maximum concentrations of each of these contaminants as well as their comparison values.

Maximum concentrations of the three PAHs that exceeded comparison values [benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene] were detected in 1998 at sampling location S2-8, one of the samples included in SED-2 as discussed above, in the southeastern corner of the Woburn Landfill (Figure 5). Detections of these PAHs were concentrated in the southeastern portion of the landfill. PAHs were not detected in sampling locations nearest to residential areas to the west and southwest (Figure 5). Maximum concentrations of benzo(a)anthracene and benzo(b)fluoranthene exceeded comparison values, but fall within the range of typical background concentrations observed in urban soils (ATSDR 1995) (see Table 3). The maximum concentration of benzo(a)pyrene (3.3 ppm) exceeded the CREG for soil (0.1 ppm) as well as the typical background range observed in urban soils.

Four metals, arsenic, cadmium, chromium, and lead, also exceeded their corresponding comparison values in wetland sediment at the Woburn Landfill (Table 3). The maximum concentrations of these metals were all located in the southeastern corner of the landfill, near sampling location SED-2 (Figure 5). The maximum detected concentrations of arsenic, cadmium, chromium, and lead were above the range of background concentrations observed for these metals in soils (ATSDR 1999; Shacklette and Boerngen 1984).

In addition to sampling that was conducted in wetland sediment around the perimeter of the Woburn Landfill, available environmental information indicates that grading and shaping materials were tested for contaminants prior to their use in the landfill closure process (MDEP 2002). These grading and shaping materials are used in landfill closure to maintain stability, prevent erosion, and promote drainage. Testing shows that low levels of PAHs and some metals were occasionally measured in construction and demolition debris (e.g. wood, metal, plaster, concrete, asphalt) used in grading and

shaping during the Woburn Landfill closure process from 1999-2003 (MDEP 2002). However, a review of monthly progress reports submitted to MDEP indicated that all material brought onsite adhered to the requirements of MDEP Policy# COMM-97-001 (MDEP 2002, Maguire 2002a). Policy No. 97-001 provides guidance on requirements, standards, and approvals for testing, tracking, transport, and reuse or disposal of contaminated soil at landfills. Both the monthly progress reports and the MDEP indicate that contaminant levels present in soil and debris used in grading and shaping did not include hazardous materials and did not include asbestos (MDEP 2002, Maguire 2002a).

According to the post-closure plan, wetland sediment sampling will be conducted annually for 2 years to monitor conditions at the landfill (Maguire 2005).

C. Surface Water

The Woburn Landfill is located at the northern edge of the Mystic River Drainage Basin within the Boston Harbor watershed (ESRI 2005). Surface water is located in wetlands in the southern and northwestern portions of the landfill as well as in a small brook flowing west to east along the southern border (Figure 2) (Maguire 2005). This brook flows east into the New Boston Drainway which flows south towards the Halls Brook Pond Area, the Aberjona River, and finally to the Mystic Lakes and the Mystic River.

In 1997 and 2004, surface water samples were collected from approximately the same locations as the wetland sediment samples described above (Figure 4). Ten samples were collected in 1997 and nine samples were collected in 2004. In March 1997, surface water samples were analyzed for VOCs, SVOCs, dissolved metals, landfill indicator parameters (i.e. pH, temperature, alkalinity, total dissolved solids, dissolved oxygen, chemical oxygen demand, sulfate, chloride, iron, manganese, specific conductance), PCBs, pesticides, and total cyanide. Samples collected in May and August 1997 were analyzed for all the same parameters except PCBs and pesticides. In June 2004, five samples were analyzed for VOCs, SVOCs, total metals, PCBs, and pesticides. In September 2004, four surface water locations were re-sampled and analyzed for both dissolved and total metals, hexavalent chromium, lead, and mercury.

There were some VOCs, SVOCs, PAHs, and metals detected in surface water at the Woburn Landfill. The highest levels measured in surface water on the entire site were compared to drinking water comparison values to help determine if further evaluation was necessary. Drinking water comparison values were used as screening values because ATSDR comparison values for surface water do not exist. The use of drinking water comparison values to screen surface water is conservative because drinking water values assume that an individual ingests 2 liters per day. Since it is unlikely that an individual would ingest 2 liters of surface water each day, exposure to contaminants in surface water would be expected to be considerably less than exposures to contaminants in drinking water.

Of the contaminants detected in surface water at the Woburn Landfill, 13 exceeded comparison values or did not have comparison values and, therefore, required further evaluation. Table 4 summarizes the maximum concentrations of each of these contaminants as well as their comparison values.

One VOC, chloroethane, detected in surface water at the Woburn Landfill exceeded the drinking water comparison value.

The maximum concentrations of two SVOCs, bis(2-ethylhexyl)phthalate and pentachlorophenol, were detected above comparison values in surface water (Table 4). The maximum concentrations of both SVOCs were detected in SW-3 in 1997 (Figure 5). In 2004, bis(2-ethylhexyl)phthalate was detected at levels similar to comparison values, while pentachlorophenol was not detected in surface water.

Maximum concentrations of the four PAHs [benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, and indeno(1,2,3-cd)pyrene] that exceeded comparison values were detected in 1997 at sampling locations SW-2 and SW-3 (Figure 5 and Table 4).

Six metals also were detected in surface water above drinking water comparison values and required further evaluation. Maximum concentrations of antimony, arsenic, cadmium, and chromium occurred at sample location SW-101 on the southwest end of the landfill (Figure 5). Maximum concentrations of dissolved lead (10 ppb) and total lead

(189 ppb) were above the Maximum Contaminant Level Guideline (MCLG) (0 ppb) and MDEP MMCL (15 ppb). The maximum concentration of dissolved manganese (3,500 ppb) was detected above the adult and child RMEG (2,000 ppb and 500 ppb, respectively).

According to the post-closure plan, surface water sampling will be conducted bi-annually for 2 years to monitor conditions at the landfill (Maguire 2005).

D. Landfill Gas

Landfill gases are a mixture of a number of contaminants formed during decomposition processes within a landfill. Landfill gases are primarily composed of methane (45-60% by volume) and carbon dioxide (40-60% by volume), both odorless, colorless gases. Landfill gases may also contain small amounts (0-1% by volume) of other gases such as hydrogen sulfide, which often gives landfill gas its objectionable rotten-egg odor, and other organic compounds (ATSDR 2001b).

During closure activities in 2003, the City of Woburn had installed a system for gas collection and control that monitors surface methane emissions at the Woburn Landfill. Landfill gases are managed by an enclosed flare and active gas collection and destruction system in order to prevent the occurrence of nuisance odor conditions or public health and safety problems (Maguire 2002b). The gas destruction system is located at the southern boundary of the landfill. The capping of the landfill in 2003 along with the installation of the active gas collection system and flare system was designed to provide an effective long-term solution to past odor concerns from nearby residents.

Landfill gas monitoring and sampling was conducted in 1997 as part of a comprehensive site assessment at the Woburn Landfill (ENSR 1998). Landfill gas monitoring was conducted concurrently with groundwater monitoring in March, May, and August of 1997. Gases were monitored for hydrogen sulfide, methane, total (non-methane) VOCs, and oxygen. The monitoring points were GW-X and GW-2 through GW-6. GW-X was located near the center of the landfill, GW-2, GW-3, and GW-4 were located near the eastern boundary of the landfill, and GW-5 and GW-6 were located near the western boundary of the landfill. The 1997 landfill gas monitoring points were destroyed during

closure activities and have been replaced by 17 wellheads that are part of the active gas collection and destruction system, and are used for weekly monitoring (Lynnfield 2005a, 2005b).

In 1997, neither hydrogen sulfide nor methane was detected at landfill gas monitoring wells around the Woburn Landfill property. Total VOCs were detected in one gas monitoring well, GW-6. Total VOCs in GW-6 were detected at 32 ppm in March, 23 ppm in May, and 20 ppm in August of 1997 (no comparison values exist for evaluating total VOCs in air). GW-6 was located near the northeastern corner of the property and nearby groundwater monitoring well MW-13 (Figure 4). Groundwater at MW-13 had levels of VOCs above drinking water comparison values in 1997. Oxygen at all the landfill gas monitoring wells was consistently measured at 21%, which is the typical concentration of oxygen in ambient air.

It is important to note that the 1997 sampling events were conducted prior to the installation of the active gas collection and destruction system at the Woburn Landfill in 2003. The combustion of landfill gases by an enclosed flare typically achieves 98% destruction of organic compounds, such as VOCs (ATSDR 2001b). Combustion also converts methane gas to carbon dioxide, which greatly reduces greenhouse gas emissions (ATSDR 2001b).

According to available information, landfill gas monitoring is being conducted on a weekly basis (Lynnfield 2005a, 2005b), with reports submitted monthly to MDEP and to the City of Woburn (J. Morey, MDEP, personal communication, 2007). MDEP requires monitoring for oxygen, carbon dioxide, hydrogen sulfide, and methane and reporting of any hazardous levels of methane to proper authorities (MDEP 2005).

VI. EVALUATION OF POTENTIAL EXPOSURE PATHWAYS

An evaluation of potential exposure pathways was conducted to determine whether contamination identified at Woburn Landfill could be impacting residents of Woburn in the past, present, or future. Exposure to a chemical must first occur before any potential adverse health effects can result. Five conditions must be present for exposure to occur.

First, there must be a source of that chemical. Second, an environmental medium must be contaminated by either the source or by contaminants transported away from the source. Third, there must be a location where a person can potentially contact the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, and dermal absorption. Fifth, there must be a potentially exposed population. Examples of exposed populations might include residents, workers, or trespassers. Even if all five elements of an exposure pathway are present, adverse health effects will not necessarily occur. The chemical must actually reach the target organ susceptible to the toxic effects caused by that particular substance at a sufficient dose and for a sufficient exposure time for an adverse health effect to occur (ATSDR 2005a).

A completed exposure pathway exists when all of the five elements are present. A potential exposure pathway exists when one or more of the five elements is missing or uncertain and indicates that exposure to a contaminant could have occurred in the past, could be occurring in the present, or could occur in the future. An exposure pathway can be eliminated if at least one of the five elements is missing and will not likely be present in the future.

To evaluate the potential for health effects, ATSDR Minimal Risk Levels (MRLs) were compared to exposure estimates for the contaminants of concern at Woburn Landfill. The MRL is an estimate of daily exposure to a contaminant below which noncancer, adverse health outcomes are unlikely to occur. In addition, exposure estimates for contaminants of concern were combined with USEPA cancer slope factors to evaluate potential cancer risk. Refer to Table 5 for a summary of exposure pathways discussed in this section.

A. Exposure to Groundwater

The groundwater wells sampled at the Woburn Landfill are for monitoring purposes only; these wells are not used for any other drinking or non-drinking water purposes. Past exposure to contaminated groundwater from the landfill is not a potential pathway for exposure because houses in the vicinity have been serviced by municipal water since at

least the 1960s, when the landfill began accepting waste (D. Dulong, Woburn Water Department, personal communication, 2006). Currently, the ingestion of groundwater is not a potential exposure pathway because there are no public or private drinking water wells in the vicinity of the landfill. According to the Woburn Water Department, nearly 100% of Woburn households obtain drinking water via the public water supply. Most of the public water supply (60%) comes from an underground aquifer in the Horn Pond area in southern Woburn, over 2 miles south of the Woburn Landfill. The remaining 40% comes from the Massachusetts Water Resources Authority (MWRA), which primarily obtains water from the Quabbin Reservoir in western Massachusetts (City of Woburn 2006; MWRA 2006; D. Dulong, Woburn Water Department, personal communication, 2006). Public water supplies are tested and treated on a routine basis in accordance with state and federal laws. More information on Woburn's water supply, including testing results, can be found at www.cityofwoburn.com or by contacting the Woburn Water Department. The Woburn Water Department and the Woburn Board of Health are not aware of any private wells used for non-drinking water purposes (e.g. filling swimming pools, watering gardens, or washing cars) in Woburn in the vicinity of the landfill (J. Fralick, Woburn Board of Health, personal communication, 2006; D. Dulong, Woburn Water Department, personal communication, 2006). Woburn city officials are aware of only two Woburn homes that use private drinking water wells; both homes are located in southern Woburn, over 2 miles from the Woburn Landfill (D. Dulong, Woburn Water Department, personal communication, 2006).

According to the City of Woburn, new private wells used for drinking water purposes would be unlikely to be permitted in the vicinity of the Woburn Landfill (Maguire 2005). In addition, the plumbing inspection process in Woburn would prevent any new well from being connected to a home that is presently connected to the municipal water supply (J. Fralick, personal communication, 2007). However, there is no official moratorium to restrict the installation of new private wells in Woburn (D. Dulong, Woburn Water Department, personal communication, 2006). Therefore, future exposures to contaminants identified in groundwater at the Woburn Landfill are possible if new private drinking water wells are installed in the path of contaminated groundwater, which flows in a southeasterly direction from the landfill. If nearby residents were to ingest

contaminated groundwater in the future at concentrations detected in onsite monitoring wells, noncancer and cancer health impacts could be possible due to exposure to some metals, PAHs, and VOCs, in particular, arsenic, lead, benzo(a)pyrene, benzo(a)anthracene, and pentachlorophenol.

The Woburn Landfill is traversed by two Boston Edison electric power line easements, both on the southern portion of the site; one traverses in a south to northwesterly direction, while the other traverses the landfill in a south to northeasterly direction towards New Boston Street. Future exposure to contaminants identified at the Woburn Landfill is possible if Boston Edison workers dig trenches and encounter groundwater (2.75 feet below ground surface). A conservative exposure scenario assumes that an adult worker ingests 0.025 liters of groundwater contaminated with the maximum concentration of contaminants for 5 days a week (typical work week) over 26 weeks for a 30-year period and assumes that the worker may contact the groundwater on his/her hands, forearms, lower legs, and feet for 1 hour each day. If utility workers were to come in contact with contaminated groundwater under these conditions in the future at concentrations detected in onsite groundwater monitoring wells, then neither increased cancer risks nor increased noncancer health impacts would be expected. Calculations were done for all contaminants of concern in groundwater. For example, exposure to benzene in groundwater is not expected to result in increased cancer risk to utility workers at the landfill[1].

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[1] $$\text{Cancer Effects Exposure Factor} = \frac{(5\ \text{days/week})\,(26\ \text{weeks/year})\,(30\ \text{years})}{(70\ \text{year lifetime})\,(365\ \text{days/year})} = 0.15$$

$$\text{Cancer Effects Exposure Dose(Adult Worker)} = \frac{(\text{max benzene concentration})\,(\text{ingestion rate})\,(\text{exposure factor})}{\text{body weight}}$$

$$= \frac{(0.013\ \text{mg/L})\,(0.025\ \text{L/day})\,(0.15)}{70\ \text{kg}} = 7.1x10^{-7}\ \textit{mg / kg - day}$$

$$\text{Cancer Risk} = (\text{Cancer Effects Dose})\,(\text{Cancer Slope Factor})$$

$$= (7.1 \times 10^{-7}\ \text{mg/kg - day})\,(0.055\ \text{mg/kg - day}^{-1})$$

$$= 3.9 \times 10^{-8}$$

B. Exposure to Soils and Sediment

The Woburn Landfill closure plan allowed for soils and debris with low levels of contamination to be brought from sites such as the Boston Convention and Exhibition Center and the MBTA South Piers Transitway Project for grading and shaping material and for daily or intermediate cover materials (MDEP 2002). According to monthly progress reports prepared during the final closure activities in 2000-2003, the material used for grading, shaping, and cover generally adheres to the requirements of MDEP Policy #COMM-97-001 *Reuse and Disposal of Contaminated Soil at Massachusetts Landfills*. This policy allows for the reuse of contaminated soils at unlined landfills in Massachusetts provided that the soil does not exceed allowable contaminant levels. Trespassers could have potentially encountered these contaminated soils on the landfill property in the past. However, soil and debris brought to the landfill during the closure process was required by MDEP to be analyzed for the presence of hazardous materials. While levels in debris occasionally exceeded the MDEP guidelines for lead and PAHs, according to the MDEP, the levels in those soils do not indicate that any hazardous materials were brought to the Woburn Landfill during closure (MDEP 2002). It is possible that some contaminant concentrations could have been above health based comparison values set by EPA and ATSDR; however, it is important to consider that these comparison values are based on a residential exposure scenario, and it is unlikely that a trespasser on the Woburn Landfill would have had contact with soil for a comparable frequency and duration of time. Therefore, it is unlikely that adverse health effects would result from this occasional exposure.

Current or future exposure to contaminated soil located on the landfill property is not a potential pathway for exposure as there is a security fence topped with barbed wire surrounding the property and an approximately 3-foot thick cap placed over the grading and shaping material (Maguire 2001; Lynnfield 2001). The cap consists of a 12-inch vegetative support layer (including 6 inches of loam and 6 inches of silty sandy soil) planted with native grasses and wildflowers, a 12-inch protective layer comprised of gravel and/or soils that shall not contain oil or hazardous material in excess of S1 Reportable Concentrations listed in 310 CMR 40.1600 of the Massachusetts Contingency

Plan, a drainage layer, a 6-inch gas venting layer composed of broken glass, a 40 mil (about 1.0 mm) high-density polyethylene cap layer, and a minimum of 6 inches of subgrade layer composed of soils that do not contain oil or hazardous material in excess of Reportable Concentrations for Soil (RCS1) listed in 310 CMR 40.1600 (Lynnfield 2001; Maguire 2001).

The only soil samples collected from the Woburn Landfill were wetland sediment samples collected from the adjacent wetland areas located to the east, west, and southwest and from an unnamed brook located immediately south (Figure 2). Some of these areas once contained landfill waste that was improperly disposed of in wetland areas. The waste was removed from the wetlands and relocated within the boundaries of the landfill area to be capped during closure activities in 1999-2003. Of the wetland sediment samples taken at the Woburn Landfill, three samples (SED-3, SED-103, and SED-203) were located within the chain-link fence that restricts access to the landfill property. The remaining samples were taken in the wetlands that surround portions of the site.

Since there are no physical barriers to restrict access to some of these wetland areas, incidental ingestion and dermal contact with contaminants detected above comparison values in wetland sediment could have been possible for children or adults who may have accessed the wetlands in the past, or those who may access them in the present and future. The majority of the sediment contaminants were detected below or within the range of residential soil comparison values; therefore, potential exposures to these contaminants in wetland sediment would not be expected to result in health effects. Of all contaminants evaluated in wetland sediment, only arsenic and benzo(a)pyrene were outside of the range of both comparison values and background soil concentrations (Table 3). However, it is important to note that although some contaminants are above comparison values, these values are based on a residential exposure scenario, and it is unlikely that a resident would have had contact with wetland sediments for a comparable frequency and duration of time due to the nature of the area and its distance from residences.

Arsenic was detected in wetland sediment above comparison values for residential soil and the range of typical background soil concentrations. Arsenic is classified as a known human carcinogen by the USEPA and International Agency for Research on Cancer (IARC), and ingestion of inorganic arsenic has been reported to increase the risk of developing cancers of the bladder, kidney, liver, lung and certain types of skin cancer (ATSDR 1993). Assuming a child accessing the Woburn Landfill wetlands touched or inadvertently ingested wetland sediment with the maximum concentration of arsenic detected (330 ppm) for 5 days each week for 22 weeks (i.e., the warmer months of May through September), they could have been exposed to arsenic at a level that could have presented an increased cancer risk[2]. However, these exposure assumptions are conservative, and it is very unlikely that a child would have had consistent contact with wetland sediment containing the highest concentration of arsenic, which was in the southeastern end of the landfill about 0.3 miles (approx. 1,600 feet) from the nearest residential area and bordered by industrial areas. In fact, wetland sediment located nearer to residential areas to the west and southwest had arsenic concentrations (ranging from not detected to 15 ppm) within the range of typical background levels for soil. Therefore, it is more likely that sediment with a range of concentrations could be encountered over time. Using the same exposure assumptions as listed above and using a more realistic assumption that a child could be exposed to the average concentration of arsenic (rather

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[2] Cancer Effects Exposure Factor $= \dfrac{(5\text{ days/week})\,(22\text{ weeks/year})\,(12\text{ years})}{(70\text{ years})\,(365\text{ days/year})} = 0.052$

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

$$= \frac{(330\text{ mg/kg})\,(200\text{ mg/day})\,(0.052)\,(1\text{kg}/10^{6}\text{ mg})}{35\text{ kg}} = 9.7x10^{-5}$$

$$\text{Cancer Risk} = (\text{Cancer Effects Dose})\,(\text{Cancer Slope Factor})$$

$$= (9.7 \times 10^{-5}\text{ mg/kg/day})\,(1.5\text{ mg/kg/day}^{-1})$$

$$= 1.5 \times 10^{-4}$$

than the maximum concentration) detected in sediment (84 ppm), the exposure dose would be 2.5 x 10^{-5} mg/kg/day, and an increased cancer risk would be unlikely[3].

Benzo(a)pyrene was also evaluated using exposure assumptions similar to those used to evaluate arsenic exposure for an adult or child resident. If area residents were to come in contact with sediment contaminated with the maximum measured concentrations of benzo(a)pyrene, then increased cancer risks would not be expected.[4]

Calculations for all other contaminants of concern in wetland sediment, using the above exposure scenario, indicate that neither increased cancer risk nor increased noncancer health impacts would be expected.

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[3] Cancer Effects Exposure Factor $= \frac{(5\text{ days/week})\,(22\text{ weeks/year})\,(12\text{ years})}{(70\text{ years})\,(365\text{ days/year})} = 0.052$

$$\text{Cancer Effects Exposure Dose(Child)} = \frac{(\text{avg arsenic concentration})\,(\text{ingestion rate})\,(\text{exposure factor})\,(1\text{kg}/10^6\text{ mg})}{\text{body weight}}$$

$$= \frac{(84\text{ mg/kg})\,(200\text{ mg/day})\,(0.052)\,(1\text{kg}/10^6\text{ mg})}{35\text{ kg}} = 2.5x10^{-5}$$

Cancer Risk = (Cancer Effects Dose) (Cancer Slope Factor)

$= (2.5 \text{ x } 10^{-5} \text{ mg/kg/day}) (1.5 \text{ mg/kg/day}^{-1})$

$= 3.7 \text{ x } 10^{-5}$

[4] Cancer Effects Exposure Factor $= \frac{(5\text{ days/week})\,(22\text{ weeks/year})\,(12\text{ years})}{(70\text{ years})\,(365\text{ days/year})} = 0.052$

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

$$= \frac{(3.3\text{ mg/kg})\,(200\text{ mg/day})\,(0.052)\,(1\text{kg}/10^6\text{ mg})}{35\text{ kg}} = 9.7x10^{-7}$$

Cancer Risk = (Cancer Effects Dose) (Cancer Slope Factor)

$= (9.7 \text{ x } 10^{-7} \text{ mg/kg/day}) (7.3 \text{ mg/kg/day}^{-1})$

$= 7.1 \text{ x } 10^{-6}$

C. Exposure to Surface Water

Surface water samples were collected from the adjacent wetland areas located to the east, west, and southwest and from an unnamed brook located immediately south of the Woburn Landfill (Figure 2). Some of these areas once contained landfill waste that was improperly disposed of in wetland areas. The waste was removed from the wetlands and relocated within the boundaries of the landfill area to be capped during closure activities in 1999-2003. Incidental ingestion and dermal contact with contaminants detected in surface water in these wetlands could be possible for children or adults who may have accessed surface water bodies around the landfill for wading or playing in the past, present, and future. Several surface water contaminants were detected above comparison values. However, it is important to note that the comparison values used in this evaluation represent a daily drinking water exposure. Individuals accessing the wetlands would likely be exposed less frequently and to significantly less volume of contaminated surface water through incidental ingestion and dermal contact than to drinking water. For example, while benzo(a)pyrene (0.3 ppb) was detected above the drinking water comparison value, the ATSDR CREG for cancer health effects (0.005 ppb), if a child ingested 0.05 liters (about a mouthful) of surface water contaminated with the maximum concentration of benzo(a)pyrene for 5 days/week over 22 weeks/year for 12 years, an increased cancer risk would not be expected [5]. Calculations for all other contaminants of concern in surface water, using the above exposure scenario, indicate that neither increased cancer risk nor increased noncancer health impacts would be expected.

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[5] $\text{Cancer Effects Exposure Factor} = \dfrac{(5\text{ days/week})\,(22\text{ weeks/year})\,(12\text{ years})}{(70\text{ years})\,(365\text{ days/year})} = 0.052$

$$\text{Cancer Effects Exposure Dose(Child)} = \frac{(\text{max contaminant concentration})\,(\text{ingestion rate})\,(\text{exposure factor})}{\text{body weight}}$$

$$= \frac{(.0003\text{ mg/L})\,(0.05\text{ L/day})\,(0.052)}{35\text{ kg}} = 2.2x10^{-8}$$

$$\text{Cancer Risk} = (\text{Cancer Effects Dose})\,(\text{Cancer Slope Factor})$$

$$= (2.2 \times 10^{-8}\text{ mg/kg/day})\,(7.3\text{ mg/kg/day}^{-1})$$

$$= 1.6 \times 10^{-7}$$

D. Exposure to Landfill Gas

Potential exposure to landfill gas by nearby residents in the past was evaluated using the limited environmental sampling data from 1997. As stated in Section V, hydrogen sulfide and methane were not detected and oxygen was measured at 21%, which is the typical concentration in ambient air. Total (non-methane) VOCs were detected at a maximum concentration of 32 ppm (there are no comparison values available to evaluate total VOCs in outdoor or ambient air). To evaluate whether the levels of total VOCs detected in landfill gas at the site could present an exposure concern at nearby residences, MDPH asked ATSDR to run a model using conservative assumptions (ATSDR 2007). ATSDR used the information on VOCs detected in landfill gas and applied a model to determine what concentrations of total VOCs might be present in the air surrounding nearby homes. Modeling results indicated that if a concentration of total VOCs of 32 ppm was measured on the landfill, then the concentration of total VOCs present at the nearest residence 1,500 ft away would be about 0.18 ppm (ATSDR 2007). It is not possible to know if health effects were possible because there were no specific compounds measured in the landfill gas. However, based on general guidelines for total VOCs in air, the concentrations predicted at nearby homes are within a comfort range (<0.3 ppm) where odor and irritation complaints are seldom observed (Commission of the European Communities 1992). In addition, VOCs were not detected in 15 other landfill gas samples collected at five onsite locations that are closer to nearby residences.

Present or future exposure to landfill gases is unlikely due to the installation of a gas collection system at the Woburn Landfill during closure activities in 2003. A landfill gas collection and control system can have the greatest impact on minimizing migration and exposure to nearby residents (ATSDR 2001b). An active gas collection system, like that installed at the Woburn Landfill, is considered the most effective means of gas collection. Unlike a passive system, an active gas collection system includes vacuums or pumps that create a preferred pathway for the migration of landfill gas. Once collected, the gas is combusted using an enclosed gas flare which can typically achieve over 98% destruction of organic compounds (ATSDR 2001b).

E. Exposure to Indoor Air

While current information indicates that there are no private or public wells in the path of contaminated groundwater from the Woburn Landfill, exposure to VOCs detected in groundwater could occur through indoor air in homes if VOCs are present in offsite groundwater at sufficient concentrations and if groundwater is shallow. Maguire determined that the onsite depth to groundwater ranges from 2.75–20 feet below ground surface and that groundwater flows in a southeastern direction, away from nearby residences (Maguire 2005). There were no offsite groundwater sampling data available, so it is unknown whether the concentrations of VOCs such as benzene, chloroform, and trichloroethylene detected in onsite groundwater monitoring wells exist offsite. However, to evaluate a possible vapor intrusion exposure scenario, the MDPH used a model called the Johnson-Ettinger Model for Subsurface Vapor Intrusion into Buildings that incorporates site-specific information on groundwater, soil, and housing for a particular area.

To evaluate a very conservative scenario, the Johnson-Ettinger (USEPA 2004b) mathematical model (GW-SCREEN-Feb04) was run using the maximum concentration of the contaminants of concern together with the assumption that the water table could be as shallow as about 1 foot below a basement floor. Based on these parameters, (i.e., if a house with a basement were located on top of the groundwater monitoring well with the highest concentration of a VOC and the shallowest groundwater), the model predicted indoor air concentrations that would not be expected to increase the risk of noncancer health effects or the risk of cancer. All contaminants evaluated (acetone, benzene, carbon disulfide, chlorobenzene, chloroethane, chloroform, chloromethane, 1,2-dichlorobenzene, 1,4-dichlorobenzene, cis-1,2-dichloroethene, ethylbenzene, methyl-tert-butyl-ether, naphthalene, styrene, toluene, trichloroethylene, and xylenes) showed a low incremental

cancer risk from vapor intrusion to indoor air of 3×10^{-5} or less[6] or were below levels of concern for noncancer health effects.

Some additional considerations suggest that model estimates would be even lower if certain site characteristics were taken into account. First, it is important to consider that groundwater flow is in a southeasterly direction, away from the closest residences located 500–1,000 feet west and southwest of the landfill. Since maximum contaminant concentrations are typically located closest to the source of contamination, actual down-gradient concentrations in groundwater are expected to be less than concentrations detected in onsite monitoring wells. Also, as contaminants travel with groundwater, they typically move deeper in the groundwater, resulting in a lower possibility of vapor intrusion into basements. In addition, since groundwater flow is in the direction of a brook that traverses the southern portion of the landfill, it is possible that vapors could have escaped through the exposed stream; therefore, down-gradient groundwater concentrations would be even lower. Further, the exposure assumptions used in the exposure calculations are very conservative. They assume that a resident is breathing the maximum concentration of a contaminant for 365 days per year for 30 years. Thus, while offsite groundwater was not sampled, based on the levels detected in onsite groundwater, the distance of homes from the landfill, the direction of groundwater, and the indoor air concentrations predicted by the Johnson-Ettinger model using very conservative assumptions, it appears unlikely that VOCs detected in groundwater at the Woburn Landfill would present an exposure concern for indoor air in nearby homes.

VII. CANCER INCIDENCE ANALYSIS

In response to community concerns, the MDPH conducted an evaluation of cancer incidence from 2000–2003 in census tract (CT) 3336, where the Woburn Landfill and

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[6] Benzene Cancer Effects Exposure Dose $= \frac{(2.42\ \mu g/m^3)\ )\ (365\ days/yr)\ (30\ yrs)}{(70\ yrs)\ (365\ days/yr)} = 1.04\ \mu g/m^3$

Benzene Cancer Risk = $1.04\ \mu g/m^3 \times 2.3 \times 10^{-5}\ (\mu g/m^3)^{-1} = 2.4 \times 10^{-5}$

nearby residential areas are located. In addition, the geographic pattern of cancer was evaluated at the neighborhood level to identify any unusual patterns of cancer diagnoses in proximity to the Woburn Landfill or in any other area of CT 3336. This cancer analysis is an update of the *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999*, which evaluated the most recent and complete cancer incidence data available at that time (MDPH 2004). This current cancer analysis focuses on cancer types with potential associations to contaminants of concern identified at the Woburn Landfill and evaluates them for more recent years that are now available.

A. Methods

1) Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 2000–2003 were obtained for Woburn CT 3336 from the Massachusetts Cancer Registry (MCR), a division of the Bureau of Health Information, Statistics, Research and Evaluation within the MDPH. Five cancer types were evaluated: bladder cancer, brain and central nervous system (CNS) cancer, kidney cancer, leukemia, and lung and bronchus cancer. Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. (See Appendix A for the incidence coding definitions used in this report for these cancer types.) These cancer types were selected for evaluation because of their potential associations with contaminants of concern at the Woburn Landfill and/or residents' concerns about suspected elevations in some cancer types. Only cases reported to the MCR as a primary cancer for one of the five cancer types and diagnosed among residents of Woburn CT 3336 were included in the analyses. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis.

The MCR is a population-based surveillance system that began collecting information on Massachusetts residents diagnosed with cancer in the state in 1982. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111s.111B). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for

accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment). Once these steps are finished, the data for that year are considered "complete." Due to the volume of information received by the MCR, the large number of reporting facilities, and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of 2 years prior to the current date. At the time of this analysis, the period from 1982–2003 constitutes the period for which the most recent and complete cancer incidence data were available from the MCR.[7]

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

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses would be made by the MCR after consulting with the reporting hospital or diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers would be included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different primary site, or a new cancer of the same histology (cell type) as an earlier

[7] The data summarized in this report are drawn from data entered on MCR computer files up to September 2006. The numbers presented in this report may change slightly in future reports, reflecting late reported cases, address corrections, or other changes based on subsequent details from reporting facilities.

cancer, if diagnosed in the same primary site (original location in the body) more than 2 months after the initial diagnosis (MCR 1996). Therefore, duplicate reports of an individual diagnosed with cancer would be removed from the analyses whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases. In Woburn CT 3336, no duplicate reports were identified during the years 2000–2003.

2) Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer diagnoses occurred in Woburn CT 3336, cancer incidence data were tabulated by gender according to 18 age groups to compare the observed number of cancer diagnoses to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were calculated for the period 2000–2003 for each of the five primary cancer types evaluated for Woburn CT 3336.

Because accurate age group and gender-specific population data are required to calculate SIRs[8], the CT is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a county as defined by the United States Census Bureau. According to the 2000 United States Census, the city of Woburn is subdivided into seven census tracts (CTs 3331, 3332, 3333, 3334, 3335.01, 3335.02, and 3336) as shown in Figure 1 (U.S. DOC. 2000). The Woburn Landfill and nearby residential areas are located in CT 3336.

3) Interpretation of a Standardized Incidence Ratio (SIR)

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as a larger comparison population designated as "normal" or average. Usually, the state as a whole is selected to

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

be the comparison population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates.

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

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

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

4) Calculation of the 95% Confidence Interval

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

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

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval (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 than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of diagnoses, statistical significance was not assessed when fewer than five diagnoses were observed.

5) Evaluation of Risk Factor Information

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes the individual's age at diagnosis, the stage of disease, and the individual's smoking history and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to bladder, kidney, and lung and bronchus cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer-causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for residents of Woburn CT 3336 who were diagnosed with the five cancer types evaluated in this report. However, information about personal risk factors such as family history, hormonal events, diet, and other factors that may also influence the development of cancer is not collected by the MCR or any other readily accessible source; therefore, it was not possible to evaluate these factors in this investigation.

6) Determination of Geographic Distribution

Address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2005). This allowed for the assignment of census tract location for each diagnosis as well as an evaluation of the spatial distribution of individual diagnoses at a smaller geographic level within a census tract (e.g., neighborhoods). The geographic distribution was determined using a qualitative evaluation of the point pattern of cancer diagnoses in Woburn CT 3336. In instances where the address information from the MCR was incomplete (i.e., did not include specific streets or street numbers), efforts were made to research those cases using Registry of Motor Vehicle records and telephone books issued within 2 years of an

individual's diagnosis. The residences of two individuals diagnosed with cancer could not be mapped due to insufficient address information. In accordance with Massachusetts laws aimed at protecting the confidentiality of patients (M.G.L. c.111. s 24A), maps of the locations of the residence of individuals with cancer cannot be provided in this report.

B. Cancer Incidence in Woburn Census Tract 3336

The following section presents the results of the cancer incidence analyses for Woburn CT 3336 during the 4-year time period 2000–2003. Although SIRs and 95% confidence intervals were not calculated for some cancer types due to small numbers of observed diagnoses (i.e., fewer than five), the expected number of diagnoses was calculated to determine whether excess numbers of diagnoses were occurring. These data are summarized in Table 6.

Of the five cancer types evaluated in this report, one statistically significant elevation was observed (i.e. bladder cancer) during the time period 2000–2003 in Woburn CT 3336, the census tract where the Woburn Landfill is located. Bladder cancer occurred more often than expected (nine diagnoses observed versus 3.2 expected, SIR = 282, 95% CI = 129-535). Six males were diagnosed with bladder cancer during 2000–2003 compared to about two males expected. The elevation among males was not statistically significant (SIR = 259, 95% CI = 94-563). Three females were diagnosed with bladder cancer compared to about one expected.

The incidence of brain and CNS cancer was slightly elevated in Woburn CT 3336 during 2000–2003 (4 diagnoses observed compared to 1.8 expected). This elevation was based on one additional diagnosis over the expected number for males and approximately one additional diagnosis over the expected number for females when evaluated separately.

There were five diagnoses of kidney cancer compared to 3.4 expected (SIR = 148, 95% CI = 48-344) in CT 3336 during 2000–2003. This elevation was not statistically significant.

From 2000–2003, leukemia occurred at approximately the expected rate in Woburn CT 3336 (3 diagnoses observed versus 2.8 expected). However, all three diagnoses occurred in children 0-19 years of age. Among children 0–19 years of age, less than one diagnosis of leukemia would be expected during 2000–2003 in Woburn CT 3336.

Lung and bronchus cancer occurred less often than expected in Woburn CT 3336 during 2000–2003 (13 diagnoses observed compared to 17.7 expected, SIR = 73, 95% CI = 39–125). Seven males were diagnosed with lung and bronchus cancer compared to about nine expected (SIR = 75, 95% CI = 30-155). Among females, there were six diagnoses observed compared to about eight diagnoses expected (SIR = 71, 95% CI = 26-155).

C. Evaluation of Cancer Risk Factors

As previously mentioned, cancer is not just one disease but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (i.e., period between exposure and development of disease), characteristics and trends in survival. Available information from the MCR related to age and gender patterns, as well as other factors related to the development of cancer (i.e., smoking and occupation), was reviewed for individuals diagnosed with one of the five cancer types in Woburn CT 3336 during 2000–2003. Information for each of the five cancer types was compared to known or established incidence trends to assess whether any unexpected patterns exist among the cases. For more information regarding risk factors associated with the cancer types evaluated in this report, please refer to Appendix B.

Age and gender are risk factors in many types of cancers, including bladder cancer, kidney cancer, and lung and bronchus cancer. A review of SIRs specific to age groups was not possible because of the small numbers of diagnoses in each age group; however, the distribution of diagnoses by age was reviewed.

Tobacco use is also a known or suggested causal risk factor in several types of cancer, including bladder cancer, kidney cancer, and lung and bronchus cancer. The smoking history of individuals diagnosed with these cancer types was reviewed to assess the role

tobacco smoking may have played in the development of these cancers among residents of Woburn CT 3336.

In some studies, an association has been found with exposures specific to certain occupations and an increase in the incidence of bladder cancer, brain and CNS cancer, kidney cancer, leukemia, and lung and bronchus cancer. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types to determine the role that occupational factors may have played in their development in Woburn CT 3336. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. Further, these data are often incomplete because occupational information can be reported as unknown, at home, or retired.

Finally, histologic (cell type) distribution was reviewed for diagnoses of leukemia and brain and CNS cancer in Woburn CT 3336. Patterns of disease were compared to known or established incidence trends to assess whether any unusual patterns exist.

1) Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 61,420 people in the Unites States in 2006, accounting for 6% of all cancers diagnosed in the United States among men and 2% among women (ACS 2006a). White males have the highest prevalence of bladder cancer across all racial groups. A male to female ratio of four to one has been observed among whites, while a slightly lower male to female ratio of three to one has been observed among most other racial groups. Further, the occurrence of bladder cancer rises with increasing age. The mean age at diagnosis in Massachusetts for the years 2000–2003 was 72 years.

Bladder cancer is strongly associated with a history of cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS 2006a). Tobacco use is associated with approximately 25-60% of all bladder cancers (Johansson and Cohen 1997). Almost 72% of those diagnosed with bladder cancer in

Massachusetts from 2000–2003 who had a known smoking history reported current or former tobacco use.

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

(a) Age and Gender

A review of individuals diagnosed with bladder cancer in Woburn CT 3336 from 2000–2003 revealed that 67% of diagnoses were male (n = 6). Males comprised 71% of bladder cancers statewide for this time period. The mean age at diagnosis in Woburn CT 3336 was 74 years, which is consistent with statewide bladder cancer incidence (mean age of 72 years).

(b) Tobacco Use

Of the nine individuals in Woburn CT 3336 who were diagnosed with bladder cancer during the years 2000–2003, eight had a known smoking history. Of those eight

individuals, seven were current/former smokers (88%). This is greater than the 72% of individuals who were current/former smokers among those who were diagnosed with bladder cancer in Massachusetts during 2000–2003 and reported a smoking history.

(c) Occupation

Review of occupation for individuals diagnosed with bladder cancer in Woburn CT 3336 revealed that at least two individuals might have worked at a job in which occupational exposures potentially related to the development of bladder cancer may have been possible. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not available. The occupation reported for the remaining individual is not likely to be related to an increased risk of this cancer type. However, occupation was reported as retired or unknown for most individuals (67%, n = 6).

2) Brain and Central Nervous System (CNS) Cancer

The American Cancer Society estimates that 18,820 Americans (10,730 men and 8,090 women) will be diagnosed with brain and CNS cancer in 2006 (ACS 2006b). According to epidemiological literature, brain tumor incidence (cancerous and non-cancerous) declines after a peak in childhood (under 10 years of age), increases from age 25 to 75, and levels off after age 75 (Preston-Martin and Mack 1996). Certain types of brain tumors are more likely to develop in children and others are more typically seen in adults (Black 1991, NCI 1996). Malignant brain and spinal cord tumors are the second most common cancers (following leukemia) in children and account for about 17% of all cancer types diagnosed among children (ACS 2005a).

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

(a) Age and Gender

From 2000 to 2003, males and females statewide were diagnosed with brain and CNS cancer nearly equally. In Woburn CT 3336, males and females were diagnosed with brain and CNS cancer equally during that time period (2 males and 2 females). The age at diagnosis for individuals with brain or CNS cancer was consistent with the pattern expected based on the scientific literature for this cancer type. All of the individuals diagnosed with brain or CNS cancer were diagnosed after age 25. The average age of individuals diagnosed with brain or CNS cancer in Woburn CT 3336 during 2000–2003 was 63 years; the state mean for the same time period was 54 years.

(b) Histology

In general, each diagnosis of cancer is classified by its histology, which is the tissue or cell type from which the cancer originates. Astrocytomas and oligodendrogliomas are the most common primary tumors of the adult brain. Both tumors are types of gliomas, meaning they develop from glial cells. Gliomas can be slowly growing (low-grade, grades 1 and 2), or rapidly growing (high-grade, grades 3 and 4). Glioblastomas are malignant astrocytomas that grow and spread aggressively (high-grade). These make up about two-thirds of all astrocytomas and are the most common malignant brain tumors of adults (ACS 2006b). In addition to these sub-types of brain cancer, there are a number of less common sub-types, including medulloblastomas, meningiomas, and primitive neuroectodermal tumors (PNET).

The distribution of brain and CNS cancers by cell type was reviewed for individuals diagnosed with brain and CNS cancer in Woburn CT 3336. Patterns of disease were compared to known or established incidence trends to assess whether any unexpected patterns exist. Of the four individuals in Woburn CT 3336 reported to the MCR with a diagnosis of brain or CNS cancer from 2000 to 2003, one individual was diagnosed with a non-specified type of glioma, while the remaining three individuals (75%) were diagnosed with a glioblastoma (a specific sub-type of glioma), which is the most common malignant brain tumor for adults. This is similar to trends in histologic distribution for the state of Massachusetts as a whole. Specifically, 84% (n=1644) of individuals

diagnosed with brain or CNS cancer in Massachusetts were diagnosed with a glioma and the remaining 16% of individuals were diagnosed with a less common histology type.

(c) Occupation

Among the four individuals in Woburn CT 3336 diagnosed with brain or CNS cancer, an occupation was reported for two individuals. Review of occupation for these individuals revealed that one individual might have worked at a job in which occupational exposures potentially related to the development of brain or CNS cancer may have been possible. However, information regarding specific job duties that could help to further define exposure potential for this individual was not available. The occupation reported for the other individual is not likely to be related to an increased risk of this cancer type. Occupation was reported as retired or unknown for the remaining two individuals.

3) Kidney Cancer

Kidney cancer is twice as common in males as it is in females, and the incidence of kidney cancer is highest between the ages of 55 and 84 (ACS 2005b). Epidemiological studies have shown that incidence rates of kidney cancer rise with increasing age before reaching a plateau at approximately age 70 (McLaughlin et al. 1996). The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 40% (ACS 2005b). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of those in women may be caused by cigarette smoking (McLaughlin et al. 1996).

Although kidney cancer is not generally considered an occupationally associated cancer, some studies have suggested that environmental and occupational factors may be associated with its development. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. In

addition, exposure to cadmium is associated with an increased incidence of kidney cancer, particularly among men who smoke. In addition, workplace exposure to organic solvents, such as TCE, may increase the risk of this cancer (ACS 2005b). More recently, renal cell carcinoma, the most common type of kidney cancer, has been suggested to be associated with occupational exposure to petroleum, tar, and pitch products. However, studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between exposure to gasoline or other petroleum products and kidney cancer (Linehan et al. 1997, McLaughlin et al. 1996).

(a) Age and Gender

Kidney cancer in Woburn CT 3336 occurred more often in males (four diagnoses) than in females (one diagnosis), which is consistent with state trends and with the epidemiological literature. The average age of individuals diagnosed with kidney cancer in Woburn CT 3336 during 2000–2003 was 65 years, which is comparable to the state mean of 64 years for the same time period.

(b) Tobacco Use

As stated above, cigarette smoking is the most important known risk factor for kidney cancer. Of the five individuals diagnosed with kidney cancer in Woburn CT 3336 during 2000–2003, four had a known smoking history. Of those four, three individuals (75%) reported being current or former smokers. Of individuals diagnosed with kidney cancer in Massachusetts during 2000–2003 who had a known smoking history, 57% were current/former smokers.

(c) Occupation

Among the five individuals in Woburn CT 3336 diagnosed with kidney cancer from 2000 to 2003, an occupation was reported for two individuals. Review of occupation for these individuals revealed that one individual might have worked at a job in which occupational exposures that could be related to the development of kidney cancer may have been possible. However, information regarding specific job duties that could help to further define exposure potential for this individual was not available. Occupation

reported for the remaining individual is not likely to be related to an increased risk of kidney cancer. Occupation was reported as unknown or retired for the remaining three individuals.

4) Leukemia

In 2006, leukemia is expected to affect approximately 35,000 individuals in the United States (ACS 2006c, 2006d). In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, representing more than 2% of all cancer diagnoses (ACS 2006c, 2006d). There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also several rare types of leukemia (e.g., hairy cell leukemia, myelomonocytic leukemia). In adults, the most common types are AML and CLL.

Leukemia is the most common type of childhood cancer, accounting for more than 30% of all cancers diagnosed in children. In 2006, leukemia is expected to affect approximately 3,100 children between 0 and 14 years of age in the United States (ACS 2006e). ALL is most common in early childhood, peaking between 2 and 3 years of age. AML is most common during the first 2 years of life and is less common among older children. AML diagnoses start to increase again during the teenage years (ACS 2006e).

The various subtypes of leukemia occur with different frequencies in the population. For the purpose of classification in this evaluation, if the histology (i.e., cell type) of the leukemia diagnosis was not otherwise specified or not classified as one of the four main subtypes, then the individual case was categorized as "other." Available information regarding the expected distribution of leukemia by histology types can vary considerably depending on coding methods, making comparisons of type-specific incidence rates from different cancer registries difficult (Linet and Cartwright 1996). In the state of Massachusetts during the time period 2000–2003, 34% of all leukemia cases were AML, 30% were CLL, 12% were ALL, 12% were CML, and 13% were other histology types.

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

(a) Age and Gender

All three individuals diagnosed with leukemia in Woburn CT 3336 during 2000–2003 were male children 0-19 years of age.

(b) Histology

The four main leukemia subtypes have different risk factors suspected to be associated with their development and generally occur with different frequency among adults and children. Of the three individuals diagnosed with leukemia in Woburn CT 3336 during 2000–2003, two children were diagnosed with the AML subtype. AML is most common during the first 2 years of life and is less common among older children. The incidence of AML starts to increase again during the teenage years, with AML becoming the most common acute leukemia in adults. One of the children diagnosed with AML was a young child under the age of 5, and the other was an older child over the age of 15. ALL, the other subtype diagnosed in one child in Woburn CT 3336, is most common in early childhood and peaks between 2 and 3 years of age. The child diagnosed with ALL was a young child under the age of 5.

(c) Occupation

Because two individuals were young children at the time of their diagnosis, occupation is not relevant as a risk factor. Although the other individual worked at a job in which occupational exposures could have occurred, it is not possible to assess the role of

occupation as a risk factor due to a number of uncertainties including specific job duties and length of employment.

5) Lung and Bronchus Cancer

The American Cancer Society estimates that lung cancer will be diagnosed in 174,470 people in the United States in 2006, accounting for about 13% of all cancers (ACS 2006f). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS 2006f). According to epidemiological literature, the incidence of lung cancer increases sharply with age and peaks around approximately age 60 to 70. Only 2% of lung cancers occur before the age of 40. In addition, lung cancer is generally observed more often among men than women (Blot and Fraumeni 1996, MCR 2006).

Lung cancer is divided into two main types: small cell lung cancer and nonsmall cell lung cancer. Nonsmall cell lung cancer is further sub-divided into three types: adenocarcinoma, squamous cell carcinoma, and large-cell undifferentiated carcinoma. The different types of lung cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung cancers are adenocarcinomas, 25-30% are squamous cell carcinomas, 20% are small cell cancers, and 10-15% of cases are large cell carcinomas (ACS 2006f).

About 87% of all lung cancers are thought to be caused directly by smoking cigarettes or by exposure to second hand smoke, or environmental tobacco smoke (ACS 2006f). An increase in cigarette smoking among women has produced lung cancer incidence rates that more closely resemble those experienced by males. The risk of developing lung cancer depends on the intensity of one's smoking habits (e.g., duration of habit, amount smoked, tar yield of cigarette, and filter type). Smoking cessation decreases the elevated risk by about 50%; however, former smokers still carry a greater risk of developing lung cancer than those who have never smoked.

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

for this disease (ACS 2006f). Underground miners exposed to radon and uranium are also at an increased risk for developing lung cancer (ACS 2006f; Samet and Eradze 2000). Other occupations potentially associated with this cancer include chemical workers, talc miners and millers, paper and pulp workers, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers. In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer. Occupational exposure to these compounds in conjunction with cigarette smoking can dramatically increase the risk of developing lung cancer (Blot and Fraumeni 1996).

(a) Age and Gender

Among the 13 individuals diagnosed with lung and bronchus cancer in Woburn CT 3336 during 2000–2003, the average age at diagnosis was 68 years. None of these individuals were under the age of 40 at the time of diagnosis and males and females were diagnosed with lung and bronchus cancer about equally, which is consistent with the literature.

(b) Tobacco Use

As stated above, cigarette smoking is known to be the major risk factor for a majority of lung and bronchus cancer diagnoses. Of the 13 individuals diagnosed with lung or bronchus cancer in Woburn CT 3336 during 2000–2003, 12 had a known smoking history. Of those 12 individuals, 92% (n = 11) were current or former smokers, which is consistent with trends in Massachusetts.

(c) Occupation

Among the 13 individuals in Woburn CT 3336 diagnosed with lung or bronchus cancer, an occupation was reported for 12 individuals. Review of occupation revealed that seven individuals might have worked at a job in which occupational exposures to the chemicals listed above may have been possible. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not

available. The occupations reported for the remaining five individuals are not likely to be related to an increased risk of this cancer type. Occupation was reported as unknown for the remaining individual.

D. Geographic Distribution

In addition to determining incidence rates for each cancer type, a qualitative evaluation of the geographic pattern of cancer diagnoses was conducted, particularly as it relates to areas of environmental concern. Place of residence at the time of diagnosis was mapped for each individual diagnosed with the cancer types evaluated in this report to assess any possible geographic concentrations of cases in relation to each other or in the vicinity of the Woburn Landfill, or other potential locations of environmental concern (e.g., MDEP 21E hazardous material and oil releases) located in Woburn CT 3336. As previously mentioned, cancer is one word that describes many different diseases. Therefore, for the purposes of this evaluation, the geographic distribution of each cancer type was evaluated separately to determine whether an atypical pattern of any one type was occurring.

Based on a review of address at the time of diagnosis for each individual diagnosed with the cancer types evaluated in this report, no apparent concentrations of cancer diagnoses (of any type) were observed in any one area of Woburn CT 3336. There was no geographic pattern observed among the individuals diagnosed with bladder cancer during the time period 2000–2003, when a statistically significant elevation in bladder cancer incidence among males and females was observed. There was no geographic pattern observed among the three individuals diagnosed with leukemia in CT 3336 during this time period. The individuals diagnosed with leukemia were approximately 0.5 miles from each other and approximately 1 mile from the landfill. No apparent geographic concentrations of cancer diagnoses were noted in neighborhoods surrounding the Woburn Landfill.

No other unusual spatial patterns or concentrations of cases at the neighborhood level that would suggest a common factor (environmental or nonenvironmental) related to cancer diagnoses among residents was apparent for any of the five cancer types evaluated. Any patterns that were observed appeared to be consistent with what would be expected based

on the population distribution and areas of higher population density. For example, in Woburn CT 3336, the majority of individuals with each type of cancer tended to be located in areas of the town where population and housing density are greater. Although elevations in the incidence of some cancer types were noted in Woburn CT 3336, in general, the geographic distribution of diagnoses for these cancer types seemed to coincide closely with the pattern of population and cases did not appear to be concentrated in any one area of the census tract.

VIII. DISCUSSION

As mentioned previously, the MDPH conducted an evaluation of possible environmental exposures and a review of cancer incidence in relation to the Woburn Landfill at the request of the Woburn Neighborhood Association, concerned residents, and State Representative James R. Miceli. The landfill began accepting waste for disposal around 1966 and officially ceased operations in 1986. Closure activities at the Woburn Landfill began in 1999 and continued until completion in late 2003. The evaluation was initiated based on community concerns about possible environmental exposures to contaminants located at the landfill and potential adverse health effects for nearby residents.

The investigation reviewed available environmental data for the Woburn Landfill and considered potential ways that people may come into contact with contaminants detected in groundwater, surface water, air, and wetland sediment. A review of information available on drinking water sources indicates that there are no private drinking water wells located in the vicinity of Woburn Landfill. Woburn residents obtain their drinking water from municipal wells in southern Woburn and from MWRA sources in western Massachusetts. The MDEP Zone II protection area for Woburn's municipal wells is located over 2 miles south of the landfill. This means that even under the most severe pumping conditions at the municipal wells, groundwater beneath the landfill would not contribute to Woburn municipal water. Therefore, it is not expected that groundwater with contaminants originating from the Woburn Landfill would be consumed as municipal drinking water. However, future exposures to some metals, PAHs, and VOCs

in groundwater are possible if private drinking water wells are installed in the path of contamination.

While current information indicates that residents are not consuming contaminated groundwater from the Woburn Landfill, exposure could occur if contaminated groundwater was beneath nearby homes and VOCs migrated into indoor air in those homes. The Johnson-Ettinger model, which incorporates site-specific information on groundwater, soil, and housing, was used to assess the possibility of adverse health effects due to vapor intrusion. The modeling and subsequent calculations indicate that VOCs in groundwater from the Woburn Landfill would be unlikely to cause adverse health effects in nearby residents. In addition, potential indoor air exposures are unlikely based on the distance of homes from the landfill and the direction of groundwater away from homes.

Unfortunately, past exposure for potential trespassers to soil at the Woburn Landfill can not be evaluated due to the lack of soil sampling data prior to closure activities in 1999–2003. However, the limited information available on soil used during closure activities indicates that adverse health effects are unlikely. Current or future exposure to contaminated soil is unlikely due to physical barriers that restrict access.

Wetlands located near the southwest portion of the Woburn Landfill could have been visited by residents living nearby in the past, present and future since there are no physical barriers to restrict access. Adult and child residents may have contact with wetland sediment as well as surface water while playing in the wetlands. However, upon considering conservative exposure scenarios, adverse health effects or increased cancer risk due to exposure to contaminants in wetland sediment and surface water were determined to be unlikely.

In the past, residents may have been exposed to landfill gases on a limited basis, given the distance from the only sample where air contaminants were detected to the closest homes. Present or future exposure to landfill gases is unlikely due to the installation of an active gas collection and control system installed during closure activities. This

system is regularly monitored and, along with the completion of capping activities, has eliminated odor complaints from nearby residents.

The cancer incidence analysis in this report assessed the pattern of cancer in Woburn CT 3336, where the Woburn Landfill is located, and focused in particular on residential neighborhoods closest to the landfill. The analysis is an update of the *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999,* which concluded that the majority of the five cancer types evaluated in Woburn CT 3336 occurred approximately at or below expected rates during 1995 to 1999 (MDPH 2004). Bladder cancer and lung and bronchus cancer occurred statistically significantly more often than expected; however, data regarding smoking history for individuals diagnosed with these cancer types suggested that smoking likely played a role in the incidence of these cancers.

Using data from the MCR, cancer incidence rates were calculated for five cancer types (bladder, brain, kidney, leukemia, and lung and bronchus) from 2000 to 2003, the time period for which the most recent and complete data were available. Available information about risk factors, including environmental factors, related to the development of cancer was also considered. In addition, the pattern of cancer was evaluated in neighborhoods within CT 3336 to identify any unusual concentrations of cases.

The cancer types evaluated in this report were selected based on their potential association with contaminants of concern identified at the Woburn Landfill and residents' concerns about specific cancer types. Although conservative estimates of exposure were determined to be unlikely to result in adverse health effects or increased cancer risk, certain cancer types (e.g., bladder, brain and CNS, kidney, lung and bronchus) were evaluated because of known or suspected links to contaminants identified at the landfill. One of these cancer types evaluated was statistically significantly elevated in CT 3336 (i.e., bladder cancer). Lung and bronchus cancer, which has also been linked to arsenic exposure, occurred less often than expected. Kidney cancer, which has also been associated with arsenic, was slightly elevated; however, an analysis of risk factor

information suggested that tobacco use likely played a role in diagnoses of this cancer type for some individuals.

Bladder cancer among males and females occurred at a rate that was statistically significantly elevated in CT 3336 during 2000–2003. The geographic pattern of bladder cancer did not indicate a concentration or an atypical distribution of individuals diagnosed in the area surrounding the Woburn Landfill or in any other area of the census tract. An analysis of available risk factor information suggested that tobacco use likely played a major role in diagnoses of this cancer type for some individuals. This follows trends discussed in MDPH's *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999* where the incidence of bladder cancer showed a statistically significant elevation and data regarding smoking history for individuals diagnosed with bladder cancer suggested that smoking likely played a role in its incidence among residents in CT 3336 during 1995–1999.

Leukemia incidence in Woburn CT 3336 demonstrated that three individuals age 0–19 were diagnosed during 2000–2003, while less than one diagnosis would have been expected. As previously discussed, leukemia is classified into four main subtypes of disease that each have different risk factors. From 2000 to 2003 in CT 3336, two leukemia subtypes were diagnosed. The geographic pattern of leukemia did not indicate an atypical distribution of individuals diagnosed near the Woburn Landfill or in any other area of the census tract nor did it suggest that environmental factors played a primary role. Each place of residence at time of diagnosis for the three children diagnosed with leukemia was over 1 mile from the Woburn Landfill and a distance of approximately one half-mile separated the three diagnoses from one another. The pattern of leukemia observed in Woburn CT 3336 appears consistent with what would be expected based on the population distribution and areas of higher population density. For example, within Woburn CT 3336, the three individuals with leukemia were located in areas of the town where population and housing density is greater. For these reasons, it does not appear that the occurrence of leukemia observed among males in Woburn CT 3336 from 2000 to 2003 is related to a common factor.

In addition to an evaluation of cancer incidence rates, available risk factor information for those diagnosed with cancer was compared to known or established trends to assess whether any unexpected patterns existed in Woburn CT 3336 for the time period evaluated. In general, the cancer trends observed were similar to those seen in the general population and in Massachusetts. Available risk factor information suggest that smoking likely played some role in the diagnosis of certain cancer types (bladder, kidney, and lung and bronchus cancers) among some individuals in Woburn CT 3336. Also, occupational exposures may have played a role for some individuals in the development of all five cancer types evaluated. However, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in CT 3336 due to incomplete information for some risk factors (i.e., occupation and smoking status).

Finally, analysis of the geographic distribution of place of residence for individuals diagnosed with one of the five cancer types did not reveal any atypical spatial patterns that would suggest a common factor related to the incidence of cancer in Woburn CT 3336. That is, no unusual concentrations of individuals diagnosed with the five cancer types were observed in the vicinity of the Woburn Landfill or any other residential areas in CT 3336. Based on the information reviewed in this evaluation, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in the census tract where the Woburn Landfill is located during the 4-year time period, 2000–2003.

IX. CHILD HEALTH CONSIDERATIONS

ATSDR and MDPH recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at greater risk than adults from certain kinds of exposure to hazardous substances emitted from waste sites. They are more likely exposed because they play outdoors and because they often bring food into contaminated areas. Because of their smaller stature, they may breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of chemical 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 pattern of cancer among children in Woburn CT 3336 is discussed in Section VII ("Cancer Incidence Analysis") of this report. While three diagnoses of leukemia among individuals 0-19 years of age were observed, and less than one diagnosis would be expected, it is important to note that no diagnoses were observed in the previous 5-year time period, 1995–1999. Also, a review of the geographic distribution of these three diagnoses indicated that they were approximately 0.5 miles from each other and approximately 1 mile from the Woburn Landfill.

As discussed before, future exposure to metals, PAHs, and VOCs could be possible for children if private drinking water wells are installed in the path of contamination. The MDPH recommends that the City of Woburn carefully review future private well installation requests if proposed wells are to be installed in the path of the estimated extent of contaminated groundwater. Also, past, present, and future exposures to arsenic in sediment could be possible for children who access the site. However, based on conservative exposure estimates, it is unlikely that anyone would have contact with sediment near the Woburn Landfill for a sufficient frequency and duration of time to result in adverse health effects. No other exposures were identified that would indicate that children are more likely than adults to be impacted by the Woburn Landfill.

X. LIMITATIONS

This public health assessment is an investigation that considers descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers is unusual. The purpose of this investigation is to evaluate the patterns of cancer in a geographical context in relation to available information about factors, including environmental factors, related to cancer to see whether further investigation seems warranted. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further public health actions may be warranted. Inherent limitations in this type of analysis and the available

data make it impossible to determine the precise causal relationships or synergistic roles that may have played a part in the development of individual cancers in this community. Also, this type of analysis cannot determine what may have caused any one individual's cancer. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the disease that could not be evaluated in this report. It is believed that many cancers are related largely to behavioral factors such as cigarette smoking, diet, and alcohol consumption. Other factors associated with cancer are socioeconomic status, heredity/genetics, race, and geography. It is beyond the scope of this report to determine the causal relationship of these factors and the development of cancer or other health outcomes in Woburn CT 3336.

XI. CONCLUSIONS

- Future exposures to some metals, PAHs, and VOCs detected in groundwater at the Woburn Landfill are possible if private drinking water wells are installed down-gradient of the site. Ingestion of contaminants detected in groundwater drawn into potential future private wells could result in health concerns.

- Even under extreme drought conditions, it is unlikely that groundwater contaminants detected at the Woburn Landfill would reach the Zone II protection area for Woburn's community wells located over 2 miles south; therefore, exposures through municipal drinking water would not be expected.

- Based on the levels of VOCs detected in onsite groundwater, conservative indoor air concentrations predicted by the Johnson-Ettinger model, the general flow of groundwater beneath Woburn Landfill, and the distance of homes located west and southwest of the site, it is unlikely that contaminants detected in groundwater at the Woburn Landfill would present an exposure concern for indoor air in nearby homes.

- Intermittent exposures to surface water and sediment adjacent to the Woburn Landfill could be possible for individuals trespassing in the past, present, and future. However, based on the contaminant levels detected and the frequency and

duration of contact expected, it is unlikely that potential exposures would result in adverse health effects.

- The majority of cancer types evaluated during the 4–year time period, 2000–2003, occurred near expected rates in Woburn CT 3336, where the Woburn Landfill and nearby residences are located. Bladder cancer was statistically significantly elevated in males and females evaluated together. An evaluation of available risk factor information suggested that tobacco use likely played a possible role in diagnoses of bladder cancer for some individuals.

- While three diagnoses of leukemia among individuals 0-19 years of age were observed, and less than one would be expected, it is important to note that no diagnoses were observed in the previous 5-year time period, 1995–1999. Also, a review of the geographic distribution of these three diagnoses of leukemia indicated that the diagnoses were approximately 0.5 miles from each other and approximately 1 mile from the Woburn Landfill.

- A review of the geographic distribution of the five cancer types in Woburn CT 3336 revealed no apparent spatial patterns at the neighborhood level, including in the vicinity of the Woburn Landfill and nearby residential areas to the west and southwest.

- Based on the information reviewed in this evaluation, including available environmental data for the Woburn Landfill and risk factor information for individuals diagnosed with cancer, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer from 2000 to 2003 in CT 3336, where the Woburn Landfill is located.

ATSDR requires that one of five conclusion categories be used to summarize findings of a public health assessment. These categories are as follows: (1) Urgent Public Health Hazard; (2) Public Health Hazard; (3) Indeterminate Public Health Hazard; (4) No Apparent Public Health Hazard; (5) No Public Health Hazard. A category is selected from site-specific conditions such as the degree of public health hazard based on the

presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns. Therefore, based on MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to the cancer types evaluated in this analysis, ATSDR would classify the Woburn Landfill site as posing no apparent public health hazard to the public in the past and present. Since contaminants detected in groundwater at Woburn Landfill could be drawn into potential future private wells, the Woburn Landfill would pose a public health hazard in the future should wells be installed in contaminated groundwater areas.

XII. RECOMMENDATIONS

- The MDPH recommends that the city of Woburn develop a testing and approval process for all new private wells to ensure that contaminated groundwater from the Woburn Landfill is not consumed in the future as drinking water by nearby residents.

- The MDPH recommends that if any residents living in the vicinity of the Woburn Landfill are using private well water for non-drinking water purposes (such as filling swimming pools, watering gardens, or washing cars), that they follow EPA and MDEP guidance that recommends owners test their wells initially for all contaminants, then at a minimum of once every ten years (yearly for bacteria and nitrite/nitrate) (MDEP 2004).

- The MDPH recommends that the Woburn Board of Health be available to answer questions from residents who have concerns about the source of drinking water in their homes and the source of municipal water in Woburn.

- The MDPH recommends no further investigation of cancer incidence in relation to the Woburn Landfill at this time, but will continue its efforts to monitor cancer incidence in the city of Woburn and notably leukemia among children through the Massachusetts Cancer Registry.

XIII. PUBLIC HEALTH ACTION PLAN

The Public Health Action Plan for the Woburn Landfill contains a description of actions to be taken by the ATSDR and/or the MDPH at and in the vicinity of the site subsequent to completion of this public health assessment. The purpose of the Public Health Action Plan is to ensure that this public health assessment not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the ATSDR/MDPH to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by ATSDR/MDPH are as follows:

- Upon request, the MDPH is available to assist the Woburn Board of Health in defining a testing and approval process for new private well construction in the vicinity of the Woburn Landfill.

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

XIV. REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1993. ***Toxicological Profile for Arsenic***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. ***Toxicological Profile for Polycyclic Aromatic Hydrocarbons***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1996. ***Toxicological Profile for Methyl tert-Butyl Ether***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. ***Toxicological Profile for Cadmium***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2000a. ***Toxicological Profile for Manganese***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2001a. ***Toxicological Profile for Pentachlorophenol***. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2001b. *Landfill Gas Primer: An Overview for Environmental Health Professionals*. Atlanta: U.S. Department of Health and Human Services. November 2001.

Agency for Toxic Substances and Disease Registry (ATSDR). 2005a. ATSDR Public Health Assessment Guidance Manual (UPDATE). Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2006a. Drinking Water Comparison Values. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2006b. Soil Comparison Values. Atlanta: U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. E-mail from Gregory Zarus, environmental health scientist, concerning total volatile organic compounds (VOCs) measured in landfill gas. Atlanta. May 14, 2007.

American Cancer Society (ACS). 2005a. Brain / CNS Tumors in Children. Available at: http://www.cancer.org.

American Cancer Society (ACS). 2005b. Kidney Cancer. Available at: http://www.cancer.org.

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

American Cancer Society (ACS). 2006b. Brain / CNS Tumors in Adults. Available at: http://www.cancer.org.

American Cancer Society (ACS). 2006c. Leukemia - Acute Myeloid (AML). Available at: http://www.cancer.org.

American Cancer Society (ACS). 2006d. Leukemia - Acute Lymphocytic (ALL). Available at: http://www.cancer.org.

American Cancer Society (ACS). 2006e. Leukemia - Children's. Available at: http://www.cancer.org.

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

Berg JW. Morphologic Classification of Human Cancer. In: *Cancer Epidemiology and Prevention*, 2nd ed.; Schottenfeld D, Fraumeni JF, Eds.; Oxford University Press: New York 1996; pp 28-44.

Black PM. 1991. Brain tumors (first of two parts). New Eng J Med 324(21):1471-76.

Blot WJ, Fraumeni JF, Jr. Cancers of the lung and pleura. In: Schottenfeld D, Fraumeni JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996.

City of Woburn, 2006. "Water Department." Accessed July 2006. Available online at: http://www.woburndpw.com/WaterDept.htm

ENSR. 1998. Comprehensive Site Assessment: Volumes I, II, and III. Woburn Sanitary Landfill, Merrimac Street, Woburn, Massachusetts. January 1998.

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

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

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

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

Lynnfield Engineering (Lynnfield). 2001. Supplemental Transmittal No. 1 Application for Solid Waste Management Facility Modification BWP SW 22 Landfills - Minor Modification - Woburn Sanitary Landfill, 202 Merrimac Street, Woburn, Massachusetts. Transmittal #W 015409. October 16, 2001.

Lynnfield Engineering (Lynnfield). 2005a. Monthly Landfill Gas Monitoring Report No. 07-04, Woburn Sanitary Landfill Facility, 202 Merrimac Street, Woburn, Massachusetts. January 7, 2005.

Lynnfield Engineering (Lynnfield). 2005b. Monthly Landfill Gas Monitoring Report No. 03-05, Woburn Sanitary Landfill Facility, 202 Merrimac Street, Woburn, Massachusetts. April 19, 2005.

Maguire Group, Inc. (Maguire). 1989. Site Assessment and Closure Plan. Woburn Municipal Landfill. July 1989.

Maguire Group, Inc. (Maguire). 2001. Supplemental Transmittal Application for Solid Waste Management Facility Modification BWP SW 22 – Landfill – Minor Modification -

Woburn Sanitary Landfill, 202 Merrimac Street, Woburn, Massachusetts. Transmittal No. W015409. December 2001.

Maguire Group, Inc. (Maguire). 2002a. Woburn Sanitary Landfill Final Closure: Grading and Shaping Monthly Progress Report No. 37. August 2002.

Maguire Group, Inc. (Maguire). 2002b. Application for Solid Waste Management Facility Minor Modification for Woburn Sanitary Landfill, Woburn, Massachusetts. BWP SW 22. Transmittal No. W029105. May 2002.

Maguire Group, Inc. (Maguire). 2005. Supplemental Comprehensive Site Assessment and Post Closure Monitoring Plan. Woburn Sanitary Landfill, Woburn, Massachusetts. March 2005.

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

Massachusetts Cancer Registry (MCR). Cancer incidence and mortality in Massachusetts 1999-2003: statewide report. Boston: Massachusetts Department of Public Health, Bureau of Health Statistics, Research and Evaluation; 2006.

Massachusetts Department of Environmental Protection (MDEP). 1995. Guidance for Disposal Site Risk Characterization In Support of the Massachusetts Contingency Plan. Interim Final Policy #WSC/ORS-95-141. July 1995.

Massachusetts Department of Environmental Protection (MDEP). 1997. The Massachusetts Contingency Plan 310 CMR 40.0000. Massachusetts Department of Environmental Protection, Bureau of Hazardous Waste Site Cleanup. May 1997.

Massachusetts Department of Environmental Protection (MDEP). 2002. Report on the Closure and Capping of the City of Woburn's Landfill. March 19, 2002.

Massachusetts Department of Environmental Protection (MDEP) 2004. Private Well Guidelines. Drinking Water Program.

Massachusetts Department of Environmental Protection (MDEP). 2005. Letter to Mr. Jack Fralick from Mr. John Morey RE: Woburn – Solid Waste – Woburn Sanitary Landfill – Supplemental Comprehensive Site Assessment Conditional Approval FMF 39928. Boston, MA. June 8, 2005.

Massachusetts Department of Environmental Protection (MDEP). 2006a. Standards and Guidelines for Contaminants in Massachusetts Drinking Waters.

Massachusetts Department of Environmental Protection (MDEP). 2006b. Bureau of Waste Site Cleanup. Downloadable Site Lists. Available at: http://www.mass.gov/dep/cleanup/sites/sdown.htm (accessed July 2006).

Massachusetts Department of Public Health (MDPH). 2004. Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999.

Massachusetts Water Resources Authority (MWRA) "How the MWRA Water System Works." Accessed July 2006. Available online at http://www.mwra.state.ma.us

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

National Cancer Institute (NCI). Brain and other nervous system. In: Harras A, editor. Cancer rates and risks. 4th ed. Washington, D.C.: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health; 1996.

Preston-Martin S, Mack W. Neoplasms of the nervous system. In: Schottenfeld D, Fraumeni, JF, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 1231-81.

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

Samet JM, Eradze GR. 2000. Radon and lung cancer risk: taking stock at the millennium. Environ Health Perspect 108(suppl 4):635-41.

Shacklette HT, Boerngen JG. 1984. Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States. U.S. Geological Survey Professional Paper 1270. United States Printing Office, Washington: 1984.

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

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

U.S. Department of Commerce (U.S. DOC). 2002. Massachusetts: 2000 Summary Population and Housing Characteristics, 2000 Census of Population and Housing. U.S. Department of Commerce, Washington, DC: US Government Printing Office.

U.S. Environmental Protection Agency Region 1(USEPA). 2002. "Blox Chemical Site Fact Sheet and Narrative." Accessed on June 28, 2006. Available online at: http://www.epa.gov/region1/superfund/findsite/fndindex.htm

U.S. Environmental Protection Agency (USEPA). 2004a. User's Guide for Evaluating Subsurface Vapor Intrusion into Buildings. Office of Emergency and Remedial Response. February 22, 2004.

U.S. Environmental Protection Agency (USEPA). 2004b. Johnson and Ettinger (1991) Model for Vapor Intrusion into Buildings. Version 3.1. February. Available on the Web at: *http://www.epa.gov/oswer/riskassessment/airmodel/johnson_ettinger.htm*

U.S. Environmental Protection Agency Region 9 (USEPA). 2004c. Region 9 Preliminary Remediation Goals. Available from: http://www.epa.gov/region9/waste/sfund/prg/index.html

U.S. Environmental Protection Agency (USEPA). 2006a. Record of Decision: Industri-Plex Superfund Site Operable Unit-2 (and Including Wells G&H Superfund Site Operable Unit-3, Aberjona River Study) City of Woburn, Middlesex County, Massachusetts. January 2006.

U.S. Environmental Protection Agency (USEPA). 2006b. CERCLA Overview. Accessed on June 28, 2006. Available from: http://www.epa.gov/superfund/action/law/cercla.htm

U.S. Environmental Protection Agency Region I (USEPA). 2006c. Industri-Plex Site Fact Sheet and Narrative. Accessed on June 28, 2006. Available from: http://www.epa.gov/region1/superfund/findsite/fndindex.htm

U.S. Environmental Protection Agency Region I (USEPA). 2006d. Olin Chemical Site Fact Sheet and Narrative. Accessed on June 28, 2006. Available from: http://www.epa.gov/region1/superfund/findsite/fndindex.htm

U.S. Environmental Protection Agency (USEPA). 2006e. National Priorities List Olin Chemical. Accessed on December 5, 2006. Available from: http://www.epa.gov/superfund/sites/npl/nar1741.htm.

U.S. Environmental Protection Agency (USEPA). 2006f. EPA Region III Risk Based Concentrations Table.

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, Bureau Director of BEH/MDPH at 250 Washington Street, 7th Floor, Boston, MA 02108.

XV. CERTIFICATION

The Public Health Assessment, *Evaluation of Cancer Incidence, 2000–2003, and Environmental Concerns Related to the Woburn Landfill in Woburn, Middlesex County, Massachusetts,* was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Public Health Assessment was initiated. Editorial review was conducted by MDPH.

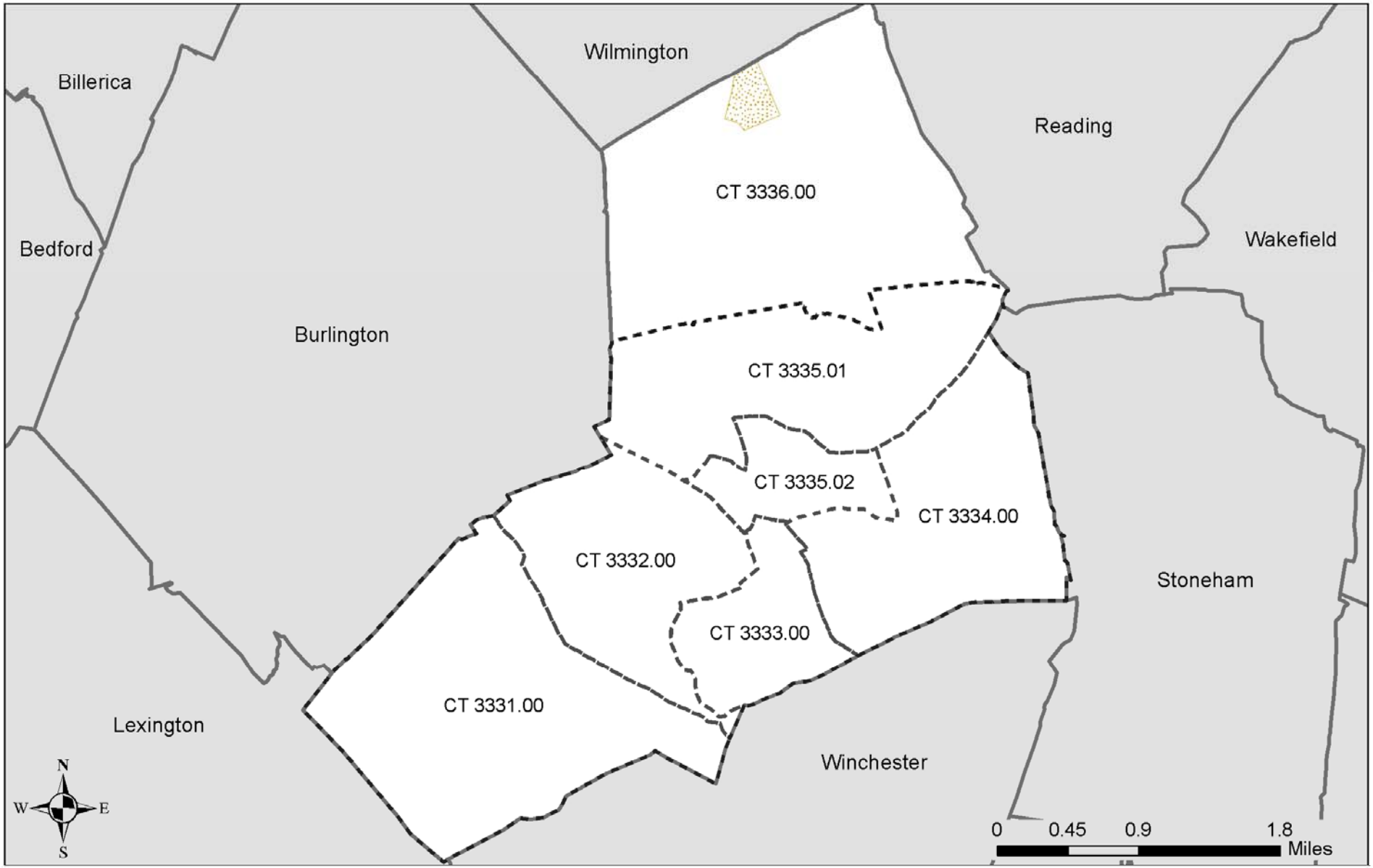
Technical Project Officer, CAT, CAPEB, DHAC, ATSDR

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

Team Lead, CAT, SPAB, DHAC

Figures

Figure 1
Location of Census Tracts
Woburn, Massachusetts

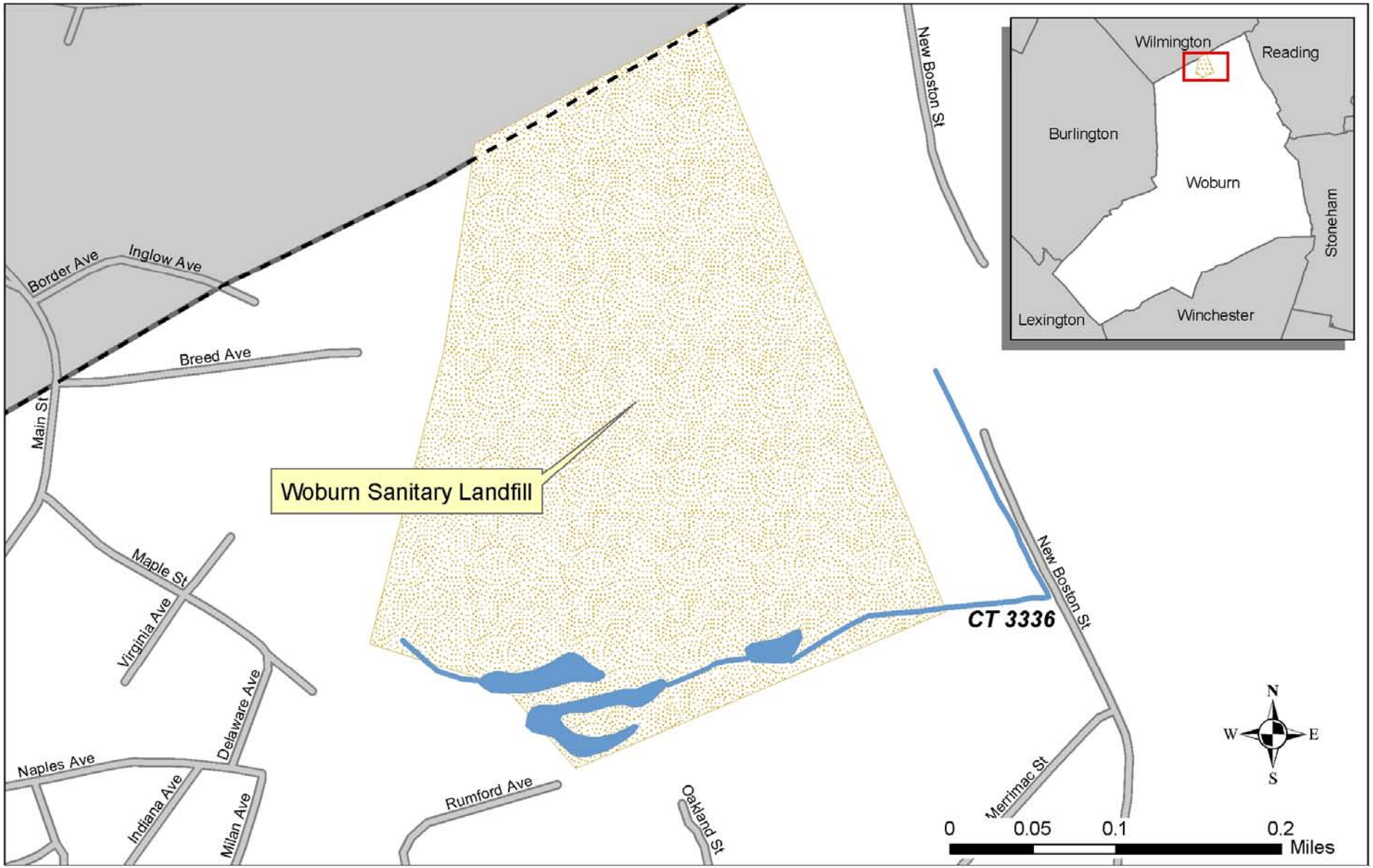


Bureau of BEH Environmental Health

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

Woburn Landfill
Town Boundary
Census Tract Boundary

Figure 2
Location of Woburn Landfill
Woburn, Massachusetts



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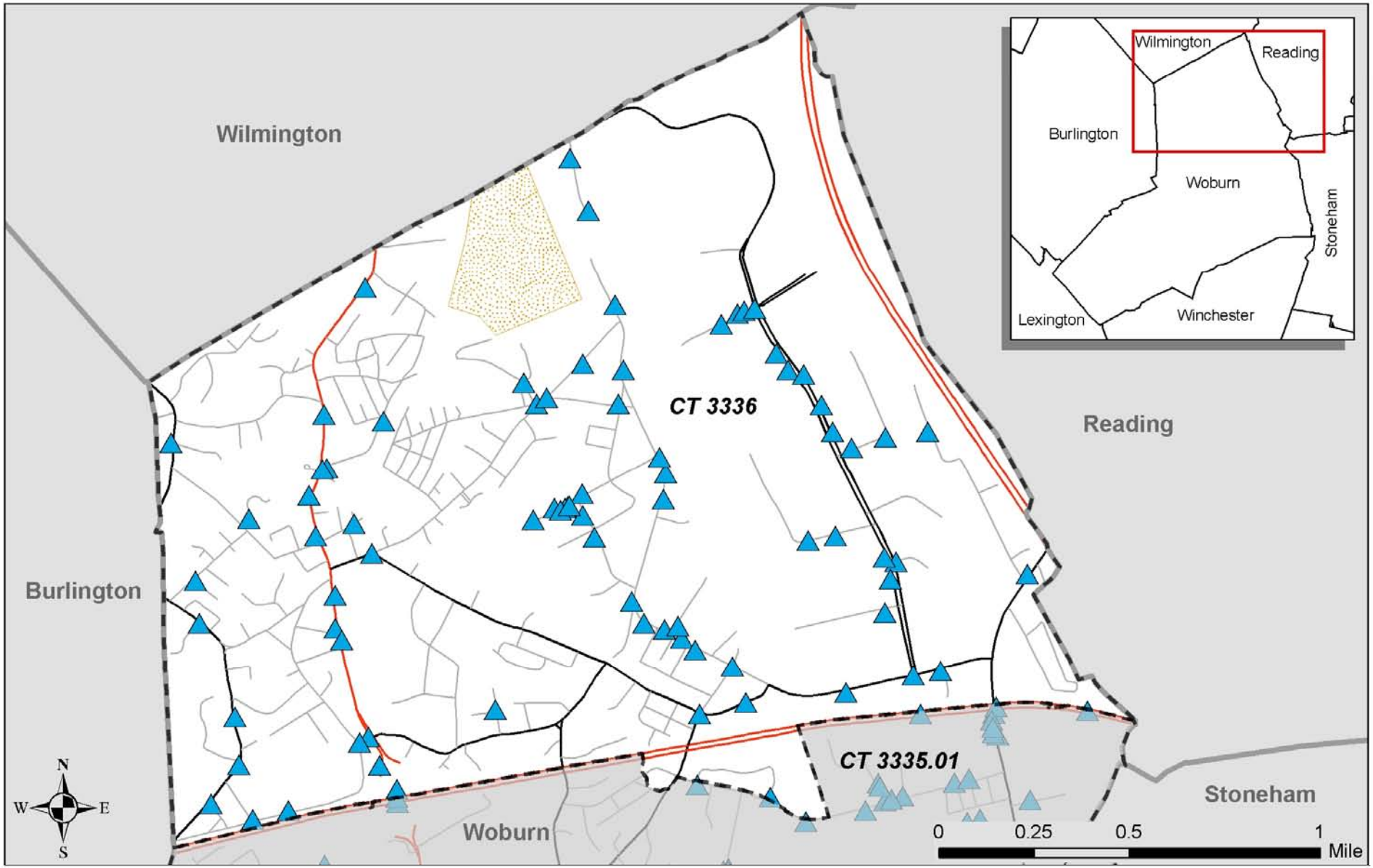
Geographic data supplied by: Massachusetts Executive Office of Environmental Affairs, MassGIS; Geographic Data Technology, Inc.

Landfill
Road
Census Tract Boundary
Town Boundary
Approx. Location of Brook

Figure 3
Location of MDEP 21E Hazardous Material and Oil Releases in Census Tract (CT) 3336
Woburn, Massachusetts

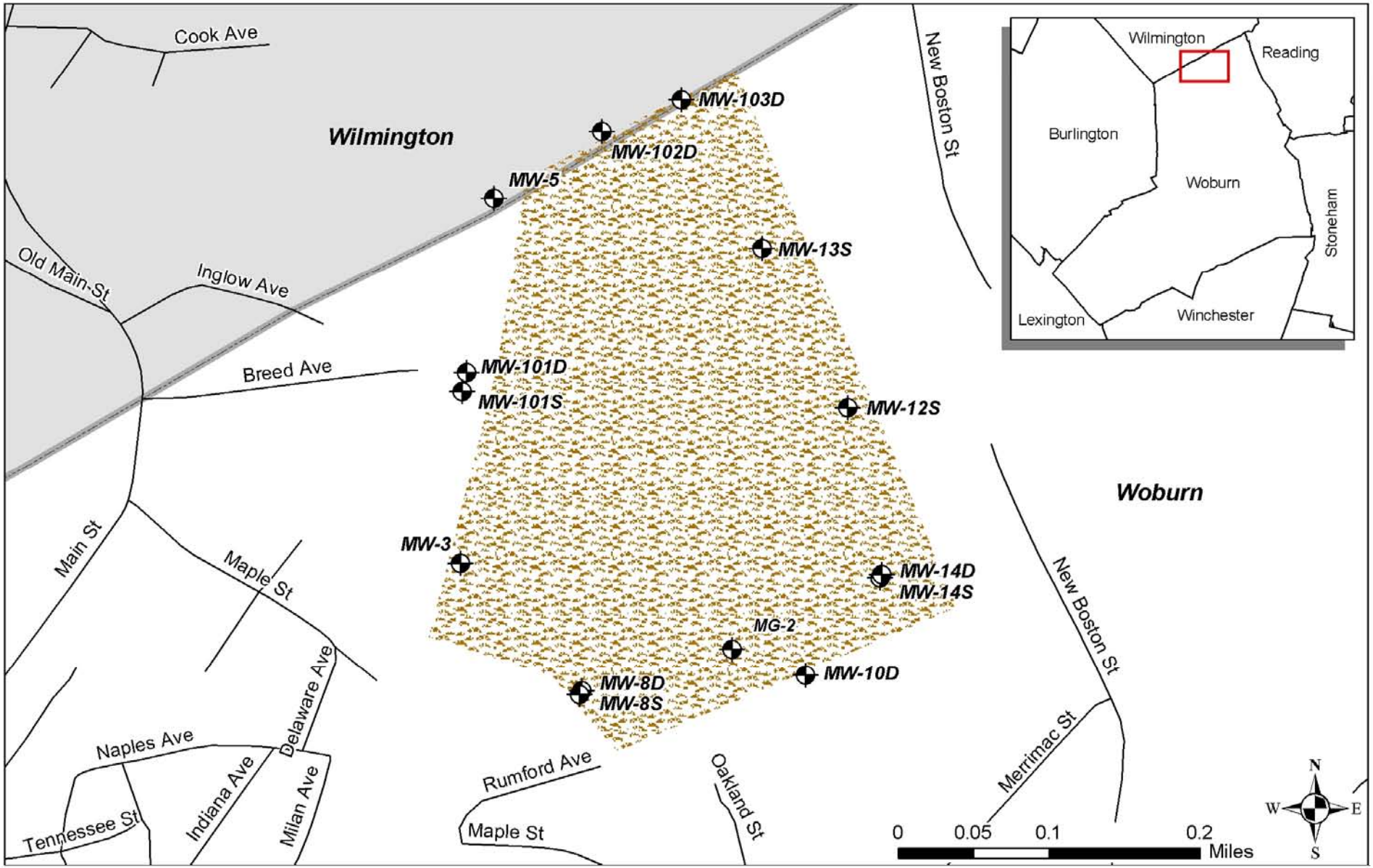


Bureau of BEH Environmental Health

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

MDEP 21E Site
Woburn Landfill
Town Boundary
Census Tract (CT) Boundary
Limited Access Highway
Primary Highway
Secondary Highway
Local Road

Figure 4
Approximate Locations of Groundwater Monitoring Wells at Woburn Landfill
Woburn, Massachusetts

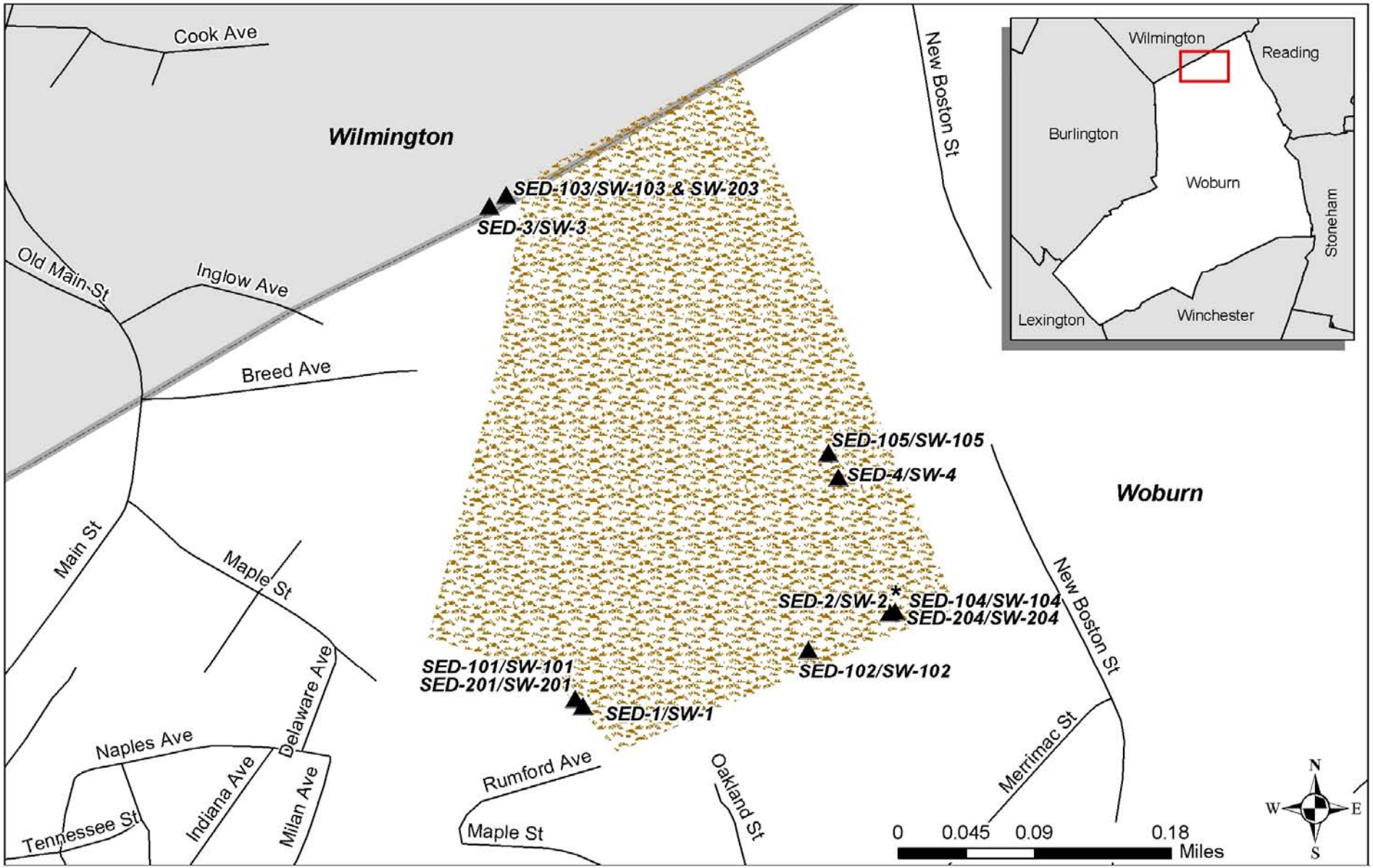


Bureau of BEH Environmental Health

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

Monitoring Well
Local Road
Woburn Landfill
Town Boundary

Figure 5
Approximate Locations of Surface Water and Wetland Soils and Sediment Samples at Woburn Landfill
Woburn, Massachusetts



Bureau of BEH Environmental Health

▲ Sediment/Surface Water Sample
Woburn Landfill
Local Road
Town Boundary

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

* SED-2 includes S2-1 through S2-10, SED2-D-1, and SED2-D-2, which were taken at different depths and at 30-foot intervals east and west of SED-2. (See text for further discussion.)

Tables

Table 1

Massachusetts Department of Environmental Protection (MDEP) 21E Hazardous Material and Oil Releases (1980 - 2003[1,2]) in Census Tract 3336
Woburn, Massachusetts

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
3-0021944		36 SIXTH RD	7/17/2002	FUEL OIL #2 (100 PPMV), PETROLEUM BASED OIL (122 PPMV)	UST	RAO
3-0021905	COMMERCIAL, MUNICIPAL, OPENSPACE	NEW BOSTON RD	6/30/2002	UNKNOWN CHEMICAL OF TYPE - OIL	DRUMS	RAO
3-0021848	RESIDENTIAL	954 MAIN ST	6/13/2002	FUEL OIL #2 (200 GAL), FUEL OIL #2 (275 GAL)	AST	RAO
3-0020664	COMMERCIAL	2 ELM ST	5/3/2001	PETROLEUM BASED OIL (20 GAL), PETROLEUM BASED OIL (30 GAL)	UST	RAO
3-0020469	COMMERCIAL	78 DRAGON CT	3/13/2001	DIESEL FUEL (20 GAL), DIESEL FUEL (30 GAL)	AST	RAO
3-0020233	COMMERCIAL, INDUSTRIAL, ROADWAY	185 NEW BOSTON RD	12/18/2000	UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (40 GAL)	TRANSFORM	RAO
3-0019894	INDUSTRIAL	8 COMMONWEALTH AVE	8/31/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (73 LBS), UNKNOWN CHEMICAL OF UNKNOWN TYPE (75)	DRUMS	RAO
3-0019740	ROADWAY	48 6TH RD	7/19/2000	DIESEL FUEL (50 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE	SADDLETANK	RAO
3-0019395	INDUSTRIAL	101 COMMERCE WAY	3/25/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE (11 GAL)	VEHICLE	RAO
3-0019334	ROADWAY	NEW INDUSTRIAL RD	3/3/2000	DIESEL FUEL (25 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	VEHICLE	RAO
3-0019323	ROADWAY	EAST NICHOLS ST APP CT	2/26/2000	PETROLEUM BASED OIL, PETROLEUM BASED OIL (3 GAL)	PIPE	RAO
3-0017904	COMMERCIAL, INDUSTRIAL, RESIDENTIAL	32 WEBSTER ST	2/4/1999	CYANIDE, PHENANTHRENE (82 PPB), TOTAL PETROLEUM HYDROCARBONS (TPH) (5250 PPM), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL	ABANDONED, PLATE SHOP	RAO

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
3-0017242	COMMERCIAL	80 COMMERCE WAY	9/1/1998	MINERAL OIL (120 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (200 GAL)	TRANSFORM	RAO
3-0015393	COMMERCIAL, INDUSTRIAL	225 MERRIMAC ST	8/6/1997	MINERAL OIL (120 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (120 GAL)	TRANSFORM	RAO
3-0015228	COMMERCIAL	181 NEW BOSTON ST	6/20/1997	1,1'-BIPHENYL, CHLORO-DERIVS. (121 MG/KG), 1,1'-BIPHENYL, CHLORO-DERIVS. (211 MG/KG), MINERAL OIL (40 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL)	TRANSFORM	RAO
3-0015197	COMMERCIAL, ROADWAY	MERRIMAC ST	6/12/1997	DIESEL FUEL (60 GAL)	PIPE	RAO
3-0014790	COMMERCIAL	10 INDUSTRIAL PARK DR	2/4/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	PIPE	RAO
3-0014776	COMMERCIAL, INDUSTRIAL	23 ATLANTIC AVE	1/29/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	VEHICLE	RAO
3-0013150	COMMERCIAL	100 SYLVAN RD	1/9/1997	ASBESTOS	ASBESTOS, IMPROPER, REMOVAL	RAO
3-0014639	INDUSTRIAL	ATLANTIC AVE	12/11/1996	FUEL OIL #6 (36200 MG/KG), TOTAL PETROLEUM HYDROCARBONS (TPH) (12 MG/L)	UST	ADQREG
3-0013856	COMMERCIAL	30 COMMERCE WAY	6/5/1996	FUEL OIL #2 (110 PPMV)	UST	RAO
3-0013017	INDUSTRIAL	ATLANTIC AVE COMMERCE WAY	10/9/1995	PETROLEUM BASED OIL (40 GAL)	PIPE, VEHICLE	RAO
3-0012506	SUPERFUND	41 ATLANTIC AVE	5/24/1995	PETROLEUM BASED OIL (55 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (55 GAL)	PIPE, VEHICLE	RAO
3-0012396	COMMERCIAL, INDUSTRIAL	COMMERCE AND ATLANTIC	4/17/1995	ASBESTOS	UNKNOWN	RAO
3-0012114	COMMERCIAL	482 WASHINGTON ST	1/24/1995	GASOLINE (8.88 INCH)	UNKNOWN	TIER1C

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
3-0011396	RESIDENTIAL	3 FOREST GLEN CIR	7/30/1994	FUEL OIL #2 (100 GAL), FUEL OIL #2 (70 GAL)	AST	RAO
3-0010859		181 NEW BOSTON ST	4/14/1994	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (.25 INCH)	UNKNOWN	RAONR
3-0010759	DRAINAGE, RESIDENTIAL	36R WINTER ST	3/28/1994	FUEL OIL #2, LUBRICATING OIL (2 GAL)	UNKNOWN	RAO
3-0010733	COMMERCIAL, INDUSTRIAL	8 COMMONWEALTH AVE	3/23/1994	CHLOROUS ACID, SODIUM SALT (500 GAL), HYPOCHLOROUS ACID, SODIUM SALT	AST	RAO
3-0010578	COMMERCIAL, INDUSTRIAL	64 INDUSTRIAL PARK WAY	2/14/1994	2-BUTANONE, BENZENE, METHYL-, BENZENE, METHYL- (10 GAL)	PIPE	RAO
3-0010002	COMMERCIAL	83 COMMERCE WAY	10/1/1993	DIESEL FUEL, FUEL OIL #2	PIPE, UNKNOWN, UST	RAO
N93-1289	ASBESTOS	COMMERCE WAY/ATLANTIC AVE	9/22/1993	ASBESTOS (1-10)	UNKNOWN	
N93-0841	J AMICONE CO INC	324 NEW BOSTON ST	6/23/1993	OTHER MATERIAL, SOY BEAN OIL (GAL)	A.S.T.	
N93-0792	TOYS R US	299 MISHAWUM RD	6/8/1993	OTHER MATERIAL, PETROLEUM (UNK)	UNKNOWN	
N93-0454	SADDLE TANK	134 NEW BOSTON ST	4/13/1993	DIESEL FUEL (GAL)	VEH. FUEL TANK	
N93-0393	LA DONOVAN CO	32 WEBSTER ST	4/5/1993	OTHER MATERIAL, CYANIDE & CHROMIC ACID (UNK)	DRUMS	
N93-0104	INDUSTRIPLEX	ATLANTIC AVE	1/26/1993	HYDRAULIC FLUID (GAL)	PIPE/HOSE/LINE	
N92-1163	UST	17 GATTA CIR	9/10/1992	#2 FUEL OIL (UNK)	U.S.T.	
N92-0853	RESIDENTIAL DUMPING	19 PEARL ST	7/4/1992	WASTE OIL (<1 GAL)	OTHER SOURCE, CRANKCASE	
N92-0759	TRUCK ROLLOVER	RTE 38 @ RTE 128	6/15/1992	DIESEL FUEL (101-250 GAL)	VEH. FUEL TANK	
N91-1188	ABOVE-GRND TANK LEAK	355 MISHAWAM RD	8/24/1991	#2 FUEL OIL (1-10 GAL)	ABOVE-GRND TANK	
N91-1121	CHROMERICS	8 COMMONWEALTH AVE	8/15/1991	OTHER MATERIAL, BLEACH (15% NA HYPOCHLORIE)	PIPE/HOSE/LINE	
N91-1022	CHOMERICS	8 COMMONWEALTH AVE	7/25/1991	OTHER MATERIAL, BLEACH/SODIUM HYPOCHLORITE	PIPE/HOSE/LINE	
N91-0999	HAZARDOUS WASTE DUMPING	17 EVERBERG RD (REAR)	7/20/1991	UNKNOWN, UNKNOWN HAZARDOUS MATTER (1-10)	OTHER SOURCE, CANS	

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
N91-0913	HALL'S BROOK	SCHOOL ST & MERRIMAC ST	7/5/1991	MISCELLANEOUS OIL		
N91-0460	REPAIR OIL SPILL	2 BARTLET DR	4/4/1991	MISCELLANEOUS OIL		
N91-0239	RTE 38/RTE 128 ROTARY	RTE 38/RTE 128 ROTARY	2/21/1991	#2 FUEL OIL (251-500 GAL)	TANKER TRUCK	
N90-1882		20 COMMERCE WAY	11/14/1990	#2 FUEL OIL (UNK)	PIPE/HOSE/LINE	
N90-1162		85 6TH RD	7/17/1990	BATTERIES/BATTERY ACID (<1 GAL)	OTHER SOURCE, SM PKG	
N90-0950		181 NEW BOSTON ST.	6/13/1990	OTHER MATERIAL, HEXANE (UNK)	PIPE/HOSE/LINE	
N90-0735		RTE 128 S.WASHINGTON/RTE 38	5/10/1990	DIESEL FUEL (10-50 GAL)	VEH. FUEL TANK	
N90-0271	KEYBOARD CARRIAGE, INC	5 WHEELING AVE	2/27/1990	DIESEL FUEL (10-50 GAL)	VEH. FUEL TANK	
N90-0215	AC DISPOSAL CO	1071 MAIN ST.	2/10/1990	OTHER MATERIAL, SHEEN (UNK)	OTHER SOURCE, UNKNOWN	
N89-2154		20 CABOT ST	12/28/1989	OTHER MATERIAL, CLYCOHEXANE (FUMES)	OTHER SOURCE, 5 GAL JUG	
N89-1948	FLEET SALES	91 COMMERCE WAY	11/16/1989	MISCELLANEOUS OIL (UNK)	DRUM	
N89-1901		3 OAKLAND ST.	11/8/1989	DIESEL FUEL (1-10 GAL)		
N89-1753		110 COMMERCE WAY	10/18/1989	OTHER MATERIAL, CU PLATING SOL'N (UNK)	DRUM	
N89-1450		42 SIXTH ST	8/27/1989	OTHER MATERIAL, PESTICIDES (UNK)	OTHER SOURCE, FIRE	
N89-1142	BOSTON EDISON CO	2 GILL ST, POLE # 2	8/11/1989	TRANSFORMER OIL (10-50 GAL)	TRANSFORMER	
N89-1192		175-181 NEW BOSTON ST	7/18/1989	DIESEL FUEL (UNK)	VEH. FUEL TANK	
N89-0574		MISHAWAUM & COMMERCE ST.	4/18/1989	LUBRICATING OIL (10-50 GAL)	DRUM	
N88-1723		CABOT RD	11/1/1988	#2 FUEL OIL (NONE)	VEH. FUEL TANK	
N88-1708		185 NEW BOSTON STREET	10/25/1988	HYDROGEN SELENIDE (NONE)		
N87-1070	THURSTON MOTOR LINES	181 NEW BOSTON ST	8/4/1987	DIESEL FUEL (51-100 GAL)	VEH. FUEL TANK	
N87-1063		8 COMMONWEALTH AVE	8/3/1987	SODIUM HYPOCHLORIDE (NONE)		
N87-1062		185 NEW BOSTON ST.	8/2/1987	HYDROGEN SELENIDE (NONE)		
N87-1025	CVD INC	185 NEW BOSTON ST.	7/26/1987	ZINC, DUST (NONE)		
N87-0984		185 NEW BOSTON ST.	7/22/1987	HYDROGEN SULFIDE (NONE)		
N87-0888	WOBURN MA	945 MAIN ST.	7/2/1987	GASOLINE (NONE)	U.S.T.	
N87-0805		185 NEW BOSTON ST	6/27/1987	HYDROGEN SELENIDE		
N87-0770		185 NEW BOSTON ST	6/10/1987	HYDROGEN SULFIDE GAS		
N87-0753		185 NEW BOSTON ST	6/8/1987	HYDROGEN SULFIDE GAS		

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
N87-0690		185 NEW BOSTON RD	5/22/1987	CHEMICAL RELEASE		
N87-0615		185 NEW BOSTON ST	5/7/1987	SULFUR GAS		
N87-0556		MISHAWUM RD/RYAN RD/SCHOOL ST	4/23/1987	DIESEL FUEL		
N87-0086		MISHAWAM RD/NEAR PARKING MBTA	1/26/1987	RESINS MATERIAL		
N87-0034		10 GILL ST	1/12/1987	DIESEL FUEL		
N86-0811		20 SYLVAN STREET	9/2/1986	NO. 4 FUEL OIL		
N86-0749		39 INDUSTRIAL WAY	8/18/1986	HYDROGEN SULFIDE ODOR		
N86-1116		110 A COMMERCE WAY	7/31/1986	FORMALDEHYDE		
N86-0626		22 NO. MAPLE STREET	7/22/1986	DIESEL FUEL	U.S.T.	
N86-0482		RT. 128 SO. LANE AT RT. 38	6/12/1986	DIESEL FUEL		
N86-0464		20 COMMERCE WAY	6/7/1986	NO.2 OIL	U.S.T.	
N86-0417		20 SYLVAN RD.	5/28/1986	MIXED WASTE		
N86-0280		185 NEW BOSTON ST.	4/23/1986			
N86-0266		33 COMMONWEALTH AVE. -HARVEY I	4/16/1986	DIESEL FUEL		
N86-0085		181 NEW BOSTON ST.	2/10/1986	UNKNOWN		
N85-0921		3C COMPANY 181 NEW BOSTON ST.	12/5/1985	DIESEL FUEL		
N85-0845		891 MAIN ST.	11/4/1985	GASOLINE		
N85-0709		WOBURN MALL REAR OF MARKET BAS	9/13/1985	DIESEL FUEL		
N85-0649		880 MAIN ST.	8/26/1985	DIESEL FUEL		
N85-0623		920 MAIN ST.	8/19/1985	DIESEL FUEL		
N85-0609		482 WASHINGTON ST.	8/7/1985	WASTE OIL		
N85-0561		891 MAIN ST.	7/24/1985	GASOLINE		
N85-0413		38 6TH RD.	6/6/1985	YELLOW MATERIAL		
N85-0414		27 6TH RD.	6/6/1985	UNKNOWN SOLVENT		
N84-0335		RT 128 & RT.38	6/6/1984	DIESEL FUEL		
N84-0181		110 COMMERCE WAY	3/15/1984	PYRIDINE		
N84-0137		20 COMMERCE WAY	3/7/1984	#2 FUEL OIL		
N83-0350		39 INDUSTRIAL WAY	10/26/1983	OTHER MATERIAL, METHANE GAS		
N83-0218		980 MAIN ST	7/21/1983	DIESEL FUEL		
N83-0187		3 ELM ST.	7/2/1983	#2 FUEL OIL		
N82-5211		5-7 6th RD.	10/16/1982	HAZARDOUS WASTE		

Spill ID/RTN	Location Aid	Address	Notification Date	Materials	Source	Current Status
N82-5152		INTERSECTION COMM. WAY & MISHI	6/28/1982			
N82-5120		35 INDUSTRIAL PARKWAY	5/14/1982	DIESEL FUEL		
N82-5092		11 WEST DEXTER AVE.	4/13/1982	#2 FUEL OIL		
N82-5071		33 MILAN AVE.	3/12/1982	#2 FUEL OIL		
N81-5064		RTS.128 & 38	3/12/1981	#2 FUEL OIL		
N87-0848		309 NEW BOSTON STREET		UNKNOWN (NONE)		
N89-0366	KIMBALL COURT APT	PEARL STREET		WASTE OIL (1-10 GAL)		
N90-1925	SHELL STATION	875 MAIN ST.		OTHER MATERIAL, POSSIBLY RADIOACTIVE WAST (UNK)	DRUM	
N88-0122	MARSHALLS	83 COMMERCE WAY		#2 FUEL OIL (NONE)	U.S.T.	
N88-0306		ATLANTIC AVE		UNKNOWN (251-500 GAL)	DRUM	
N88-0505	MOBIL STATION	780 MAIN STREET		GASOLINE (NONE)	U.S.T.	
N88-1082		9 NORTH MAPLE STREET		DUCT LINE ADHESIVE (NONE)	OTHER SOURCE, BURNING OF RESI	
N88-1503	DOBBINS AUTO	65 PEARL STREET		WASTE OIL (NONE)		
N88-2014		35 INDUSTRIAL PARKWAY		OTHER MATERIAL, PHOTO CHEMICALS (NONE)		
N89-0124		205-207 NEW BOSTON STREET		UNKNOWN (NONE)		
N89-0132		29 COMMERCE WAY		SEWAGE (51-100 GAL)	PIPE/HOSE/LINE	
N89-0157	OLYMPIA ROAD	30 COMMERCE WAY		DIESEL FUEL (NONE)		

[1] If a site identified prior to 1993 was not closed out (i.e., cleanup was not complete) by 1993, it was carried forward into the new MDEP 21E database designed by the Bureau of Waste Site Cleanup in 1993.

[2] This table includes releases categorized by 2-hour or 72-hour reporting categories. See text Section IV (B) for further discussion.

Data Source: MDEP. 2006. Bureau of Waste Site Cleanup 21E Sites Database. http://www.mass.gov/dep/cleanup/sites/sdown.htm. Information contained in this table is presented as downloaded. Accessed July 2006.

Notes:
Spill ID/RTN - Identification number or release tracking number (RTN) assigned to the spill/release
Location Aid - Additional information regarding the location of the spill/release
Address - Street location of spill/release
Notification Date - Date MDEP was notified of the spill/release
Materials - Information regarding specific chemicals (and amounts)
Sources - Origin(s) of release contamination. Definitions: AST Aboveground Storage Tank; UST Underground Storage Tank
Current Status - Remediation status of release. Definitions: ADQREG Adequately Regulated; RAO Response Action Outcome; RAONR Response Action Outcome Not Required; REMOPS Remedy Operation Status

GAL - Gallon
LBS - Pounds
MG/KG - milligrams per kilogram
MG/L - milligrams per liter
PPMV - Parts per million by volume

Table 2
Maximum concentrations of contaminants detected in groundwater samples at the Woburn Landfill that exceeded comparison values (samples taken from 1988 and 1997 - 2004)

Contaminant	Detection Frequency (# detected/total samples)	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Benzene	13 / 33	Mar-97	MW-14D (Southeast end of landfill)	13	CREG = 0.6 RMEG (child) = 40 RMEG (adult) = 100 U.S. EPA MCL & MDEP MMCL = 5
Chloroethane	5 / 33	Mar-97	MW-14D (Southeast end of landfill)	11	EPA RBC = 3.6 EPA PRG = 4.6
1,4-Dichlorobenzene	1 / 33	Jul-04	MW-14S (Southeast end of landfill)	5.5	Intermediate EMEG (child) = 700 Intermediate EMEG (adult) = 2,000 LTHA = 75 U.S. EPA MCL = 75 MDEP MMCL = 5
Methyl tert-butyl ether (MTBE)	1 / 30	Jul-04	MW-8D (Southwest end of landfill, north of Rumford Avenue)	3.7	Intermediate EMEG (child) = 3,000 Intermediate EMEG (adult) = 10,000 EPA PRG = 11 EPA RBC = 2.6
Trichloroethylene (TCE)	1 / 30	Aug-97	MW-14S (Southeast end of landfill)	0.9	EPA RBC = 0.026 EPA PRG = 0.028 U.S. EPA MCL & MDEP MMCL = 5
Bis(2-Ethylhexyl)phthalate	4 / 26	May-97	MW-14D (Southeast end of landfill)	38	Chronic EMEG (child) = 600 Chronic EMEG (adult) = 2,000 CREG = 3 U.S. EPA MCL & MDEP MMCL = 6
Carbazole	3 / 23	Mar-97	MW-13S (Northeast side of landfill)	26	EPA RBC = 3.3 EPA PRG = 3.4
Dibenzofuran	3 / 26	Mar-97	MW-13S (Northeast side of landfill)	29	EPA PRG = 12
4,6-Dinitro-2-methylphenol	2 / 26	Mar-97	MW-13S (Northeast side of landfill)	65	Intermediate EMEG (child) = 40 Intermediate EMEG (adult) = 100

Table 2
Maximum concentrations of contaminants detected in groundwater samples at the Woburn Landfill that exceeded comparison values (samples taken from 1988 and 1997 - 2004)

Contaminant	Detection Frequency (# detected/total samples)	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
N-Nitrosodiphenylamine	5 / 26	Mar-97	MW-14D (Southeast end of landfill)	23	CREG = 7 EPA PRG/RBC = 14
Pentachlorophenol	4 / 26	May-97	MW-13S (Northeast side of landfill)	50	Chronic/Intermediate EMEG (child) = 10 Chronic/Intermediate EMEG (adult) = 40 CREG = 0.3 U.S. EPA MCL & MDEP MMCL = 1
Benzo(a)anthracene	7 / 26	Mar-97	MW-13S (Northeast side of landfill)	3.3	EPA RBC = 0.03 EPA PRG = 0.092
Benzo(b)fluoranthene	6 / 26	Mar-97	MW-13S (Northeast side of landfill)	1.5	EPA RBC = 0.03 EPA PRG = 0.092
Benzo(a)pyrene	5 / 26	Mar-97	MW-13S (Northeast side of landfill)	0.9	CREG = 0.005 U.S. EPA MCL & MDEP MMCL = 0.2
Dibenzo(a,h)anthrancene	2 / 26	Mar-97	MW-13S (Northeast side of landfill)	0.1	EPA RBC = 0.003 EPA PRG = 0.0092
Indeno(1,2,3-cd)pyrene	5 / 26	Mar-97	MW-13S (Northeast side of landfill)	0.3	EPA RBC = 0.03 EPA PRG = 0.092
Naphthalene	10 / 26	Mar-97	MW-13S (Northeast side of landfill)	130	Intermediate EMEG (child) = 6,000 Intermediate EMEG (adult) = 20,000 RMEG (child) = 200 RMEG (adult) = 700 EPA LTHA = 100
Arsenic	3 / 31	Jul-04	MW-14S (Southeast end of landfill)	50	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3 Chronic EMEG (Adult), RMEG (adult) = 10 U.S. EPA MCL & MDEP MMCL = 10
Lead	6 / 33	1988	MG-2 (Unknown location)	160	U.S. EPA MCL & MDEP MMCL** = 15 MCLG = 0
Manganese	5 / 33	1988	MG-2 (Unknown location)	2,500	RMEG (child) = 500 RMEG (adult) = 2000

Table 2
Maximum concentrations of contaminants detected in groundwater samples at the Woburn Landfill that exceeded comparison values (samples taken from 1988 and 1997 - 2004)

Data sources:
Maguire Group, Inc. 1989. Site Assessment and Closure Plan. Woburn Municipal Landfill. July 1989.
Maguire Group, Inc. 2005. Supplemental Comprehensive Site Assessment and Post Closure Monitoring Plan. Woburn Sanitary Landfill, Woburn, Massachusetts. March 2005.

Notes:
ppb - Parts per billion
MW - Monitoring well

Comparison values (source organization, reference):
CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR, ATSDR 2006a)
Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR, ATSDR 2006a)
Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, 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, ATSDR 2006a)
LTHA = Lifetime Health Advisory (U.S. EPA, ATSDR 2006a)
MCLG = Maximum Contaminant Level Goal for drinking water (U.S. EPA, ATSDR 2006a) (** = Action level for lead. Action must be taken if more than 10% of tap water samples exceed this value.)
U.S. EPA MCL = United States Environmental Protection Agency Maxiumum Contaminant Level for drinking water (U.S. EPA, ATSDR 2006a)
MDEP MMCL = Massachusetts Department of Environmental Protection Maxiumum Contaminant Level for drinking water (MDEP, MDEP 2006a)
EPA PRG = Environmental Protection Agency Region 9 Preliminary Remediation Goal for tap water (U.S. EPA, U.S. EPA 2004c)
EPA RBC = Environmental Protection Agency Region 3 Risk Based Concentration for tap water (U.S. EPA, U.S. EPA 2006f)
RMEG (adult/child) = Reference Dose Media Evaluation Guides (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure). (ATSDR, ATSDR 2006a)

Table 3
Maximum concentrations of contaminants detected in wetland soil and sediment samples at the Woburn Landfill that exceeded comparison values (samples taken from 1997 - 2004)

Contaminant	Detection Frequency (# detected/total samples)	Date of sample	Descriptive location of sample	Maximum concentration (parts per million [ppm])	Soil Background (ppm)	Soil comparison value (ppm)
1,4-Dichlorobenzene	2 / 20	Nov-97	S2-3 (Southeast end of landfill)	3	---	Intermediate EMEG (child) = 4,000 Intermediate EMEG (adult) = 50,000 EPA PRG (residential soil) = 3 EPA RBC (residential soil) = 27
Benzo(a)anthracene	3 / 20	Jan-98	S2-8 (Southeast end of landfill)	2.9	0.005 - 0.02 (rural soil)† 0.169 - 59 (urban soil)†	EPA PRG (residential soil) = 0.62 EPA RBC (residential soil) = 0.22
Benzo(b)fluoranthene	7 / 20	Jan-98	S2-8 (Southeast end of landfill)	3.5	0.02 - 0.03 (rural soil)† 15 - 62 (urban soil)†	EPA PRG (residential soil) = 0.62 EPA RBC (residential soil) = 0.22
Benzo(a)pyrene	5 / 20	Jan-98	S2-8 (Southeast end of landfill)	3.3	0.002 - 1.3 (rural soil)† 0.165 - 0.22 (urban soil)†	CREG = 0.1 EPA PRG (residential soil) = 0.062 EPA RBC (residential soil) = 0.022
Arsenic	22 / 23	Mar-97	SED-2 (Southeast end of landfill)	330	7.4 (range: <0.1 - 73)*	Chronic EMEG; RMEG (child) = 20 Chronic EMEG; RMEG (adult) = 200 CREG = 0.5
Cadmium	14 / 20	Nov-97	S2-3 (Southeast end of landfill)	12	0.01 - 1‡	Chronic EMEG (child) = 10 Chronic EMEG (adult) = 100 RMEG (child) = 50 RMEG (adult) = 700
Chromium (total)	23 / 23	Nov-97	S2-3 (Southeast end of landfill)	1,400	52 (range: 1 - 1,000)*	Hexavalent Chromium: RMEG (child) = 200 Hexavalent Chromium: RMEG (adult) = 2,000
Lead	22 / 23	Nov-97	SED2-D-2 (Southeast end of landfill)	490	17 (range: <10 - 300)*	EPA PRG (residential soil) = 400
Polychlorinated Biphenyls (PCBs)	3 / 15	Jun-04	SED-102 (Southeast end of landfill)	1.4	---	CREG = 0.4 Intermediate EMEG (child) = 2 Intermediate EMEG (adult) = 20 Aroclor 1254: Chronic EMEG; RMEG (child) = 1 Aroclor 1254: Chronic RMEG; EMEG (adult) = 10

^ Samples S2-3, S2-8, and SED2-D-2 were taken at the approximate location of SED-2 (See Figure 5 and text for further discussion).

Data sources:
Maguire Group 2005. Supplemental Comprehensive Site Assessment and Post Closure Monitoring Plan. Woburn Sanitary Landfill, Woburn, Massachusetts. March 2005.

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 = Environmental Protection Agency Region 9 Preliminary Remediation Goal for residential soil (U.S. EPA 2004c)
EPA RBC = Environmental Protection Agency Region 3 Risk Based Concentration for tap water (U.S. EPA 2006f)
RMEG (adult/child) = Reference Dose Media Evaluation Guides (estimate of 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)

Sources of soil background values:
† Range of background Soil Concentrations. ATSDR Toxicological Profiles 2005 (on CD-ROM), Table 5-3. ATSDR 2005b.
‡ ATSDR 1999. Toxicological Profile for Cadmium. Atlanta: U.S. Department of Health and Human Services.
* Arithmetic mean (observed range) for the Eastern United States (east of 96th meridian). USGS. Shacklette HT, Boerngen JG. 1984. Element Concentrations in Soils and Other Surficial Materials of the conterminous United States. U.S. Geological Survey Professional Paper 1270. Washington: United States Government Printing Office, 1984.

Table 4
Maximum concentrations of contaminants detected in surface water samples at the Woburn Landfill that exceeded comparison values (samples taken from 1997 - 2004)

Contaminant	Detection Frequency (# detected/total samples)	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Chloroethane	3 / 15	Aug-97	SW-2 (Southeast end of landfill)	10	EPA RBC = 3.6 EPA PRG = 4.6
Bis(2-Ethylhexyl)phthalate	4 / 15	May-97	SW-3 (North end of landfill)	58	Chronic EMEG (child) = 600 Chronic EMEG (adult) = 2,000 RMEG (child) = 200 RMEG (adult) = 700 CREG = 3 U.S. EPA MCL & MDEP MMCL = 6
Pentachlorophenol	1 / 15	May-97	SW-3 (North end of landfill)	7	Chronic/Intermediate EMEG (child) = 10 Chronic/Intermediate EMEG (adult) = 40 CREG = 0.3 U.S. EPA MCL & MDEP MMCL = 1
Benzo(a)anthracene	4 / 15	May-97	SW-2 (Southeast end of landfill) & SW-3 (North end of landfill)	0.4	EPA RBC = 0.03 EPA PRG = 0.092
Benzo(b)fluoranthene	4 / 15	May-97	SW-3 (North end of landfill)	0.8	EPA RBC = 0.03 EPA PRG = 0.092
Benzo(a)pyrene	3 / 15	May-97	SW-3 (North end of landfill)	0.3	CREG = 0.005 U.S. EPA MCL & MDEP MMCL = 0.2 EPA RBC = 0.003 EPA PRG = 0.0092
Indeno(1,2,3-cd)pyrene	3 / 15	May-97 & Aug-97	SW-2 (Southeast end of landfill) & SW-3 (North end of landfill)	0.2	EPA RBC = 0.03 EPA PRG = 0.092
Antimony	1 / 5	Jun-04	SW-101 (Wetlands southwest of landfill property)	12 (total)	RMEG (child) = 4 RMEG (adult) = 10 LTHA, U.S. EPA MCL = 6 EPA RBC/PRG = 15

Table 4
Maximum concentrations of contaminants detected in surface water samples at the Woburn Landfill that exceeded comparison values (samples taken from 1997 - 2004)

Contaminant	Detection Frequency (# detected/total samples)	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Arsenic	2 / 7 (dissolved)	May-97 & Sep-04	SW-3 (North end of landfill) & SW-201 (Wetlands southwest of landfill property)	20 (dissolved)	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3
	4 / 6 (total)	Jun-04	SW-101 (Wetlands southwest of landfill property)	860 (total)	Chronic EMEG (adult), RMEG (adult) = 10 U.S. EPA MCL & MDEP MMCL = 10
Cadmium	1 / 5	Jun-04	SW-101 (Wetlands southwest of landfill property)	16 (total)	Chronic EMEG (child) = 2 Chronic EMEG (adult) = 7 U.S. EPA MCL & MDEP MMCL = 5
Chromium (total)	7 / 9	Jun-04	SW-101 (Wetlands southwest of landfill property)	1,620	Hexavalent Chromium: RMEG (child) = 30 Hexavalent Chromium: RMEG (adult) = 100 U.S. EPA MCL & MDEP MMCL = 100
Lead	1 / 8 (dissolved)	Sep-04	SW-203 (North end of landfill)	10 (dissolved)	U.S. EPA MCL & MDEP MMCL** = 15
	8 / 9 (total)	Jun-04	SW-103 (North end of landfill)	189 (total)	MCLG = 0
Manganese	9 / 10	May-97	SW-2 (Southeast end of landfill)	3,500 (dissolved)	RMEG (child) = 500 RMEG (adult) = 2,000

Data source:
Maguire Group 2005. Supplemental Comprehensive Site Assessment and Post Closure Monitoring Plan. Woburn Sanitary Landfill, Woburn, Massachusetts. March 2005.

Notes:
SW - Surface water
ppb - Parts per billion

Comparison values (source organization, reference):
CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR, ATSDR 2006a)
Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR, ATSDR 2006a)
Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, 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, ATSDR 2006a)
LTHA = Lifetime Health Advisory (U.S. EPA, ATSDR 2006a)
MDEP MMCL = Massachusetts Department of Environmental Protection Maxiumum Contaminant Level for drinking water (MDEP, MDEP 2006a)
U.S. EPA MCL = United States Environmental Protection Agency Maxiumum Contaminant Level for drinking water (U.S. EPA, ATSDR 2006a)
MCLG = Maximum Contaminant Level Goal for drinking water (U.S. EPA, ATSDR 2006a) (** = Action level for lead. Action must be taken if more than 10% of tap water samples exceed this value.)
EPA PRG = Environmental Protection Agency Region 9 Preliminary Remediation Goal for tap water (U.S. EPA, U.S. EPA 2004c)
EPA RBC = Environmental Protection Agency Region 3 Risk Based Concentration for tap water (U.S. EPA, U.S. EPA 2006f)
RMEG (adult/child) = Reference Dose Media Evaluation Guides (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure). (ATSDR, ATSDR 2006a)

Table 5
Summary of Possible Exposure Pathways for the Woburn Landfill
Woburn, Massachusetts

Environmental Medium	Exposure Pathway	Contaminant(s)	Point of Exposure	Route of Exposure	Receptor Population	Time Frame	Type of Pathway	Notes
Groundwater	Tap Water from potentially contaminated public or private wells	VOCs, SVOCs, PAHs	Off-site wells	Ingestion, Dermal contact, Inhalation while showering	Resident	Past, Present	Eliminated	No private wells within 500ft of landfill. Public groundwater supply wells in area are currently closed due to contamination (served Wilmington).
	Tap Water from potentially contaminated public or private wells	VOCs, SVOCs, PAHs	Off-site wells	Ingestion, Dermal contact, Inhalation while showering	Resident	Future	Potential	City of Woburn indicates that wells would never be permitted for construction in vicinity of landfill. (Maguire Group 2005).
	GW Contamination volatilizing to indoor air	VOCs, SVOCs, PAHs	On-Site buildings	Inhalation	Workers (Utility, Construction, Maintenance)	Past, Present, Future	Eliminated	No buildings located on landfill (Maguire Group 2005)
	GW Contamination	VOCs, SVOCs, PAHs	On-Site groundwater while digging for utilities/maintenance	Incidental ingestion, Dermal Contact	Worker	Future	Potential	Two Boston Edison utility easements traverse the landfill and depth to groundwater ranges from 2.75 to 20 feet below grade. (Maguire Group 2005)
	GW Contamination volatilizing to indoor air	VOCs, SVOCs, PAHs	Off-site residences	Inhalation	Resident	Past, Present, Future	Potential	No data; Johnson & Ettinger Model used to model potential indoor air concentrations from groundwater

Table 5
Summary of Possible Exposure Pathways for the Woburn Landfill
Woburn, Massachusetts

Environmental Medium	Exposure Pathway	Contaminant(s)	Point of Exposure	Route of Exposure	Receptor Population	Time Frame	Type of Pathway	Notes
Sediment/Soil	Sediment/Soil	VOCs, SVOCs, PAHs, Metals, PCBs	Wetlands	Incidental Ingestion, Dermal Contact	Resident	Past, Present, Future	Potential	
	Fugitive Dust from Sediment/Soil	VOCs, SVOCs, PAHs, Metals, PCBs	Wetlands	Inhalation	Resident	Past, Present, Future	Potential	Community concerned over past exposure to coal ash/dust used as daily cover. We have no data to evaluate that exposure. Present and future exposure is eliminated by the 3 foot vegetated cover on the landfill, as well as the minimal dust from wetland soil
Surface Water	Surface Water	VOCs, SVOCs, PAHs, Metals	Wetlands	Incidental Ingestion, Dermal Contact	Resident	Past, Present, Future	Potential	

TABLE 6
Cancer Incidence
Census Tract 3336, Woburn, Massachusetts
2000-2003

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	9	3.2	282	* 129 -- 535	6	2.3	259	94 -- 563	3	0.9	NC	NC -- NC
Brain & CNS	4	1.8	NC	NC -- NC	2	1.0	NC	NC -- NC	2	0.8	NC	NC -- NC
Kidney	5	3.4	148	48 -- 344	4	2.1	NC	NC -- NC	1	1.3	NC	NC -- NC
Leukemia	3	2.8	NC	NC -- NC	3	1.6	NC	NC -- NC	0	1.3	NC	NC -- NC
Lung & Bronchus	13	17.7	73	39 -- 125	7	9.3	75	30 -- 155	6	8.4	71	26 -- 155

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

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

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

Appendix A

Cancer Incidence Coding Definitions

Appendix A: International Classification Of Diseases For Oncology (Third Edition) (ICD-O-3) Codes Used for This Report[1]

Cancer Site / Type	Site code	Histology code[2]
Bladder, Urinary	C67.0-C67.9	all except 9590-9989
Brain & Other Central Nervous System (CNS)	C70.0-C72.9	all except 9590-9989
Kidney & Renal Pelvis[3]	C64.9, C65.9	all except 9590-9989
Leukemia	C00.0-C80.9	includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964
	AND	AND
	C42.0, C42.1, C42.4	includes 9823, 9827
Lung & Bronchus	C34.0-C34.9	all except 9590-9989

[1] Includes codes added to the *International Classification Of Diseases For Oncology, Third Edition* since its publication.

[2] Only invasive cancers (those with invasive behaviors) are included in this publication.

[3] Massachusetts hospital coding conventions may have assigned some cases to a "not otherwise specified" site category that is not included in this cancer type.

Appendix B

Risk Factor Information for Selected Cancer Types

Bladder Cancer

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

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

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

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

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

Silverman et al. 1996). Long term exposure to chlorinated by-products in drinking water has also been suggested to increase the risk of developing bladder cancer, particularly among men (Villanueva 2003).

References

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

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

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

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

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

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

Brain and Central Nervous System (CNS) Cancer

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

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

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

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

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

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

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

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

The association between the development of brain cancer and nitrites and other N-nitroso compounds, among the most potent of carcinogens, has been heavily researched. N-nitroso compounds have been found in tobacco smoke, cosmetics, automobile interiors, and cured meats. A study concluded that an increased risk of pediatric brain tumor may be associated with high levels of nitrite intake from maternal cured meat consumption during pregnancy (Pogoda and Preston-Martin 2001). However, the role of nitrites and cured meats in the

development of brain cancer remains controversial (Blot et al. 1999; Bunin 2000). Because most people have continuous, low level exposure to N-nitroso compounds throughout their lives, further studies, especially cohort studies, are needed to determine if this exposure leads to an increased risk of brain tumors (Preston-Martin 1996).

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

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

References

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

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

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

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

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

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

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

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

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

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

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

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

Kidney Cancer

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

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

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

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

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

genetic and hereditary risk factors may be important in the development of kidney cancer (ACS 2006; McLaughlin et al. 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS 2006; Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS 2006). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al. 1997; McLaughlin et al. 1996).

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

References

American Cancer Society. 2006. Detailed Guide: Kidney Cancer (Adult) – Renal Cell Carcinoma. Available at: http://www.cancer.org. Cited April 4, 2006.

American Cancer Society. 2006a. Detailed Guide: Wilms' Tumor. Available at: http://www.cancer.org . Cited April 4, 2006.

American Cancer Society. 2000. High Blood Pressure, Obesity Linked to Rise in Kidney Cancers. Available at: http://www.cancer.org/. Cited April 4, 2006.

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

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

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

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

Leukemia

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

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

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

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ

transplant patients) may be more likely to develop ALL (ACS 2006c). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al. 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS 2006c). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection. However, no specific virus has been identified as related to ALL (Linet and Cartwright 1996). Reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care do not support this hypothesis (Greaves MF 1997; Kinlen and Balkwill 2001; Rosenbaum et al. 2000).

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

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

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 2006e). Twice as many men as women are affected by this type of leukemia (Deisseroth et al. 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS 1999; Weinstein and Tarbell 1997). It is thought that individuals with a family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright 1996). Although viruses

have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al. 1997).

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

References

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

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

American Cancer Society. 2006c. Detailed Guide: Leukemia – Children's. Available at: http://www.cancer.org/ Cited April 18, 2006

American Cancer Society. 2006d. Detailed Guide: Leukemia –Acute Myeloid. Available at: http://www.cancer.org/ Cited April 18, 2006

American Cancer Society. 2006e. Detailed Guide: Leukemia –Chronic Lymphocytic. Available at: http://www.cancer.org/ Cited April 18, 2006

American Cancer Society. 2006f. Detailed Guide: Leukemia –ChronicMyeloid. Available at: http://www.cancer.org/ Cited April 18, 2006

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

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

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

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

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

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

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

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

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

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

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

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

Lung and Bronchus Cancer

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

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

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

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS 2005). Underground miners exposed to radon and

uranium are at an increased risk for developing lung cancer (Samet and Eradze 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni 1996; Pohlabeln et al. 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS 2005; Blot and Fraumeni 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al. 2001; Steenland and Sanderson 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni 1996).

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

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

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

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

References

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

American Cancer Society. 2005. Detailed Guide: Lung Cancer - Non-small Cell. Available at: http://www.cancer.org. Cited April 18, 2006.

American Cancer Society. 2002. Workplace Exposure to Secondhand Smoke Ups Women's Lung Cancer Risk. Available at: http://www.cancer.org. Cited May 9, 2006.

Bandera EV, Freudenheim JL, Marshall JR, et al. 1997. Diet and alcohol consumption and lung cancer risk in the New York State Cohort (United States). Cancer Causes Control 8:828-40.

Blot WJ, Fraumeni JF, Jr. 1996. Cancers of the lung and pleura. In: Cancer Epidemiology and Prevention. 2nd Ed, edited by Schottenfeld D, Fraumeni. JF. New York: Oxford University Press.

Brownson RC, Alavanja MCR, Caporaso N, et al. 1998. Epidemiology and prevention of lung cancer in nonsmokers. Epidemiol Rev 20(2):218-36.

Kreienbrock L, Kreuzer M, Gerken M, et al. 2001. Case-control study on lung cancer and residential radon in western Germany. Am J Epidemiol 153(1):42-52.

Lubin JH, Boice JD. 1997. Lung cancer risk from residential radon: meta-analysis of eight epidemiologic studies. J Natl Cancer Inst 87:49-57.

Tomasek L, Kunz E, Muller T, et al. 2001. Radon exposure and lung cancer risk – Czech cohort study on residential radon. Sci Total Environ 272(1-3):43-51.

Pohlabeln H, Boffetta P, Ahrens W, et al. 2000. Occupational risks for lung cancer among nonsmokers. Epidemiology 11:532-38.

Rice FL, Park R, Stayner L, et al. 2001. Crystalline silica exposure and lung cancer mortality in diatomaceous earth industry workers: a quantitative risk assessment. Occup Environ Med 58(1):38-45.

Samet JM, Eradze GR. 2000. Radon and lung cancer risk: taking stock at the millenium. Environ Health Perspect 108(Suppl 4):635-41.

Steenland K, Sanderson W. 2001. Lung cancer among industrial sand workers exposed to crystalline silica. Am J Epidemiol 153:695-703.

Warner KE, Mendez D, Courant PN. 1996. Toward a more realistic appraisal of the lung cancer risk from radon: the effects of residential mobility. Am J Public Health 86:1222-7.

Appendix C

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

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

General Terms

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

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

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

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

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

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

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

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

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

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

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

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

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

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

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

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

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

Body burden

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

CAP [see Community Assistance Panel.]

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

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

Carcinogen
A substance that causes cancer.

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

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

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

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

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

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

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

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to

confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

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

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

Completed exposure pathway [see exposure pathway].

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

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

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

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

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

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

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

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

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

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

DOD
United States Department of Defense.

DOE
United States Department of Energy.

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

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

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

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

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

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

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

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

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

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

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

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

Exposure registry
A system of ongoing follow-up of people who have had documented environmental exposures.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

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

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

Migration
Moving from one location to another.

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

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

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

Mutagen
A substance that causes mutations (genetic damage).

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

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

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

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

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

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

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

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

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

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

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

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

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

ppb
Parts per billion.

ppm
Parts per million.

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

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

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

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

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

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

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

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

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

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

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

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

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

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

Radionuclide
Any radioactive isotope (form) of any element.

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

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

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

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

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

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

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

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

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

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

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

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

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

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

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

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

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

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

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

Substance
A chemical.

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

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

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

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

Surveillance [see public health surveillance]

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

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

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

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

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health

effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

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

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

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

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

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

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

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

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

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Appendix D

Response to Public Comments

Appendix D

Response to Public Comments on "Evaluation of Environmental Concerns and Cancer Incidence, 2000–2003, Related to the Woburn Landfill in Woburn, Middlesex County, Massachusetts"

Listed below are comments received from the public regarding the Public Health Assessment (PHA) for the Woburn Landfill in Woburn, Massachusetts, that was released on November 26, 2007. The public comment period ended on January 10, 2008. MDPH and ATSDR received written comments from one resident of Woburn. The comments received are summarized with responses provided below.

Comment 1: "*The ATSDR report states that the Landfill did not contribute to these elevated [bladder, brain and CNS, and kidney cancer] rates, our question and comment is what risk factors contributed to this significantly higher rate? Should a further study be conducted to include the past 25 years? For example: one street with ten houses within the CT 3336 during the 1980's had 10 cases of cancer. The difficult question we are left with is, why?"*

Response 1: Please refer to pages 41 to 48 of the PHA for a detailed discussion of the risk factors associated with bladder, brain and CNS, and kidney cancer. A risk factor is anything that has been shown, as a result of scientific research, to increase your chance of getting a specific disease. Studies have shown that different cancer types have different disease causes, patterns of incidence, latency periods, and risk factors. For example, tobacco use has been linked to bladder, kidney, and lung and bronchus cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers.

Bladder cancer occurred more often than expected from 2000–2003 in Woburn CT 3336 (nine diagnoses observed versus 3.2 expected, SIR = 282, 95% CI = 129-535), which was a statistically significant elevation. According to the American Cancer Society, smoking is the greatest risk factor for bladder cancer. Smokers are more than twice as likely to

develop bladder cancer compared to nonsmokers (ACS 2006a). Of the nine individuals in Woburn CT 3336 who were diagnosed with bladder cancer during the years 2000–2003, eight had a known smoking history. Of those eight individuals, seven were current or former smokers (88%). Other risk factors for bladder cancer include occupational exposures, race, age, and gender. See pages 42 to 44 for a detailed discussion of age, gender, and occupation for the nine individuals diagnosed with bladder cancer. Based upon all of the information reviewed, it appears that these risk factors likely played a role in the development of bladder cancer among these individuals in Woburn CT 3336.

There were five diagnoses of kidney cancer compared to 3.4 expected (SIR = 148, 95% CI = 48-344) in CT 3336 during 2000–2003. This elevation was not statistically significant. Smoking is also a major risk factor for kidney cancer. Smoking increases the risk of developing renal cell cancer by 40% (ACS 2005). Of the five individuals diagnosed with kidney cancer in Woburn CT 3336 during 2000–2003, four had a known smoking history. Of those four, three individuals reported being current or former smokers. Other risk factors for kidney cancer include age, gender, obesity, occupational exposures, and genetic or hereditary factors. See pages 46 to 48 for a detailed discussion of age, gender, and occupation for the five individuals diagnosed with kidney cancer. Based upon all of the information reviewed, it appears that these risk factors likely played a role in the development of kidney cancer among these individuals in Woburn CT 3336.

The incidence of brain and CNS cancer was slightly elevated in Woburn CT 3336 during 2000–2003 (4 diagnoses observed compared to 1.8 expected). The most well-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 2006b). One of the four individuals diagnosed with brain or CNS cancer in Woburn CT 3336 had a previous cancer reported to the Massachusetts Cancer Registry (MCR); however, it is unknown whether radiation was used for treatment of that cancer.

Occupation, histology, age, and gender were reviewed for the four individuals diagnosed with brain or CNS cancer in Woburn CT 3336 (page 44 to 46). The patterns from this review were compared to known or established trends of brain and CNS cancer to assess whether any unexpected patterns exist. Although it is difficult to discern clear trends in small populations, the trends for age, gender, and histology seen in individuals diagnosed with brain or CNS cancer in Woburn CT 3336 were generally similar to trends in the state of Massachusetts as a whole. A review of occupation for the four individuals revealed one individual who might have worked at a job in which occupational exposures potentially related to the development of brain or CNS cancer may have been possible. However, information regarding specific job duties that could help to further define exposure potential for this individual was not available. Other risk factors for brain and CNS cancers include genetic or hereditary factors.

As stated above, kidney cancer risk factors include obesity and genetics, and brain and CNS cancer risk factors include hereditary factors. However, information about personal risk factors such as family history, genetics, diet, and other factors that may influence the development of cancer is not collected by the MCR or any other readily accessible source; therefore, it was not possible to evaluate these factors in this investigation.

Earlier years of cancer incidence data were reviewed in *Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999* (MDPH 2004). Bladder cancer occurred more often than expected during 1995–1999 among residents of Woburn CT 3336. Ten individuals were diagnosed with this cancer type compared to about five expected. This elevation was statistically significant (SIR = 212, 95% CI = 102-390). As stated above, cigarette smoking is the most well-established risk factor for bladder cancer. Among the nine individuals diagnosed with bladder cancer with known smoking history, eight reported being current or former smokers at the time of diagnosis and one individual was a non-smoker. As reported in 2004, review of this information suggested that smoking likely played an important role in the incidence of bladder cancer among residents in Woburn CT 3336. Review of this risk factor information and place of residence at diagnosis for individuals diagnosed with bladder cancer did not suggest any pattern or trend that was inconsistent with established incidence patterns.

Brain and CNS cancer occurred at approximately the expected rate (2 diagnoses observed versus 2.1 expected) from 1995–1999. Kidney cancer also occurred at approximately the expected rate (3 diagnoses observed versus 3.5 expected) during that time period.

To further address Comment 1 regarding cancer incidence near the Woburn Landfill in the 1980s and 1990s, an analysis of all types of cancer diagnosed with ¼ mile of the landfill was completed for the years 1982 to 1999. Cancer incidence data (i.e., reports of new cancer diagnoses) were obtained for this neighborhood from the MCR. The MCR is a population-based surveillance system that began collecting information on Massachusetts residents diagnosed with cancer in the state in 1982; therefore, this is the earliest year that cancer incidence data were available. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111s.111B). MDPH examined the cancer incidence data to determine if any street or area of the neighborhood had an unusual concentration of cancer. Thirty streets, in part or in whole, were examined including Breed Ave, Inglow Ave., Border Ave., Cook Ave., Main St., Jewel Dr., Woburn St., Industrial Way, Presidential Way, North Maple St., Naples Ave., Virginia Ave., North Washington Ave., Sacramento Ave., Delaware St., Kentucky Ave., Indiana Ave., Tennessee St., Mass Ave., Baldwin Ave., Dexter Ave., Tedesco Dr., Milan Ave., Ashburton Ave., Chester Ave., Knight Ave., Oakland St., Alger St., Merrimac St., and New Boston Street.

In general, our review found no atypical pattern of cancer in the neighborhood surrounding the Woburn Landfill. From 1982 to 1999, a total of 12 different types of cancer were diagnosed among 18 residents of this area, representing the occurrence of many different diseases. Cancers of the lung and bronchus, breast, and prostate were diagnosed among residents within ¼ mile of the landfill and are among the most common types of cancer diagnosed among men and women in Massachusetts. There were also nine other cancer types diagnosed among residents of this area of Woburn over the 18-year period reviewed, including cancers of the bladder; connective, subcutaneous, or other soft tissue; kidney; small intestine; oral cavity or pharynx; ovary; and uterus as well as leukemia and non-Hodgkin's lymphoma.

As mentioned above, different cancer types have different risk factors. Age is an important risk factor in many cancers. Different cancers occur with different frequencies among the various age groups, and most cancer types occur more frequently in older populations (i.e., age 50 and over). The average age at diagnosis among individuals diagnosed with any type of cancer within a ¼-mile radius of the Woburn Landfill was approximately 54 years of age. Review of the age and gender pattern among these individuals indicates that the incidence of cancers in this area is consistent with established prevalence patterns of disease in the general population.

Tobacco use is also an important risk factor in the development of several cancer types, including cancers of the lung and bronchus, kidney, oral cavity or pharynx, and bladder. Tobacco usage or smoking history, as reported to the MCR at the time of diagnosis, was reviewed for the nine individuals who were diagnosed with smoking-related cancers and lived within ¼-mile radius of the landfill. Smoking history was reported to the MCR for eight of the nine individuals. Of the eight individuals with a known smoking history, all were current or former tobacco users at the time of diagnosis.

In summary, the types of cancer that occurred varied and there was no specific geographic pattern of any one cancer type within ¼ mile of the landfill or on a specific street within this neighborhood that would suggest that environmental factors played a primary role in the development of these cancers. Also, the years of diagnosis for these individuals varied throughout the 18 years reviewed, indicating no apparent trend or pattern in the time of diagnosis.

Comment 2: *"As we all know, cancer does not appear overnight. We are also noting that some of these people who have moved from this area did develop cancers as well. Something is wrong here."*

Response 2: While the lack of access to information about in- and out-migration can be a data gap, it is important to note that the MCR is a high quality cancer registry that captures more than 95% of all diagnosed cancers among Massachusetts residents and

records their address at the time of diagnosis. Although clearly some individuals who may have lived much of their lives in Woburn moved away before their diagnoses, it is also true that some individuals lived elsewhere and then moved to Woburn, where they were diagnosed with cancer. Thus, we generally assume that there is some off-setting of both in- and out-migration for any given community. Further, if environmental factors were likely to have played a primary role in cancer development, it is most likely that such a pattern would have emerged even with some individuals no longer living in the area.

Comment 3: *"The Woburn Sanitary Landfill is unlined and is sitting on top of an aquifer. The unknown of what is buried in that Landfill is troubling including the fact that the Woburn Landfill is listed as Atomic Weapons Employer…"*

Response 3: According to Mass GIS, Massachusetts's Office of Geographic and Environmental Information, the southern portion of the Woburn Landfill is situated over a medium yield aquifer. However, the portion of the aquifer underlying the landfill does not contribute water to public drinking water wells in Woburn or Wilmington. The area of an aquifer which contributes water to a drinking water well under the most severe pumping and recharge conditions that can be realistically anticipated is known as a Zone II protection area (MDEP 1995). The nearest Massachusetts Department of Environmental Protection (MDEP) Zone II protection area lies approximately 0.25 miles to the northwest in Wilmington. The nearest public drinking water wells are located in Wilmington, approximately 1 mile northwest of the landfill. These public wells formerly served the Town of Wilmington and are currently inactive due to contamination from the Olin Chemical site in Wilmington (USEPA 2006). Since groundwater from the Woburn Landfill flows in a southeasterly direction, areas northwest of the landfill, including wells in Wilmington, would not be impacted by Woburn Landfill groundwater contamination. As discussed in the PHA, in Woburn, nearly 100% of the households obtain drinking water via the public water supply. Most of the public water supply (60%) comes from an underground aquifer in the Horn Pond area in southern Woburn, over 2 miles south of the

Woburn Landfill, while the remaining water comes from the Quabbin Reservoir in western Massachusetts.

Even under extreme drought conditions, it is unlikely that groundwater contaminants detected at the Woburn Landfill would reach the Zone II protection area for Woburn's community wells located over 2 miles south; therefore, exposures through municipal drinking water would not be expected.

A site with the name "Woburn Landfill" (also known as Winchester Engineering Vicinity Property and Woburn Dumpsite) is listed by the U.S. Department of Energy (DOE) as an Atomic Weapons Employer (AWE). This site is the former landfill off New Boston Street and is now the location of the Woburn Industrial Park. According to the DOE's list of facilities, fifty 55-gallon drums of low-grade uranium ore from the Atomic Energy Commission (AEC) Raw Materials Development Laboratory were disposed of between 1955 and 1960 at the former landfill (DOE 2007). The subject of this PHA, the Woburn Sanitary Landfill on Merrimac Street, began accepting waste for disposal around 1966 and is located ½ mile northwest of the former landfill. Prior to construction of the Woburn Industrial Park at the site of the former landfill, the uranium ore material was excavated and taken to the new Woburn Landfill, the subject of this PHA, on Merrimac Street.

A National Institute of Occupational Safety and Health (NIOSH) investigation of 94 AWE facilities, including the former Woburn Landfill off New Boston Street, did not indicate the existence of a current, unrecognized occupational or public health threat due to radioactive materials (NIOSH 2006). A Residual Radioactivity Evaluation conducted for the former Woburn Landfill as part of NIOSH's investigation determined that the material, which was buried in 1960 at the site of the Woburn Industrial Complex, had a radioactivity level similar to granite. Prior to construction of the industrial complex, this material was excavated and taken to the new Woburn Landfill, the subject of this PHA, on Merrimac Street. NIOSH reports that radiological surveys of both the old landfill site and the new landfill site did not indicate radioactivity greater than expected background levels. Background levels refer to typical amounts of substances that occur naturally in

an environment. Based on the described low level of radioactivity initially present and the radiological surveys conducted at each landfill site, NIOSH concluded that there is no indication or reason to suspect that residual contamination existed beyond 1960 (NIOSH 2006).

Comment 4: *"Since the capping of landfills is relatively new, it has not been proven yet as to what will occur in the future when the cap fails. Nothing lasts forever. I suppose that is an issue that the residents and Department of Public Health will have to deal with when it happens."*

Response 4: During the Woburn Landfill closure process a landfill gas collection and destruction system and a landfill cap were installed. The landfill cap consists of a 12-inch vegetative support layer comprised of 6 inches of loam and 6 inches of silty sandy soil; a 12-inch protective layer comprised of gravel borrow; a drainage layer comprised of a geocomposite material; a forty mil high density polyethylene (HDPE) cap layer; and a 6-inch gas collection layer comprised of sand (Maguire 2001). The active landfill gas collection system is designed to collect gases from beneath the capped landfill and to monitor surface methane emissions at the Woburn Landfill. Landfill gases are managed by an enclosed flare and active gas collection and destruction system in order to prevent the occurrence of nuisance odor conditions or public health and safety problems (Maguire 2002).

The Woburn Landfill is owned by the City of Woburn. The City is responsible for the monitoring and maintenance of the landfill and issues associated with the cap. The City's consulting engineers, the Maguire Group, developed a post-closure monitoring and maintenance plan as part of the site assessment and closure activities at the Woburn Landfill (Maguire 2005). The plan includes monitoring the active gas collection system, monitoring and repair of the vegetative cover on a semi-annual basis and after significant precipitation events, mowing of the landfill on an annual basis, monitoring of the security system on a bi-annual basis, and monitoring or repair of settlement and erosion on an "as-needed" basis. Residents who are concerned about settling and erosion or are seeking

further information regarding the on-going monitoring and maintenance of the Woburn Landfill should contact the Woburn Board of Health at (781) 897-5920.

Solid waste facilities in Massachusetts, including Woburn Landfill, are regulated in accordance with 310 CMR 19.000 as promulgated by the MDEP. The MDEP, not the MDPH, has the authority to administer the requirements, procedures, standards, and permits according to its Solid Waste Management regulations.

Comment 5: *"There are groundwater wells in place adjacent to the Woburn Sanitary Landfill that are situated on residential property on Cook Avenue and Border Avenue in South Wilmington that are on the North Woburn/Wilmington line. This neighborhood is approximately within a half mile of the Woburn Sanitary Landfill site.*

The WNA hopes that the MDPH is available to assist the Wilmington Board of Health as well in defining a testing and approval process for new and current private wells in the Town of Wilmington, MA that are in the vicinity of the Woburn Sanitary Landfill and the Olin Chemical site on Ames Street in Wilmington, MA."

Response 5: As stated in the Woburn Landfill PHA on page 13, groundwater beneath the landfill is flowing in a southeasterly direction (MDEP 2002, Maguire 2005). While Wilmington residential properties on Cook Avenue and Border Avenue are located to the north in close proximity to the landfill, private drinking water wells on these properties would not be expected to be influenced by groundwater from beneath the landfill because they are not located in the direction of groundwater flow. In addition, drinking water from the private bedrock wells in Wilmington nearest the landfill met all health-based drinking water standards when tested in 2002 (MDEP 2002).

Additionally, the Massachusetts Department of Public Health is currently working on public health assessment activities for the Olin Chemical Superfund Site, located immediately north of landfill in Wilmington. The Olin Chemical public health assessment activities will evaluate opportunities for environmental exposures of South Wilmington residents to contamination identified in environmental media, including

groundwater. Based on this information, MDPH will make recommendations to protect the public health of Wilmington residents as deemed appropriate. As always, the MDPH will work with local health officials and community residents in developing plans and approval processes deemed necessary to ensure public health protection.

References

American Cancer Society (ACS). 2005. Kidney Cancer. Available at: http://www.cancer.org.

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

American Cancer Society (ACS). 2006b. Brain / CNS Tumors in Adults. Available at: http://www.cancer.org.

Maguire Group, Inc. (Maguire). 2001. Supplemental Transmittal Application for Solid Waste Management Facility Modification BWP SW 22 – Landfill – Minor Modification - Woburn Sanitary Landfill, 202 Merrimac Street, Woburn, Massachusetts. Transmittal No. W015409. December 2001.

Maguire Group, Inc. (Maguire). 2002. Application for Solid Waste Management Facility Minor Modification for Woburn Sanitary Landfill, Woburn, Massachusetts. BWP SW 22. Transmittal No. W029105. May 2002.

Maguire Group, Inc. (Maguire). 2005. Supplemental Comprehensive Site Assessment and Post Closure Monitoring Plan. Woburn Sanitary Landfill, Woburn, Massachusetts. March 2005.

Massachusetts Department of Environmental Protection (MDEP). 1995. Guidance for Disposal Site Risk Characterization In Support of the Massachusetts Contingency Plan. Interim Final Policy #WSC/ORS-95-141. July 1995.

Massachusetts Department of Environmental Protection (MDEP). 2002. Report on the Closure and Capping of the City of Woburn's Landfill. March 19, 2002.

Massachusetts Department of Public Health (MDPH). 2004. Evaluation of Cancer Incidence in Woburn Census Tract 3336, MA: 1995–1999.

National Institute for Occupational Safety and Health (NIOSH), 2006. Report on Residual Radioactive and Beryllium Contamination at Atomic Employer Facilities and Beryllium Vendor Facilities. December 2006.

U.S. Environmental Protection Agency (USEPA). 2006. National Priorities List Olin Chemical. Accessed on December 5, 2006. Available from: http://www.epa.gov/superfund/sites/npl/nar1741.htm.

U.S. Department of Energy (DOE), 2007. Facility List Database. http://www.hss.energy.gov/healthsafety/fwsp/advocacy/faclist/findfacility.cfm. Office of Health, Safety and Security. Accessed February 4, 2008.