Public Health Assessment

Evaluation of Cancer Incidence, 1982-2000, and Environmental Concerns Related to the Bird Landfill in Walpole, Norfolk County, Massachusetts

BIRD, INC. LANDFILL
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I. SUMMARY

At the request of the Walpole Board of Health, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health (CEH), conducted an evaluation of possible environmental exposures and cancer incidence in relation to the Bird, Inc. Landfill, located on Merchant's Way, southwest of Norfolk Street in West Walpole, Massachusetts. This evaluation was initiated based on community concerns about cancer in the MacDonald Circle and Swan Pond Village neighborhoods, and the presence of volatile organic compounds (VOCs) detected in groundwater at the landfill. This project was conducted under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct public health assessments in Massachusetts.

The investigation reviews available environmental data for the Bird Landfill site and considers potential ways that people may come into contact with contaminants detected in groundwater, surface water, and wetland soils and sediment. The evaluation also looks at the pattern of cancer in Walpole, focusing on residential neighborhoods closest to the landfill. Seven cancer types were evaluated in this investigation: cancers of the bladder, kidney, liver, lung and bronchus, and leukemia, as well as Hodgkin's Disease and mesothelioma. Using data from the Massachusetts Cancer Registry, rates for these cancer types were calculated for the town of Walpole as a whole and for the three census tracts that comprise the town. Available information about risk factors, including environmental factors, related to the development of cancer was considered.

Future exposures to VOCs and metals detected in onsite groundwater are possible in the future if private wells are installed down-gradient of the landfill, and groundwater is consumed as drinking water. Except under extreme drought conditions, it is unlikely that groundwater contaminants detected at the Bird Landfill would reach the Zone II groundwater protection area for the Mine Brook municipal wells located 0.8 miles north of the site, and therefore exposures through public drinking water would not be expected. Based on the levels of VOCs detected in onsite groundwater, groundwater flow direction, and distance to nearby homes, it is unlikely that contaminants would present an exposure concern for indoor air down-gradient of the site. While intermittent exposures to onsite surface water, wetland soil, and sediment may be possible for individuals trespassing on site in the past, present, and future, contaminant concentrations

detected in these media are low, and it is unlikely that intermittent exposures would result in adverse health effects.

In general, most of the seven cancer types occurred near the rates expected for Walpole during the 19-year time period 1982-2000. In Walpole as a whole, bladder cancer (1988-1993) and kidney cancer (1982-2000 and 1988-1993) were statistically significantly elevated among females. This was due to statistically significant elevations in bladder cancer and kidney cancer among females from 1988-1993 in census tract (CT) 4113, where the Bird Landfill site is located. There was also a statistically significant elevation in Hodgkin's disease among females in Walpole as a whole during the middle time period 1988-1993. An evaluation of available risk factor information suggested that tobacco use likely played an important role in diagnoses of kidney and bladder cancer for some individuals, and none of the seven cancer types were elevated in a consistent pattern over time or in any one area of Walpole. Review of the geographic distribution of each of the cancer types in Walpole revealed no apparent spatial patterns at the neighborhood level. For example, despite the statistically significant elevations observed among females during some time periods, the geographic pattern of bladder cancer, Hodgkin's disease, and kidney cancer did not indicate a concentration or an atypical distribution of females diagnosed in Walpole as a whole or in CT 4113. Further, no unusual concentrations of individuals diagnosed with the seven cancer types were observed in the vicinity of the Bird Landfill site or in any other area of Walpole.

Based on criteria established by ATDSR, the Bird Landfill site would be classified as posing no apparent public health hazard in the past and present. Since private wells could be installed in the path of contaminated groundwater north-northeast of the site making drinking water exposures possible, the Bird Landfill poses an indeterminate public health hazard in the future. However, based on a review of available environmental data for the Bird Landfill, analysis of possible exposure pathways, and an evaluation of the pattern of cancer in the area surrounding the site, results do not suggest that a common factor (environmental or nonenvironmental) played a primary role in the incidence of cancer in the town of Walpole as a whole or the census tracts that divide the town during the 19-year time period 1982-2000.

II. INTRODUCTION

At the request of the Walpole Board of Health, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health (CEH), conducted an evaluation of possible environmental exposures and cancer in relation to the Bird, Inc. Landfill, located on Merchant's Way, southwest of Norfolk Street in West Walpole, Massachusetts. This evaluation was initiated based on community concerns about the pattern of cancer in two nearby neighborhoods, MacDonald Circle and Swan Pond Village, and the presence of volatile organic compounds (VOCs) identified in groundwater samples collected at the landfill. The Bird Landfill, which operated from 1968 to 1997, and these two neighborhoods are located approximately 2 miles southwest of Walpole's town center (see Figures 1 and 2). This project was conducted under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) for MDPH to conduct public health assessments in Massachusetts.

This investigation provides a review of potential exposure pathways to chemicals from the Bird Landfill, as well as a review of the pattern of cancer in Walpole that focuses on residential neighborhoods near the site. Seven cancer types were selected for this investigation: cancers of the bladder, kidney, liver, lung and bronchus, and leukemia, as well as Hodgkin's Disease and mesothelioma. These cancer types were selected based on their possible relationship to contaminants identified at the Bird Landfill, resident concern over suspected elevations in some cancer types, and/or statistically significant elevations observed town-wide in published Massachusetts Cancer Registry reports (MCR). To evaluate concerns about potential environmental exposures from the Bird Landfill site, MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) to obtain and review available environmental data.

Cancer rates were calculated for the town of Walpole during the years 1982–2000, the time period for which the most recent and complete cancer incidence data were available from the MCR at the time of analysis. The town of Walpole is divided into three smaller geographic areas or census tracts (CTs): CT 4111, CT 4112, and CT 4113. A census tract is a smaller geographic subdivision of a city or town that is designated by the United States Census Bureau. Because

age-group and gender-specific population information is necessary to calculate incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated. The Bird Landfill, MacDonald Circle, and Swan Pond Village are all located in CT 4113. The town of Walpole is located 19 miles southwest of Boston and is bordered by the towns of Dover and Westwood to the north, Norwood and Sharon to the east, Foxboro to the south, and Norfolk and Medfield to the west. Walpole is primarily a suburban community and comprises an area of 20.5 square miles with 1,100 residents per square mile (U.S. DOC 2000). The 2000 United States Census reports a total of 22,824 residents in the town of Walpole (U.S. DOC 2000). Census tract locations and boundaries in Walpole are shown in Figure 1.

The results of the descriptive cancer analysis can be useful in identifying cancer patterns or trends in a geographic context, to determine if a common etiology (i.e., cause associated with the development) is possible, and may serve to identify areas where further public health investigations or actions may be warranted. Descriptive analyses may also indicate that an excess of known risk factors associated with a disease, such as environmental exposures, exists in a certain geographic area. This descriptive analysis of cancer incidence data cannot be used to establish a causal link between a particular risk factor (either environmental or nonenvironmental) and the development of cancer. In addition, this analysis cannot determine the cause of any one individual's cancer diagnosis. The purpose of this evaluation is to report the findings on the patterns of cancer in Walpole, with a particular focus on the neighborhoods in the vicinity of the Bird Landfill site, and discuss them in the context of the available environmental information to determine whether recommendations for further public health action are needed.

III. OBJECTIVES

The specific objectives of this investigation were as follows:

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

- To evaluate the incidence rates of seven cancer types in Walpole as a whole and in areas near the Bird Landfill site to determine if cancer is occurring more or less often than expected;

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

- To review available descriptive information from the Massachusetts Cancer Registry (MCR) for individuals diagnosed with cancer in Walpole 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 Bird 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 to determine whether further investigation or public health action is warranted.

IV. BACKGROUND AND COMMUNITY ENVIRONMENTAL CONCERNS

The Walpole Board of Health and community residents have expressed concerns about elevated levels of VOCs historically detected in groundwater at the Bird Landfill site and the incidence of cancer in two neighborhood areas, MacDonald Circle and Swan Pond Village, located east and northeast of the landfill (Figure 2). 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 Bird Landfill. In addition, information regarding other potential environmental sources located in the area and listed with MDEP as a location of a hazardous release or spill was reviewed (MDEP 2005).

The public health assessment titled "*Evaluation of Cancer Incidence, 1982–2000, and Environmental Concerns Related to the Bird Landfill in Walpole, Norfolk County,*

Massachusetts" was released on September 13, 2006, for a 30-day public comment period. No public comments were received by the MDPH during the public comment period.

A. Bird Landfill

The Bird Landfill is located on Merchant's Way, southwest of Norfolk Street in Walpole, Norfolk County, Massachusetts, and the property consists of approximately 85 acres of land (GZA 1997). To the northwest and northeast, the landfill is bordered by low-lying wetlands and some commercial developments. There are residential properties located immediately southwest and east of the landfill (Bucket Mill Lane and MacDonald Circle neighborhoods, respectively). Refer to Figure 2 for a map of the landfill property.

As shown in Figure 2, the landfill consists of two areas, Area I to the west and Area II to the east. Area I began operation in 1968 and was closed in 1980 (GZA 1997). Area II began receiving process waste from Bird's Norwood Roofing Plant as early as 1980, operated briefly as a recycling facility between 1992 and 1993, and subsequently operated as a landfill again through 1997. Neither Area I nor Area II is lined (John Morey, MDEP, personal communication, 2005). Area I consists of waste from Bird Manufacturing operations (mainly stone dust), wastes from Bird's Norwood Roofing Plant (rolled roofing, shingles, solid drums of asphalt, stone granules, paper, wood, and scrap metal), and Bird's Walpole Paper Mill (beater waste). Area II waste consists of scrap asphalt roofing shingles, cardboard rolls, fiberglass mat, stone granules, solid asphalt, defective wooden pallets, and paper wrapping materials. While in operation, the landfill was covered daily with at least 6 inches of stone dust from Bird, Inc.'s asphalt shingle roofing plant or silty sand/silt tailings from S. M. Lorusso's West Sand Plant, both also located in Walpole (GZA 1997). No asbestos wastes, infectious wastes, sludge, or special hazardous, liquid or banned wastes were reportedly accepted at the landfill (GZA 1997). Area I was loamed and seeded in 1980 at the time of closure and Area II was capped in 2000 (GZA 1998, John Morey, MDEP, personal communication, 2005). Post-closure groundwater monitoring continues at the landfill and sampling results are reported to the MDEP.

The Bird Landfill is located outside the Zone II groundwater protection areas for two Walpole municipal well fields that draw from the Head of the Neponset Aquifer: the Mine Brook wells to the northeast and the Washington Street wells to the east. A Zone II groundwater protection area

is defined as the area of an aquifer which contributes water to a well under the most severe pumping and recharge conditions that can be reasonably anticipated. Because the landfill is located outside the Zone II protection area, it is unlikely that groundwater from Area II of the landfill would reach municipal wells. However, based on regional groundwater flow direction, GZA GeoEnvironmental, Inc. (GZA) [1998] identified the Mine Brook No. 2 well as a municipal well that would potentially be impacted under a worst case scenario. Groundwater beneath Area I is thought to flow toward wetlands located to the north, south, and west of the landfill (GZA 1998). The closest private wells are located approximately 3,000 feet up-gradient or cross-gradient from the landfill. Two private wells were identified northeast of the landfill on Spring Street; however, GZA has reported that these private wells are located outside the estimated extent of potentially impacted groundwater (GZA 1998), and that one of the private wells is not in use.

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

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

The 21E sites are characterized by one or more releases of oil or other hazardous material. Releases can result from a variety of sources, including trucks and other vehicles, underground storage tanks, and aboveground storage drums. Releases vary widely with respect to materials involved, the relative amount of materials released, and the geographic extent of contamination. Information on hazardous material and oil releases is available from 1977 to the present, from the MDEP Bureau of Waste Site Cleanup (MDEP 2005). However, records prior to 1984 are known to contain significant data gaps.

Hazardous material and oil releases are *potential* sources of exposure to contamination. It is not possible to determine whether individuals residing in the evaluation area were actually exposed to contaminants without more 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.

In addition to the evaluation of environmental data associated with the Bird Landfill, MDPH reviewed the most recent information regarding oil or other hazardous material releases for the town of Walpole and mapped the approximate location of release sites with sufficient address information using a geographic information system (Figure 3) (ESRI 2004). A total of 222 releases were reported in the town of Walpole from 1983 to 2005. The majority of these releases were mapped to an address in town; however, approximately 18% of the releases (n = 40) could not be mapped due to insufficient address information (most unmapped releases were located along the major roads). There were no releases reported at the Bird Landfill. The full list of releases recorded as "21E sites" in Walpole is shown in Table 1.

V. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with the Bird Landfill site, MDPH reviewed information from several reports on file with MDEP. Environmental sampling data were available for groundwater, surface water, sediment and wetland soils located onsite, with groundwater being the main focus of environmental investigations conducted at the landfill. 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 ATSDR (ATSDR 2005b, 2005c). If an ATSDR comparison value was not available for a specific chemical, the maximum detected concentration of that chemical was compared to Risk-Based Concentrations (RBCs) developed by the United States EPA Region III (U.S. EPA 2004) or the applicable groundwater and soil standards developed by MDEP (2004), in that order. For compounds detected in groundwater, maximum concentrations were also compared with state or federal drinking water standards.

The ATSDR comparison values are specific concentrations of a chemical for air, soil, or water that are used by health assessors to identify environmental contaminants that require further evaluation. These comparison values are developed based on health guidelines and assumed exposure situations that represent conservative estimates of human exposure. Chemical concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, chemical concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a 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 that are unique to the specific situation under investigation need to be considered to determine if an adverse health effect from this chemical could occur.

A. Groundwater

Environmental investigations associated with the closure of the Bird Landfill identified groundwater as the primary environmental media through which site contaminants could migrate offsite (GZA 1998). Evaluation of site hydrology indicated that the majority of groundwater flow is through glacial outwash sands (GZA 1997). Groundwater at the site flows generally in a northerly direction. Local mounding in Area I is thought to cause groundwater in this area to flow toward wetlands to the north, east, and west (GZA 1997). Groundwater flow in the glacial outwash aquifer near Area II appears to be in a north-northeast direction away from MacDonald Circle homes and in the direction of the Swan Pond Village neighborhood. Groundwater in the deeper bedrock is to the northeast turning northward.

The locations of groundwater monitoring wells are depicted in Figure 4. Groundwater from both the bedrock and overburden strata was sampled (GZA 1997). In 1984, seven groundwater monitoring wells (designated LF-1 through LF-7) were installed in the overburden of Area II during an environmental site assessment conducted for refinancing purposes (GZA 1997). Four of these wells (LF-2, 3, 5 and 6) were replaced in 1988. Shallow and deep wells were installed at

two locations (LF-3 and LF-6) in 1994 to further investigate down-gradient impacts of Area II. In addition, a shallow monitoring well (LF-UP) was installed in 1994 at a location designated to be up-gradient of Area II and unaffected by Area I. In 1996, five additional monitoring wells (CSA-1, CSA-2S and CSA-2D, CSA-7S and CSA-7D) were installed down-gradient of Area II, as well as five well borings (CSA-3, CSA-4D, CSA-5, CSA-6S and CSA-6D) around the perimeter of Area I.

Most groundwater samples collected from monitoring wells in Area II were analyzed for the presence of VOCs, chloride, nitrate, nitrite, and sulfate two times per year (GZA 1997). Groundwater from this area was also evaluated every 2 years for metals, pesticides/herbicides, and oil and grease. Metals were analyzed on a yearly basis beginning in 1992. Monitoring wells installed around Area I in 1996 were analyzed for VOCs, nitrate, nitrite, sulfate, chloride, cyanide, and metals. In addition to groundwater sampling at the landfill proper, three monitoring wells (B-1S, B-1D, and B-2) were installed at the western edge of the site in 1997 at locations designed to be representative of background conditions. These 'background' wells were sampled for metals only (GZA 1998). All groundwater monitoring wells were located on the site and no off-site groundwater contamination data were available.

Table 2 summarizes the maximum concentrations of contaminants detected in Area I and Area II groundwater samples that exceeded comparison values. Because ATSDR comparison values do not exist for groundwater, drinking water comparison values were used as screening values. The following compounds were detected in groundwater at the Bird Landfill at levels above comparison values for drinking water: arsenic, barium, benzene, cadmium, chloroethane, chromium, copper, cis-1,2-dichloroethene, iron, lead, manganese, silver, tetrachloroethene, trichloroethene, and vinyl chloride.

With some exceptions, VOCs were detected above comparison values in shallow and deep groundwater samples collected from three monitoring wells down-gradient of Area II. Specifically, the maximum concentration of tetrachloroethylene (PCE) (41 parts per billion [ppb]) was detected above the EPA Risk-Based Concentration (RBC) (1.2 ppb) and the MDEP Massachusetts Maximum Contaminant Level (MMCL) (5 ppb) in LF-6D, a deep monitoring well located close to the property boundary and down-gradient of Area II (Figure 4). PCE was

detected above comparison values each of the 22 times it was detected at this monitoring well during the time period 1994 to 2005. PCE also exceeded comparison values in shallow monitoring wells LF-6S and LF-3S and deep monitoring well CSA-7D, all located down-gradient of Area II.

Like PCE, the maximum concentration of trichloroethylene (TCE) (34 ppb) was detected above the EPA RBC (1.6) and the MDEP MCL (5 ppb) at LF-6D. TCE exceeded drinking water comparison values in shallow monitoring wells LF-3S and LF-6S and in deep monitoring well CSA-7D, all located down-gradient of Area II.

The maximum concentration of vinyl chloride (24 ppb) exceeded the ATSDR Cancer Risk Evaluation Guide (CREG) for drinking water (0.03 ppb) and the MDEP MMCL (2 ppb) in shallow monitoring well LF-3S, which is close to the down-gradient property limit of Area II (Figure 4). Vinyl chloride was also detected above the CREG and MMCL in shallow and deep monitoring wells LF-6S/6D and above the CREG but below the MMCL in shallow and deep monitoring wells CSA-7S/7D.

Cis-1,2-dichloroethene exceeded the EPA RBC (61 ppb) and the MDEP MCL (70 ppb) for drinking water two times at separate locations. The maximum concentration of this contaminant (110 ppb) was detected at shallow monitoring well LF-3S. Cis-1,2-dichloroethene was also detected slightly above comparison values in deep monitoring well CSA-7D.

The maximum concentration of chloroethane (250 ppb) was detected above the EPA RBC (3.6 ppb) at monitoring well LF-3A, which is close to the down-gradient property limit of Area II (Figure 4). Choroethane also exceeded the EPA RBC at six monitoring wells located both up-gradient and down-gradient from Area II and at two monitoring wells near Area I.

While the maximum concentrations of most VOCs were detected in Area II groundwater, the maximum concentration of benzene (21 ppb) exceeded the ATSDR CREG for drinking water (0.6 ppb) and the MMCL (5 ppb) at monitoring well CSA-3 in the west end of Area I (Figure 4). Benzene was also detected above the CREG and MMCL in two monitoring wells near Area I and slightly above the CREG (but below the MMCL) at monitoring well LF-3S, which is near the down-gradient property limit of Area II.

A variety of metals have been detected in groundwater at the Bird Landfill since site investigations began in the mid-1980s. According to sampling data, metals do not appear to be concentrated in any one particular area of the site. Specifically, the maximum concentration of arsenic (189 ppb) exceeded the ATSDR CREG (0.02 ppb) and the U.S. EPA MCL for drinking water (10 ppb) at monitoring well LF-UP, a shallow monitoring well located up-gradient of Area II, but not down-gradient of Area I (Figure 4). Arsenic was also detected above the CREG and the MCL in some monitoring wells located at or down-gradient from Areas I and II. With the exception of CSA-2D, CSA-6S, and CSA-7D, iron was detected above the EPA RBC for drinking water (11,000 ppb) on at least one occasion at all monitoring wells in Areas I and II. The maximum concentration of iron (898,000 ppb) was detected at monitoring well LF-3D, a deep well located in the down-gradient portion of Area II. The maximum concentration of lead (230 ppb) exceeded the EPA Action Level (15 ppb) at monitoring well LF-UP. Lead was also detected slightly above the Action Level in one monitoring well near Area I and at six monitoring wells in both up-gradient and down-gradient locations near Area II. With a few exceptions, manganese was detected above the Reference Dose Media Evaluation Guide (RMEG) for adults (2,000 ppb) on at least one occasion at all groundwater sampling locations in Areas I and II. The maximum concentration of manganese (26,000 ppb) was detected in monitoring well LF-1, located in the northwest portion of Area II.

Several other metals were detected slightly above comparison values at various locations throughout the site. Specifically, barium was detected slightly above the RMEG (700 ppb) for childhood exposure to drinking water, but less than the adult RMEG of 2,000 ppb at two locations: north of Area I at monitoring well CSA-5 (741 ppb) and monitoring well LF-UP (986 ppb) south of Area II (Figure 4). Cadmium was detected above the MCL (5 ppb) on five occasions at monitoring wells located in Areas I and II. The maximum concentration of cadmium (16.4 ppb) occurred in monitoring well CSA-2D, which is located down-gradient from Area II. Total chromium and hexavalent chromium were detected above the MCL for hexavalent chromium at several locations in Areas I and II (comparison values for total chromium were not available). The maximum concentration of total chromium (506 ppb) was detected in monitoring well LF-UP, and the maximum concentration of hexavalent chromium (580 ppb) was detected in down-gradient shallow well LF-7S. The maximum concentration of copper (818 ppb) was detected above the ATSDR Intermediate Environmental Media Evaluation

Guide (EMEG) for adult exposure (400 ppb) in monitoring well CSA-6S. Silver was detected above the RMEG value for adults (200 ppb) on two occasions in Area II monitoring wells LF-1 and LF-6D. The maximum concentration of silver (250 ppb) was detected in LF-1.

B. Surface Water

The Bird Landfill is located at the western edge of the Neponset River Drainage Basin (GZA 1997). On the western portion of the property, surface water runoff flows south into Cedar Swamp, then to Cedar Swamp Brook approximately 8,000 feet south, and finally to the Neponset River, approximately 6,000 feet southwest of the site (GZA 1997). Runoff from the northeastern portion of the site is mostly contained by grading at the landfill's perimeter. The remainder of runoff from Area II flows northeast into a small stream, where it flows northward to another segment of the Neponset River approximately 8,000 feet to the northeast.

Surface water samples were collected from five onsite wetland areas adjacent to the landfill in 1996 and analyzed for VOCs and metals (GZA 1997). One sample (SW-1) was collected from an iron-stained groundwater seep at the western edge of Area I, another (SW-2) was collected from a channel west of Area I, and a third (SW-3) was taken from standing water in wetlands south of Area I (Figure 4). SW-4 was collected from a stream south of Area II, and SW-5 was collected from a pond located between the landfill and MacDonald Circle just east of Area II. Repeat sampling of surface water locations near Area I occurred through 1998, and sampling of surface water locations near Area II were repeated through 2005 (GZA 2005). In 1997, three additional 'background' surface water samples were collected from locations that were selected for being outside the area where surface water or groundwater from the landfill could migrate. Specifically, background surface water samples were collected at locations northwest, west, and southeast of the landfill in 1997 and 1998 and analyzed for metals (GZA 1998, 2002).

Table 3 summarizes the maximum concentrations of contaminants detected in surface water samples that exceeded comparison values. Because ATSDR comparison values do not exist for surface water, drinking water comparison values were used as screening values. This is a conservative evaluation because guidelines for chemicals in drinking water assume adults ingest 2 liters of water per day. Exposures to chemicals present in surface water not used for drinking water purposes would be expected to be less than exposures to chemicals in drinking water.

The maximum concentration of benzene (0.97 ppb) was detected in one surface water sample (SW-1) above the ATSDR CREG comparison value for drinking water (0.6 ppb), but was below the MDEP MCL for drinking water (5 ppb) (see Table 3). SW-1 was collected from the iron-stained groundwater seep at the west end of Area I. No other VOCs were detected above comparison values in surface water.

The following metals were detected in surface water at concentrations above or slightly above comparison values for drinking water: arsenic, barium, cadmium, iron, lead, manganese, and silver (Table 3). Arsenic was detected in SW-5 at 6 ppb, which is above the CREG, but below the U.S. EPA MCL of 10 ppb. Arsenic was not detected at the other four sampling locations. Barium was detected at all five sampling locations. The maximum concentration of barium (1,080 ppb), which occurred at SW-1, was above the drinking water RMEG for children (700 ppb), but below the adult RMEG (2,000 ppb). Cadmium was detected in SW-1, SW-4, and SW-5. The maximum concentration of cadmium (8.6 ppb) was detected in SW-4 at a level slightly above the adult EMEG (7 ppb) and the MCL (5 ppb). The maximum concentration of iron (52,000 ppb) was detected above the EPA RBC for drinking water (11,000 ppb) at SW-1. Iron was detected below the EPA RBC at the four other sampling locations. In 1996, lead was detected in surface water (85 ppb) above the EPA Action Level for drinking water (15 ppb) in SW-4, collected from the stream south of Area II. Lead was detected below the EPA Action Level for drinking water in all other surface water samples and was detected in just one of 10 subsequent samples collected at SW-4 since 1996. The maximum concentration of manganese (1,530 ppb) was detected above the child RMEG (500 ppb), but below the adult RMEG (2,000 ppb) for drinking water at SW-1. Silver was detected in one of five surface water samples at 85 ppb. This concentration was above the RMEG for children (50 ppb), but below the RMEG for adult exposure (200 ppb). All metals were detected within the range of background concentrations observed for surface waters (ATSDR 2003).

C. Sediment/Wetland Soils

In 1996, sediment and wetland soil samples were collected from approximately the same locations as the surface water samples described above and analyzed for metals. Refer to Figure 4 for the locations of sediment samples S-1 through S-5. In 1997 and 1998, wetland soil and

sediment samples were collected from five new locations selected to represent background conditions (GZA 1997, 1998). Based on the available environmental data, no other soil samples were collected at the Bird Landfill site.

All of the metals detected in sediment and wetland soils at the Bird Landfill were within the range of background concentrations observed for metals in eastern U.S. soils (Shacklette et al 1984).

VI. EXPOSURE PATHWAY ANALYSIS

An evaluation of potential pathways of exposure was conducted to determine whether contamination identified at the Bird Landfill site could be impacting residents of Walpole in the past, present, or future. Exposure to a chemical must first occur before any potential adverse health effects can result. Five conditions must be present for exposure to occur. First, there must be a source of that chemical. Second, an environmental medium must be contaminated by either the source or by chemicals transported away from the source. Third, there must be a location where a person can potentially contact the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, and dermal absorption. Finally, the chemical must actually reach the target organ susceptible to the toxic effects caused by that particular substance at a sufficient dose and for a sufficient exposure time for an adverse health effect to occur (ATSDR 2005a).

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

To evaluate the potential for health effects, ATSDR Minimal Risk Levels (MRLs) were compared to exposure estimates for the contaminants of concern at the Bird Landfill site. 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 United States EPA cancer slope factors provided by ATSDR to evaluate potential cancer risk. Refer to Table 4 for a summary of exposure pathways discussed in this section.

A. Exposure to Groundwater

In general, groundwater at the Bird Landfill site flows in a northerly direction with shallow groundwater near Area II flowing to the north-northeast. Local mounding present in Area I causes groundwater to flow toward wetlands located to the north, east, and west (GZA 1997). Sampling conducted in monitoring wells located at the down-gradient portion of Area II detected VOCs and metals that exceeded health-based comparison values for drinking water. The groundwater wells sampled at the Bird Landfill site were installed for monitoring purposes only, and no one ingests water from these wells.

As noted earlier, the Bird Landfill is located outside the limits of a Zone II groundwater protection area for municipal drinking water well fields, which is the area of an aquifer which contributes water to a well under the most severe pumping and recharge conditions that can be reasonably anticipated. Groundwater beneath the landfill has never been a source of municipal drinking water (GZA 1997, 1998). If contaminated groundwater from the Bird Landfill were to migrate about 0.8 miles north to the Zone II groundwater protection area for the Mine Brook wells, it is possible that under extreme drought conditions (i.e., 180 days with no precipitation to recharge aquifers) contaminated groundwater could reach and affect water quality at the well field. However, public water supplies are tested and treated on a routine basis in accordance with state and federal laws. Based on this, exposure through municipal drinking water to groundwater contamination identified at the Bird Landfill is an unlikely exposure pathway (Table 4).

Although there are two private wells located northeast of the Bird Landfill on Spring Street, one is reportedly not in use and the other is used with a residential chemical filter (GZA 1997, 1998). It is not known whether groundwater from the private well with a filter has been impacted by contaminated groundwater from the Bird Landfill. However, based on site investigations

conducted for the Bird Landfill by GZA, Inc., GZA determined this well is located outside the extent of groundwater potentially impacted by the landfill (GZA 1998). Therefore, past and current exposure to contaminated drinking water from these down-gradient private wells is not expected.

There is no moratorium in place to restrict the installation of new private wells in the town of Walpole (R. Chapell, Walpole Board of Health, personal communication, 2006); therefore, future exposure to contaminants identified at the Bird Landfill is possible if new private drinking water wells are installed in the path of contaminated groundwater. If nearby residents were to ingest contaminated groundwater in the future at concentrations detected in onsite monitoring wells, noncancer and cancer health impacts are possible due to exposure to some metals and VOCs, in particular, arsenic, manganese, and vinyl chloride.

B. Exposure to Indoor Air

While current information indicates there are no private wells in the path of contaminated groundwater, exposure to VOCs detected in groundwater at the landfill could occur through indoor air in homes with basements if VOCs are present in off-site groundwater at sufficient concentrations and if groundwater is shallow. Based on groundwater elevations in the vicinity of MacDonald Circle east of the site, GZA determined that the shallow groundwater flow is away from residences and toward an adjacent stream (GZA 1998). The direction of shallow groundwater at the Bird Landfill site is determined to be to the north-northeast (GZA 1998), in the general direction of some industrial buildings and Swan Pond Village homes. There were no off-site groundwater sampling data available, so it is unknown whether the concentrations of VOCs such as vinyl chloride detected in onsite groundwater monitoring wells exist off-site. However, to evaluate a possible vapor intrusion exposure scenario, MDPH received assistance from ATSDR to use a model incorporating site-specific information on groundwater, soil, and housing for the area (ATSDR, 2005d).

To evaluate a very conservative scenario, the Johnson-Ettinger mathematical model was first run using the maximum concentration of vinyl chloride detected in groundwater at the landfill (24 ppb) together with the shallowest groundwater depth (7 feet). Based on these parameters, (i.e., if a house with a basement were located on top of the groundwater monitoring well with the

highest vinyl chloride concentration and the shallowest groundwater), the model predicted with 95% certainty that the indoor air concentration would be at or below 3.75 micrograms per cubic meter ($\mu g/m^3$), or a low incremental cancer risk from vapor intrusion to indoor air of 1.4×10^{-5}.[1] To evaluate a slightly less conservative vapor intrusion exposure scenario, the model was also run using the average detected vinyl chloride groundwater concentration at the Bird Landfill (3.2 ppb), with an average depth to groundwater for onsite monitoring wells (20 feet). Under these conditions, the model predicted with 95% certainty that the indoor air concentration would be at or below 0.119 $\mu g/m^3$, and a low cancer risk from vapor intrusion of vinyl chloride to indoor air of 4.5×10^{-7}.[2]

Some additional considerations suggest model estimates would be even lower. First, it is important to consider that the closest down-gradient building is located approximately 850 feet away, and the closest Swan Pond Village homes are located approximately 1500 feet away from the landfill. Since the maximum contaminant concentrations are typically located closest to the source, actual down-gradient concentrations of vinyl chloride 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, if groundwater flow is in the direction of the adjacent stream, it is possible that vapors could have escaped through the exposed stream and down-gradient groundwater concentrations would be even lower (ATSDR 2005d). Further, while the maximum vinyl chloride concentration in onsite groundwater was 24 ppb, vinyl chloride was detected in just 43 out of 127 groundwater samples collected at the landfill, and all other detected concentrations of vinyl chloride were 10 to 100 times lower. Thus, while down-gradient vinyl chloride groundwater concentrations are unknown, based on the levels of vinyl

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[1] Cancer Effects Exposure Dose $= \frac{(3.75\ \mu g/m^3)\ )\ (365\ days/yr)\ (30\ yrs)}{(70\ yrs)\ (365\ days/yr)} = 1.61\ \mu g/m^3$

Cancer Risk = $1.61\ \mu g/m^3 \times 8.8 \times 10^{-6}\ (\mu g/m^3)^{-1} = 1.41 \times 10^{-5}$

[2] Cancer Effects Exposure Dose $= \frac{(0.119\ \mu g/m^3)\ )\ (365\ days/yr)\ (30\ yrs)}{(70\ yrs)\ (365\ days/yr)} = 0.051\ \mu g/m^3$

Cancer Risk = $0.051\ \mu g/m^3 \times 8.8 \times 10^{-6}\ (\mu g/m^3)^{-1} = 4.5 \times 10^{-7}$

chloride detected in onsite groundwater, the indoor air concentrations predicted by the Johnson-Ettinger model using very conservative assumptions, and the distance of the down-gradient homes from the site, it appears unlikely that vinyl chloride detected in groundwater at the Bird Landfill would present an exposure concern for indoor air down-gradient of the site.

C. Exposure to Surface Water

Surface water samples were collected from an offsite stream and pond located immediately east of Area II and from wetland areas located west and south of Area I. Incidental ingestion and dermal contact with contaminants detected in surface water could be possible for children or adults who may have accessed surface waterbodies around the landfill for wading or playing in the past, present, and future. However, the majority of surface water contaminants were detected below or within the range of drinking water comparison values; therefore, potential exposures to surface water would not be expected to result in health effects (refer to Table 3). While arsenic (6 ppb) was detected above the ATSDR CREG for cancer health effects (0.02 ppb) in surface water from a pond located east of Area II, this concentration was below the chronic EMEG (10 ppb) for noncancer health effects in adults. In addition, it is important to note that the comparison values used in this evaluation represent a daily drinking water exposure. Individuals at the Bird Landfill would likely be exposed less frequently and to significantly less contaminated surface water through incidental ingestion and dermal contact. For example, assuming a child ingests 0.05 liters (about a mouthful) of surface water contaminated with the maximum concentration of arsenic for 2 days a week over 26 weeks for a 10-year period, an increased cancer risk would not be expected.[3]

D. Exposure to Soil/Sediment

Unauthorized off-road vehicle use and trespassing have been reported at the site (GZA 2003). The only soil samples collected from the Bird Landfill site were sediment and wetland soil

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[3] Cancer Effects Exposure Factor = $\frac{(52 \text{ days/year})\ (10 \text{ years})}{(365 \text{ days/year})\ (70 \text{ years})} = 0.02$

Cancer Effects Exposure Dose (Adult) = $\frac{(0.006 \text{ mg/L})\ (0.05 \text{ L/day})\ (0.02)}{35 \text{ kg}} = 1.7 \times 10^{-7}$ mg/kg/day

Cancer Risk (Adult) = 1.7×10^{-7} mg/kg/day x 1.5 $(\text{mg/kg/day})^{-1} = 2.6 \times 10^{-7}$

samples collected from specific locations around Areas I and II. While it is not possible to evaluate incidental ingestion and dermal contact with onsite soil for trespassers for the rest of the site, it is important to note that daily cover consisting of stone dust or silty sand was used at the landfill while it was open. The stone dust was brought in from Bird, Inc.'s asphalt shingle roofing plant, and the source of the silty sand was S.M. Lorusso's West Sand Plant, also located in Walpole (GZA 1998). Both Areas I and II are now capped.

Incidental ingestion of and dermal contact with metals identified in sediment or wetland soil samples could have been possible in the past, present or future for adults or children who may trespass on the Bird Landfill site. However, with the exception of iron, none of the metals detected in sediment or wetland soils at the Bird Landfill exceeded health-based comparison values for residential soil exposure. All of the metals detected in sediment and wetland soils were within the range of background concentrations. In addition, a trespasser would likely be exposed less frequently and for a shorter duration than in a residential scenario. While iron was detected above the EPA RBC for residential soil in sediment from a groundwater seep west of Area I and in a stream south of Area II, both concentrations were below the RBC for industrial soil. Therefore, it is unlikely that individuals who might trespass in these areas would have sufficient exposure to result in adverse health effects (Table 4).

VII. CANCER INCIDENCE ANALYSES

In response to community concerns, the MDPH conducted an evaluation of the occurrence of cancer in the town of Walpole as a whole and its individual census tracts. In addition, the pattern of cancer was evaluated at the neighborhood level to identify any unusual patterns of cancer diagnoses in proximity to the Bird Landfill or in any other area of Walpole.

A. Methods for Analyzing Cancer Incidence Data

1) Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–2000 were obtained for the town of Walpole from the Massachusetts Cancer Registry (MCR), a division of the Center for Health Information, Statistics, Research and Evaluation within the MDPH. Seven

cancer types were evaluated, including cancers of the bladder, kidney, liver, and lung and bronchus, as well as Hodgkin's disease, leukemia, and mesothelioma. 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 on the basis of elevations observed at the town level in a preliminary review of cancer rates in Walpole, potential associations with contaminants of concern at the Bird 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 seven cancer types and diagnosed among residents of Walpole 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 six months of the date of diagnosis (M.G.L. c.111s.111B). This information is kept in a confidential database. Data are collected on a daily basis and are reviewed for accuracy and completeness on an annual basis. This process corrects misclassification of data (i.e., city/town misassignment). Once these steps are finished, the data for that year are considered "complete." Due to the volume of information received by the MCR, the large number of reporting facilities, and the six-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of two years prior to the current date. The 19-year period 1982–2000 constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis.[4]

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

[4] The data summarized in this report are drawn from data entered on MCR computer files before May 9, 2005. 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.

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 these analyses.

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital or diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different primary site, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than two months after the initial diagnosis (MCR 1996). Therefore, duplicate reports of an individual diagnosed with cancer were removed from the analyses whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases. In the town of Walpole, two duplicate reports were identified during the years 1982–2000 and excluded from the analyses.

2) Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer diagnoses occurred in Walpole or its individual census tracts, 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 1982–2000 for each of the seven primary cancer types for Walpole as a whole and its three census tracts (CTs) as well as for three smaller time periods (e.g., 1982–1987, 1988–1993, and 1994–2000) in order to evaluate patterns or trends in cancer incidence over time.

To calculate standardized incidence ratios, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990,

and 2000 United States census data for Walpole (U.S. DOC. 1980, 1990, 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990, 1991, and 1997). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the 10-year interval between each census.[5]

Because accurate age group and gender specific population data are required to calculate SIRs, the CT is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a county as defined by the 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). According to the 2000 United States Census, the town of Walpole is subdivided into three census tracts (CTs 4111, 4112, and 4113) as shown in Figure 1 (U.S. DOC. 2000).

3) Interpretation of a Standardized Incidence Ratio (SIR)

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

Specifically, an SIR is the ratio of the observed number of cancer 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.

[5] 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.

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 (e.g., 105-130), there is a statistically significant excess in the number of cancer diagnoses. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45-96), 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 lung and bronchus, bladder, and kidney 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 Walpole who were diagnosed with the seven 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, and 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 2004). 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 (i.e., neighborhoods). The geographic distribution was determined using a qualitative evaluation of the point pattern of cancer diagnoses in Walpole. 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 two years of an individual's diagnosis. In accordance with Massachusetts laws aimed at protecting the confidentiality of patients (M.G.L. c.111. s 24A), maps of the locations of individuals with cancer cannot be provided in this report.

B. Cancer Incidence in Walpole

The following section presents the results of the cancer incidence analyses for Walpole and its individual census tracts during the 19-year time period 1982–2000. Analysis by smaller geographic areas (i.e., census tracts) helps in understanding the incidence of cancer town-wide which may be explained by an increase or decrease in cases in a particular geographic area of the town. To evaluate possible trends over time, these data were also analyzed by three smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–2000). Although SIRs and 95% confidence intervals were not calculated for some cancer types in smaller time periods due to small numbers of observed diagnoses (i.e., fewer than five), the expected number of diagnoses was calculated to determine whether excess numbers of diagnoses were occurring. These data are summarized in Tables 6a–12d.

1) Bladder Cancer

During the 19-year time period 1982–2000, bladder cancer occurred more often than expected in Walpole as a whole (73 diagnoses observed vs. 65.5 expected, SIR = 112). The observed

elevation, which was not statistically significant (95% CI = 87-140), was primarily due to an elevation in incidence among females in the town. Specifically, 23 diagnoses occurred among females compared to 16.6 expected (SIR = 138, 95% CI = 88-207). Males were diagnosed with bladder cancer about as expected (50 diagnoses observed vs. 48.8 expected, SIR = 102). There were no apparent trends over time in the incidence of bladder cancer in Walpole. During the earliest time period evaluated, 1982–1987, this cancer type occurred approximately at or near expected rates among males and females. An elevation in incidence was noted during 1988–1993, when 26 diagnoses were observed compared to about 20 expected (SIR = 130). The overall elevation was attributed to a statistically significant elevation observed among females in the town during this time period. Specifically, 12 diagnoses were observed among females compared to five expected (SIR = 239, 95% CI = 124-418). During more recent years (i.e., 1994–2000), bladder cancer incidence returned closer to expected rates for males and females combined (29 diagnoses observed vs. 27.3 expected, SIR = 106). Bladder cancer also occurred at about the rates expected among females (8 diagnoses observed vs. 7.3 expected, SIR = 110) during this time.

In CT 4113, where the Bird Landfill, MacDonald Circle, and Swan Pond Village are located, bladder cancer occurred slightly less often than expected during 1982–2000 (19 diagnoses observed vs. 21.3 expected, SIR = 89). However, different trends were observed when these data were analyzed for males and females separately. Specifically, males were diagnosed with bladder cancer less often than expected during this time period (10 diagnoses observed vs. 15.8 expected, SIR = 63), while females were diagnosed more often than expected (9 diagnoses observed vs. 5.5 expected, SIR = 164). The observed elevation among females, which was not statistically significant (95% CI = 88-207), was attributed to an increase in bladder cancer diagnoses among females in this CT during 1988–1993 (6 diagnoses observed vs. 1.6 expected, SIR = 382). This elevation was statistically significant. Bladder cancer occurred less often than expected among both males and females in CT 4113 during 1982–1987 and 1994–2000.

Residents of CTs 4111 and 4112 experienced slight elevations in bladder cancer incidence during the 19-year time period 1982–2000 and in some smaller time periods, but the observed elevations were generally based on small numbers of diagnoses over the expected numbers and were not statistically significant. For example, in CT 4111, slight elevations were noted in the

incidence of bladder cancer among males during 1982–1987 and 1994–2000 and among females during 1988–1993. In CT 4112, males were diagnosed approximately at or near the expected rates in each of the smaller time periods. Females in this census tract experienced bladder cancer approximately at or below expected during 1982–1987 and 1988–1993, but were diagnosed more often than expected during 1994–2000 (5 diagnoses observed vs. 1.8 expected, SIR = 274). Again, this elevation was not statistically significant (95% CI = 88-640). See Tables 6a–6d for a summary of bladder cancer incidence in Walpole during 1982–2000.

2) Hodgkin's Disease

The incidence of Hodgkin's disease was elevated in Walpole during the 19-year time period 1982–2000 (19 diagnoses observed vs. 14.2 expected, SIR = 134). This elevation was the result of an increase in diagnoses among females in the town (11 diagnoses observed vs. 6.0 expected, SIR = 184). Neither of these elevations was statistically significant. During 1982–1987, one diagnosis was observed among females compared to almost two diagnoses expected. However, during 1988–1993, females experienced a statistically significant elevation in the incidence of Hodgkin's disease in the town as a whole (6 diagnoses observed vs. 2.0 expected, SIR = 301, 95% CI = 110-656). Incidence remained elevated among females during 1994–2000 (4 diagnoses observed vs. 2.3 expected), but the elevation was based on 1-2 excess diagnoses than expected. Males in Walpole experienced Hodgkin's disease about as expected during the overall time period 1982–2000 (8 diagnoses observed vs. 8.2 expected, SIR = 98) and during each of the smaller time periods evaluated.

The townwide elevations in Hodgkin's disease incidence observed among females during the overall time period and some smaller time periods was the result of slight increases in each of Walpole's three census tracts during some time periods. During 1982–1987, there were no diagnoses of Hodgkin's disease reported among females in CTs 4111 and 4112 compared to 0.6 and 0.5 expected, respectively. One female was diagnosed with this cancer type in CT 4113 compared to 0.6 expected during this time period. During 1988–1993, there were three diagnoses reported among females in CT 4111 vs. 0.7 expected, one diagnosis in CT 4112 vs. 0.6 expected, and two diagnoses in CT 4113 vs. 0.7 expected. During 1994–2000, there were no diagnoses of Hodgkin's disease reported among females in CT 4111 vs. 0.8 expected, two

diagnoses in 4112 vs. 0.6 expected, and two diagnoses in CT 4113 vs. 0.9 expected. (SIRs and 95% confidence intervals were not calculated because fewer than five diagnoses were observed in each census tract during each time period.) Males were diagnosed with Hodgkin's disease approximately as expected in each Walpole census tract during each time period evaluated. See Tables 7a–7d for a summary of this information.

3) Kidney Cancer

The incidence of kidney cancer was greater than expected in Walpole during 1982–2000 (48 diagnoses observed vs. 41.5 expected, SIR = 116). The overall elevation among males and females combined was not statistically significant (95% CI = 85-153), but females in Walpole experienced a statistically significant elevation in the incidence of kidney cancer (25 diagnoses observed vs. 15.2 expected, SIR = 164, 95% CI = 106-242). Males were diagnosed with kidney cancer less often than expected (23 diagnoses observed vs. 26.2 expected, SIR = 88). The statistically significant elevation observed among females during the overall time period reflected non-statistically significant increases in diagnoses during each of the smaller time periods evaluated. During 1982–1987, five females were diagnosed with kidney cancer compared to 3.4 diagnoses expected (SIR = 149). During 1988–1993, 10 females were diagnosed with kidney cancer compared to 4.8 diagnoses expected (SIR = 208), a borderline statistically significant result (95% CI = 100-382). Finally, during 1994–2000, ten females were diagnosed with kidney cancer compared to 7.4 diagnoses expected (SIR = 134).

Analysis of incidence by census tract for the 1982–2000 time period revealed that while males in each of Walpole's three census tracts experienced kidney cancer at or below the rates expected, females in CTs 4111 and 4113 experienced elevations in incidence, which led to the statistically significant elevation observed among females in the town as a whole. In CT 4111, 11 females were diagnosed with kidney cancer during 1982–2000 compared to 5.7 expected (SIR = 191). The elevation observed among females was not statistically significant and was due to slight increases in incidence during each of the smaller time periods. During 1982–1987, four females were diagnosed with kidney cancer in CT 4111 compared to 1.3 expected. During 1988–1993, three diagnoses occurred among females in this census tract compared to 1.8 expected. Finally, during 1994–2000, four diagnoses were observed among females compared to 2.5 expected.

Males in CT 4111 were diagnosed with kidney cancer as expected during the 19-year time period 1982–2000 (10 diagnoses observed vs. 10.1 expected, SIR = 99); there were no consistent trends over time. During 1982–1987, two kidney cancer diagnoses were observed among males compared to 2.0 expected. During 1988–1993, there were six diagnoses reported compared to 3.3 expected. Finally, during 1994–2000, two males were diagnosed with kidney cancer compared to 4.4 expected.

In CT 4113, where the Bird Landfill is located, kidney cancer occurred about as expected among both males and females during 1982–1987. During 1988–1993, males in this census tract were diagnosed about as expected while females experienced a statistically significant elevation in kidney cancer (6 diagnoses observed vs. 1.6 expected, SIR = 381, 95% CI = 139-830). The incidence of kidney cancer decreased among both males and females in CT 4113 during 1994–2000, with a total of three diagnoses observed compared to almost eight expected during this time period. In CT 4112, males and females experienced kidney cancer approximately at or below the rates expected during 1982–1987 and 1988–1993. However, both males and females in this census tract experienced elevations in incidence during more recent years. Specifically, there were five diagnoses reported among males in this census tract during 1994–2000 compared to 3.2 expected (not statistically significant) and four diagnoses among females compared to 2.0 expected. These data are summarized in Tables 8a–8d.

4) Leukemia

Leukemia occurred more often than expected in Walpole during 1982–2000 (40 diagnoses observed vs. 35.3 expected, SIR = 113). This elevation, which was not statistically significant, was the result of an increase in diagnoses among females in the town (20 diagnoses observed vs. 14.7 expected, SIR = 136). Males in the town experienced leukemia about as expected (20 diagnoses observed vs. 20.5 expected, SIR = 97). During 1982–1987, the incidence of this cancer type in Walpole was slightly lower than expected (6 diagnoses observed vs. 8.7 expected, SIR =69). Incidence increased during the 1988–1993 time period, during which time 14 diagnoses were observed compared to 9.8 expected (SIR = 143). This elevation was not statistically significant and was based on approximately three additional diagnoses over the expected number observed among males and about one additional diagnosis observed among

females. Leukemia incidence remained slightly elevated during 1994–2000 (20 diagnoses observed vs. 17.7 expected, SIR = 113). However, different trends were noted when these data were evaluated separately by gender. While males experienced fewer leukemia diagnoses than expected (6 diagnoses observed vs. 9.9 expected, SIR = 60), females were diagnosed with leukemia almost twice as often as expected (14 diagnoses observed vs. 7.8 expected, SIR = 181). Moreover, the elevation observed among females during this time period was borderline statistically significant (95% CI = 99-303). As shown in Table 9d, this townwide elevation was primarily the result of slight increases in leukemia diagnoses among females in each of Walpole's three census tracts.

While residents of CT 4111 were diagnosed with leukemia less often than expected during 1982–2000, slight elevations in leukemia incidence were noted in CTs 4112 and 4113. In CT 4111, leukemia occurred approximately at or below expected during each of the smaller time periods evaluated, although somewhat different trends were observed among males and females when evaluated separately by gender. For example, an elevation in incidence was noted among females in this census tract during 1994–2000 (5 diagnoses observed vs. 2.6 expected). The overall elevation in leukemia incidence observed in CT 4112 during 1982–2000 was primarily attributed to increases in diagnoses among females in this census tract during 1988–1993 and 1994–2000. In CT 4113, where the Bird Landfill is located, 16 individuals were diagnosed with leukemia compared to 12 expected (SIR = 134) during the overall time period 1982–2000. This elevation was not statistically significant (95% CI = 76-217). During 1982–1987, one resident of CT 4113 was diagnosed with leukemia compared to almost three diagnoses expected. Elevations in incidence were noted in this CT during 1988–1993 and 1994–2000, but these were based on fewer than three excess diagnoses and were not statistically significant. Specifically, five diagnoses occurred in CT 4113 during 1988–1993 compared to 3.2 expected (SIR = 155) and ten diagnoses occurred during 1994–2000 compared to 7.1 expected (SIR = 141). See Tables 9a–9d for a summary of leukemia incidence results by time period and census tract in Walpole.

5) Liver Cancer

The incidence of liver cancer was lower than expected in Walpole during 1982–2000. Specifically, seven diagnoses were observed during this time period compared to about 10

diagnoses expected (SIR = 69). Six diagnoses were observed among males compared to 7.6 expected (SIR = 79) and one diagnosis was observed among females compared to 2.5 expected. In addition, liver cancer occurred less often than expected in the town as a whole during each of the smaller time periods evaluated. Residents in each of Walpole's three CTs experienced liver cancer approximately at or below expected rates during the overall time period. Similar trends were observed when these data were evaluated by smaller time periods. In CT 4113, where the Bird Landfill is located, one individual was diagnosed with liver cancer during the 19-year time period evaluated compared to 3.4 diagnoses expected. See Tables 10a–10d for a summary of these data.

6) Lung and Bronchus Cancer

Lung and bronchus cancer was diagnosed less often than expected among residents of Walpole during 1982–2000 (249 diagnoses observed vs. 258.6 expected, SIR = 96). While females were diagnosed with lung and bronchus cancer as expected (102 diagnoses observed vs. 101.6 expected, SIR =100), males were diagnosed less often than expected (147 diagnoses observed vs. 157.0 expected, SIR = 94). During the earliest time period, 1982–1987, lung and bronchus cancer occurred more often than expected in Walpole (72 diagnoses observed vs. 65.0 expected, SIR = 111), with similar trends among males and females when these data were evaluated separately by gender. During 1988–1993, the incidence of lung and bronchus cancer was lower than expected (64 diagnoses observed vs. 80.1 expected, SIR = 80). Although females were diagnosed with lung and bronchus cancer at about the rate expected during this time period, males experienced lower-than-expected incidence of lung and bronchus cancer (35 diagnoses observed among males vs. 49.3 expected, SIR = 71). This SIR was statistically significant (95% CI = 49-99). In more recent years (i.e., 1994–2000), the incidence of lung and bronchus cancer was about as expected among males and females combined and among males when evaluated separately by gender. Females were diagnosed with lung and bronchus cancer less often than expected during 1994–2000 (46 diagnoses observed among females vs. 51.0 expected, SIR = 90).

Analysis by smaller geographic area revealed that the incidence of lung and bronchus cancer was at or below the state rate in each Walpole census tract during the 19-year time period 1982–2000.

The incidence of lung and bronchus cancer appears to have decreased among males over time in CT 4111 with respect to the statewide experience. No other consistent trends over time were noted in Walpole census tracts. Slight elevations in the incidence of lung and bronchus cancer were noted in some census tracts in some time periods; however, these were based on small numbers of additional diagnoses over the expected numbers and were not statistically significant. For example, elevations were noted among males in CT 4111 during 1982–1987 (22 diagnoses observed vs. 16.0 expected, SIR = 137) and among females in this census tract during 1988–1993 (15 diagnoses observed vs. 11.9 expected, SIR = 126). Residents of CT 4112 experienced a statistically significant deficit in lung and bronchus cancer diagnoses during 1988–1993 (12 diagnoses observed vs. 23.4 expected, SIR = 51, 95% CI = 27-90), with both males and females diagnosed about half as often as expected during this time period. In CT 4113, where the Bird Landfill is located, 81 individuals were diagnosed with lung and bronchus cancer during the overall time period, 1982–2000, compared to 86.7 expected (SIR = 93). During 1982–1987, the incidence of lung and bronchus cancer in this census tract was about as expected (21 diagnoses observed vs. 20.0 expected, SIR = 105). During 1988–1993, lung and bronchus cancer occurred less often than expected (19 diagnoses observed vs. 26.1 expected, SIR = 73). Finally, incidence remained lower than expected during 1994–2000 (41 diagnoses observed vs. 45.3 expected, SIR = 90). See Tables 11a–11d for these results.

7) Mesothelioma

During the overall time period, 1982–2000, mesothelioma occurred near the expected rate in Walpole. Specifically, four diagnoses were observed among males compared to 4.2 expected and two diagnoses were observed among females compared to about one expected. There was one diagnosis reported during 1982–1987, three during 1988–1993, and two during 1994–2000. SIRs for mesothelioma were not calculated for smaller time periods or individual census tracts due to small numbers of observed diagnoses (i.e., fewer than five). However, observed numbers were approximately at or near expected numbers when mesothelioma incidence was reviewed by census tract and smaller time period. In CT 4113 where the Bird Landfill, MacDonald Circle, and Swan Pond Village are located, one individual was diagnosed with mesothelioma during the 19-year time period 1982–2000. This person was diagnosed during the middle time period 1988–1993. See Tables 12a–12d for a summary of this information.

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 (e.g., smoking and occupation), was reviewed for those cancer types that were statistically significantly elevated in Walpole or one of its census tracts during 1982–2000 or one of the smaller time periods evaluated. These cancer types included bladder cancer, Hodgkin's disease, and kidney cancer. Information for each of these cancer types was compared to known or established incidence trends to assess whether any unexpected patterns exist among these cases. For more information regarding risk factors associated with these and the other 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, Hodgkin's disease, and kidney cancer. A review of age group specific SIRs by census tract was not possible because of the small numbers of diagnoses in each group. However, where there was a statistically significant elevation of cancer diagnoses in Walpole or its census tracts, the distribution of diagnoses by age was reviewed.

Tobacco use is a known or suggested causal risk factor in several types of cancer, including cancers of the bladder and kidney. The smoking history of individuals diagnosed with these cancer types in Walpole was reviewed to assess the role tobacco smoking may have played in the development of these types of cancer among residents.

In some studies, an association has been found with specific occupational exposure and an increase in the incidence of bladder cancer, Hodgkin's disease, and kidney cancer. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types to determine the role that occupational factors may have played in the development of these types of cancer in Walpole.

1) *Bladder Cancer*

Males are four times more likely to develop bladder cancer than females (ACS, 2004a). While males in Walpole experienced bladder cancer about as expected during 1982–2000, the incidence of this cancer type was elevated among females. Statistically significant elevations were noted among females townwide and in CT 4113 during 1988–1993. Nationally, the risk of bladder cancer increases with age and over 60% of people diagnosed with bladder cancer are between the ages of 65 and 85 years (ACS, 2004a). Of the 73 individuals diagnosed with bladder cancer in the town of Walpole as a whole during 1982–2000, the average age at diagnosis was 70 and 68% (n = 50) were aged 65 or older. The average age at diagnosis was also 70 for the 12 females diagnosed with bladder cancer during 1988–1993. Of the six females diagnosed during this time period in CT 4113, the average age at diagnosis was 63.

The most well established risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS, 2004a). 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). In Walpole, about two thirds of the individuals diagnosed with bladder cancer during 1982–2000 and for whom smoking history was known were current or former smokers at the time of diagnosis (66%, n = 41). Smoking history was unknown for the remaining 11 individuals. Among the 12 females diagnosed with bladder cancer during 1988–1993, six of the 10 with a known smoking status were current or former smokers at the time of diagnosis and smoking history was unknown for two other individuals. Among the six females diagnosed in CT 4113 during this time period, three were current or former smokers at the time of diagnosis, one was a nonsmoker, and smoking history was unknown for two individuals. Based on this information, it is likely that smoking played a role in the development of bladder cancer among some individuals in Walpole.

Studies have revealed a number of occupations that are associated with bladder cancer. Exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the United States (Johansson and Cohen, 1997). Occupational

exposure to aromatic amines, such as benzidine and 2-naphthylamine, increases the risk of bladder cancer (ACS, 2004a). 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, 2004a; 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, 2004a).

Occupation as reported to the MCR was reviewed for the 12 females in Walpole diagnosed with bladder cancer during 1988–1993 when a statistically significant elevation was observed. Review of this information did not indicate any jobs that are thought to be associated with the development of bladder cancer. However, occupation was reported as retired, at home, or unknown for almost half of the individuals (n=5), therefore, the role that occupational exposures may have played in the incidence of bladder cancer among females in Walpole is unclear.

2) Hodgkin's Disease

Epidemiologic studies have shown that Hodgkin's disease is more common among men than women. In Walpole, during the 19-year time period 1982–2000, males were diagnosed with Hodgkin's disease about as often as expected. However, females were diagnosed with this cancer type more often than expected. This elevation was primarily attributed to a statistically significant elevation observed among females in the town during 1988–1993. Hodgkin's disease can occur in both children and adults but is more common in two age groups: early adulthood (ages 15 to 40, usually 25 to 30) and late adulthood (ages 55 and up) (ACS, 2005). The pattern of Hodgkin's disease in Walpole during 1982–2000 was generally consistent with this trend: almost 60% (n = 11) of diagnoses occurred among individuals aged 20 to 35 and 26% (n = 5)

occurred among individuals over the age of 55. (No individuals under age 20 were diagnosed with this cancer type in Walpole.) Among the six females diagnosed with Hodgkin's disease during 1988–1993, five were between the ages of 20 and 32 and one was over age 55.

Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller, 1996). Occupational information was available for two of the seven females diagnosed with Hodgkin's disease during 1988–1993. Based on the reported occupation, workplace exposures related to Hodgkin's disease would have been unlikely for both of these individuals. Occupation was reported as at home, unknown, or retired for five of the females diagnosed during this time period. Therefore, it was not possible to determine whether occupational exposures contributed to the pattern of Hodgkin's disease among females in Walpole.

3) Kidney Cancer

The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of this cancer type. Kidney cancer is twice as common in males as it is in females, and the incidence most often occurs in the fifth and sixth decades of life (50–70 year age group) (ACS, 2004b). In Walpole, males were diagnosed with kidney cancer less often than expected during 1982–2000. However, a statistically significant elevation in the incidence of this cancer type occurred among females in the town during the overall time period. Females in CT 4113 experienced a statistically significant elevation during 1988–1993. The average age at diagnosis for the 48 males and females diagnosed with kidney cancer in Walpole during 1982–2000 was 64 years and 88% (n = 42) of the diagnoses occurred among individuals over the age of 50. Among females only, the average age at diagnosis was 65 years. In CT 4113, the average age at diagnosis for females diagnosed during 1988–1993 was 62 years. These trends are consistent with established patterns of disease in the general population.

Cigarette smoking is the most important known risk factor for kidney cancer. Smoking increases the risk of developing kidney cancer by about 40% (ACS, 2004b). In Walpole, 67% (n=28) of

individuals diagnosed with kidney cancer during 1982–2000 and with a known smoking history were current or former smokers at the time of diagnosis. Smoking history was unknown for the remaining six individuals. Similarly, 67% of females with a known smoking history were current or former smokers at the time of diagnosis and smoking history was unknown for the remaining four individuals. Among the six females diagnosed with kidney cancer in CT 4113 during 1988–1993 with a known smoking history, two were current or former smokers at diagnosis. Smoking history was unknown for the other two females.

Some studies have suggested that environmental and occupational factors may be associated with the development of kidney cancer. For example, an increased incidence of this cancer type has been observed among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is also associated with an increased incidence of kidney cancer, particularly among men who smoke. In addition, workplace exposure to organic solvents, such as trichloroethylene (TCE), may increase the risk of this cancer (AC, 2004b). More recently, renal cell carcinoma (RCC), the most common type of kidney cancer, has been suggested to be associated with occupational exposure to petroleum, tar, and pitch products. However, studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between exposure to gasoline or other petroleum products and kidney cancer (Linehan et al., 1997; McLaughlin et al., 1996).

Occupational data as reported to the MCR was evaluated for all females diagnosed with kidney cancer in Walpole during 1982–2000. Review of this information did not reveal any jobs in which exposures related to the development of kidney cancer would have been likely. One individual reported a job as a factory worker, however, it is unclear whether this individual worked in an industry associated with exposures that might cause kidney cancer. It is important to note that occupation was listed as at home, retired, or unknown for 80% of these individuals (n = 20). Therefore, the possible role that occupational factors may have played in the incidence of kidney cancer among females in Walpole could not be determined.

D. Geographic Distribution

Place of residence at the time of diagnosis was mapped for each cancer type to determine whether a geographic concentration of diagnoses exists. In addition to calculating census-tract-

specific incidence ratios for seven cancer types, a qualitative evaluation was conducted to determine whether any one cancer type appeared to be concentrated in any area(s) within the town of Walpole. For confidentiality reasons, maps of the location of individuals diagnosed with cancer cannot be provided in this report.

In general, review of the geographic distribution of cancer in Walpole during 1982–2000 revealed no apparent spatial patterns at the neighborhood level that could not be attributed to factors such as areas of higher population density (e.g., the presence of multiunit housing complexes or nursing homes). For example, although small concentrations of some cancer types (e.g., bladder cancer, Hodgkin's disease, kidney cancer, leukemia, and lung and bronchus cancer) were observed in certain areas of town, in general the distribution of diagnoses seemed to coincide closely with the pattern of population in Walpole.

Statistically significant elevations in the incidence of some cancer types were observed in Walpole or one or more of its census tracts during one or more of the time periods evaluated in this report. However, when the geographic distribution of these cancers was evaluated at the neighborhood level, the patterns of diagnoses did not appear to be unusually clustered in any one area. For example, females in Walpole as a whole and in CT 4113 experienced statistically significant elevations in the incidence of bladder cancer during the middle time period 1988–1993. Also, a statistically significant elevation in the incidence of kidney cancer was noted among females townwide during the overall time period 1982–2000 and among females in CT 4113 during 1988–1993. Review of the geographic distribution of bladder and kidney cancer diagnoses for the town as a whole indicated that the majority of diagnoses were located in and around the center of town, consistent with the population density of Walpole. Of the six bladder cancer diagnoses among females in CT 4113 during 1988–1993, two were located in the MacDonald Circle neighborhood. None of the six females diagnosed with kidney cancer in CT 4113 during this time period lived in the MacDonald Circle neighborhood or Swan Pond Village. Rather, the majority of diagnoses were located in the northern part of this census tract and not near the Bird Landfill. Females in Walpole also experienced a statistically significant elevation in the incidence of Hodgkin's disease during 1988–1993; however, diagnoses were widely distributed throughout the town and were not concentrated in any one area of Walpole.

In CT 4113 where the Bird Landfill is located, there was one diagnosis of liver cancer, which has been associated with vinyl chloride and arsenic exposure, while about three diagnoses were expected, and the individual diagnosed with liver cancer was not located in close proximity to the landfill. While leukemia, was slightly elevated in CT 4113, none of the individuals diagnosed with this cancer types were located in close proximity to the site. Lung and bronchus cancer, associated with arsenic and lead exposure, occurred less than expected in CT 4113.

When examining the MacDonald Circle neighborhood, six residents were diagnosed with one of two different cancer types during 1982-2000: two with bladder cancer and four with lung and bronchus cancer. Review of information regarding age, gender, and smoking history for individuals diagnosed with cancer in this area indicates that the pattern of cancer was generally consistent with what would be expected given the types of cancer diagnosed. For example, four of the six individuals diagnosed in this area were current or former smokers (smoking history for the remaining two was known). Also, the years of diagnosis for these individuals varied throughout the 19 years examined, that is, no single year had more than one cancer diagnosis, indicating no apparent trend or pattern over time.

Six individuals in the Swan Pond Village neighborhood were diagnosed with one of the five different types of cancer: bladder cancer, Hodgkin's disease, kidney cancer, leukemia, and lung and bronchus cancer. This indicates the occurrence of different diseases and does not suggest the presence of a common factor (environmental or nonenvironmental) related to the diagnoses. All of these diagnoses occurred in 1999 and 2000. When information regarding age, gender, and smoking history were reviewed for individuals diagnosed with cancer in the Swan Pond Village area, no unusual patterns of diagnoses were identified. For example, the mean age at diagnosis for these individuals was 70 years of age, and two of the three individuals diagnosed with bladder, lung and bronchus, and kidney cancer were reported as current or former smokers at the time of diagnosis.

The geographic distribution of each of the seven cancer types was also examined relative to the locations of 21e hazardous material and oil release sites mapped for Walpole. Based on a review of this information, there were no unusual concentrations of cancer diagnoses that would suggest a possible causal role for any of the sites evaluated.

VIII. DISCUSSION

This evaluation was initiated based on community concerns about the pattern of cancer in the Walpole neighborhoods of MacDonald Circle and Swan Pond Village and the presence of VOCs detected in groundwater at the Bird Landfill. As part of this public health assessment, MDPH evaluated both cancer incidence data for Walpole and reviewed available environmental information for the Bird Landfill to determine possible pathways of exposure for nearby residents. In addition, the pattern of cancer was evaluated within Walpole census tracts to identify any unusual concentrations of cancer diagnoses in particular areas of town or in relation to areas of environmental concern.

There are no private wells located in the path of groundwater estimated to be potentially impacted by the Bird Landfill site; however, future exposures to some metals and VOCs in groundwater are possible if private drinking water wells are installed in the path of contamination. While concentrations of contaminants such as arsenic, manganese, and vinyl chloride detected in groundwater at the Bird Landfill are likely to be lower down-gradient from the site, future installation and use of private wells close to the landfill could result in health concerns.

The Bird Landfill is located about 0.8 miles south of a Zone II water supply protection area whose aquifer deposits could be pumped for public drinking water in an extreme drought situation. Because the landfill is located outside the Zone II area, it is unlikely that groundwater from the landfill would reach municipal wells. However, if, under a worst case scenario contaminants detected in groundwater at the Bird Landfill migrate in a north/northeast direction and eventually reach the Zone II for the Mine Brook wells, it is likely that contaminants detected in groundwater at the landfill would be diluted below a level of health concern before reaching down-gradient municipal wells. In addition, municipal water supplies are tested and treated on a routine basis according to federal and state laws. Therefore, it is not expected that groundwater with contaminants originating from the Bird Landfill would be consumed as municipal drinking water.

With respect to indoor air, environmental investigations conducted at Area II of the landfill indicate that groundwater flows away from MacDonald Circle homes and therefore exposures

through vapor intrusion are not expected to be a potential exposure pathway for residents of this neighborhood (GZA 1998). To address possible vapor intrusion in Swan Pond Village homes located approximately 1500 feet down-gradient of the landfill, the Johnson-Ettinger model was employed using very conservative assumptions. The model predicted that it is unlikely that VOCs detected in groundwater at the Bird Landfill would present an exposure concern for indoor air in homes down-gradient of the site.

There is evidence of trespassing at the Bird Landfill, and thus, past, present, and future exposures to contaminants in surface water, soil, and sediment through dermal contact or incidental ingestion could be possible at the site. However, the majority of contaminants detected in surface water, sediment, and wetland soils, were below or within the range of background concentrations and comparison values and it is unlikely that a trespasser would be exposed for sufficient frequency and duration to result in adverse health effects or increased cancer risk.

The cancer types evaluated in this report were selected based on their potential association with contaminants of concern identified at the Bird Landfill site, resident concern over suspected elevations in some cancer types, and/or statistically significant elevations observed town-wide in published MCR reports. In the town of Walpole as a whole, cancer incidence rates for the seven cancer types evaluated during the 19-year time period, 1982–2000, and the three smaller time periods were generally near expected rates based on cancer incidence in the state of Massachusetts. In Walpole as a whole, bladder cancer (1988–1993) and kidney cancer (1982–2000 and 1988–1993) were statistically significantly elevated in females. This was due to statistically significant elevations in bladder cancer and kidney cancer among females during the middle time period 1988 to 1993 in CT 4113, where the Bird Landfill site is located. However, the risk factor analysis suggested that tobacco use likely played an important role in diagnoses of kidney and bladder cancer for some individuals and there were no trends in cancer diagnoses over time. There was also a statistically significant elevation in Hodgkin's disease among females in Walpole as a whole during the middle time period 1988 to 1993, with elevations in each CT contributing to the town-wide elevation. The geographic pattern of bladder cancer, Hodgkin's disease, and kidney cancer in females did not indicate a concentration or an atypical distribution of females within any CT, including CT 4113. Males in Walpole as a whole and in CT 4113 were diagnosed with these three cancer types approximately at or below the expected

rates. At the time this cancer incidence investigation was conducted, complete data records included cancer diagnoses that occurred from 1982–2000. The MCR recently released two additional years of cancer incidence data and therefore it was possible to also evaluate more recent data for bladder cancer, kidney cancer, and Hodgkin's disease. All three cancer types occurred less often than expected among Walpole males and females during 2001 and 2002. In CT 4113, three males were diagnosed with bladder cancer, kidney cancer, and Hodgkin's disease, and there were no females diagnosed with any of the three cancer types during these years.

In addition to an evaluation of cancer incidence rates, available risk factor information was reviewed for those cancer types that displayed statistically significant elevations in incidence in Walpole or one of its census tracts during 1982–2000 or one of the smaller time periods evaluated. In general, cancer trends observed in Walpole were similar to those seen in the general population and in Massachusetts. Data reviewed suggest that smoking likely played some role in the diagnosis of certain cancers (bladder and kidney cancer) among some individuals in Walpole. The role that occupational exposures may have played in the incidence of bladder cancer, Hodgkin's disease, and kidney cancer in Walpole is unclear due to incomplete information available from the MCR.

Finally, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer did not reveal any atypical spatial patterns that would suggest a common factor related to the incidence of cancer in Walpole as a whole or in the three census tracts that comprise the town. That is, no unusual concentrations of individuals diagnosed with the seven cancer types evaluated were observed in the vicinity of the Bird Landfill site, including the MacDonald Circle neighborhood, Swan Pond Village, or any other area in Walpole. 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 Bird Landfill is located, in the census tracts that divide the town, or in the town of Walpole as a whole during the 19-year time period, 1982–2000.

IX. CHILD HEALTH CONSIDERATIONS

ATSDR and MDPH recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at greater risk than adults form 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 Walpole is discussed in Section VI ("Cancer Incidence Analysis") of this report. As discussed before, future exposure to metals and VOCs could be possible for children if private drinking water wells are installed in the path of contamination. The MDPH recommends that the Town of Walpole carefully review future private well installation requests if proposed wells are to be installed in the path of the estimated extent of contaminated groundwater. Past, present, and future exposures to arsenic in surface water and iron 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 surface water and sediment at the Bird Landfill site 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 Bird Landfill site.

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 Walpole.

XI. CONCLUSIONS

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

- Except under extreme drought conditions, it is unlikely that groundwater contaminants detected at the Bird Landfill would reach the Zone II protection area for the Mine Brook wells located 0.8 miles north of the site, and therefore exposures through municipal drinking water would not be expected.

- Based on the levels of VOCs such as vinyl chloride detected in onsite groundwater, conservative indoor air concentrations predicted by the Johnson-Ettinger model, and the distance of homes north-northeast of the site, it is unlikely that contaminants detected in groundwater at the Bird Landfill would present an exposure concern for indoor air in down-gradient homes.

- Intermittent exposures to onsite surface water, sediment, and wetland soils are possible for trespassers at the Bird Landfill 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 19–year time period 1982–2000 occurred near expected rates in the Town of Walpole and in the three census tracts that comprise the town. In Walpole as a whole, bladder cancer (1988–1993) and kidney cancer (1982–2000 and 1988–1993) were statistically significantly elevated in females. The elevation was due to statistically significant elevations in bladder cancer and kidney cancer among females from 1988 to 1993 in CT 4113, where the Bird Landfill, MacDonald Circle, and Swan Pond Village are located. A statistically significant elevation in Hodgkin's disease was also observed among females from 1988 to 1993 in Walpole as a whole. An evaluation of available risk factor information suggested that tobacco use likely played an important role in diagnoses of kidney and bladder cancer for some individuals, and none of the seven cancer types were elevated in a consistent pattern over time or in any one area of Walpole.

- Review of the geographic distribution of the seven cancer types in Walpole revealed no apparent spatial patterns at the neighborhood level, including in the vicinity of the Bird Landfill site, MacDonald Circle, or the Swan Pond Village neighborhood.

- Based on the information reviewed in this evaluation, including available environmental data for the Bird Landfill site and risk factor information for individuals diagnosed with cancer, it does not appear that a common factor (environmental or nonenvironmental) played a major role in the incidence of cancer in the census tract containing the Bird Landfill site, MacDonald Circle, and the Swan Pond Village area, or in the town of Walpole as a whole during the 19-year time period, 1982–2000.

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

presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns. Therefore, based on MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to the cancer types evaluated in this analysis, ATSDR would classify the Bird Landfill site as posing no apparent public health hazard in the past and present. Since private wells could be installed in the path of contaminated groundwater north-northeast of the site making drinking water exposures possible, the Bird 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 town of Walpole review the testing and approval process currently in place for new private well construction to ensure contaminated groundwater at the Bird Landfill will not be consumed as drinking water by residents in the vicinity of the site.

- The MDPH recommends no further investigation of cancer incidence in relation to the Bird Landfill at this time, but will continue to monitor cancer incidence in the town of Walpole through the Massachusetts Cancer Registry.

XIII. PUBLIC HEALTH ACTION PLAN

The Public Health Action Plan for the Bird 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:

- The MDPH is available to assist the Walpole Board of Health in defining a testing and approval process for new private well construction in the vicinity of the Bird Landfill.

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

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PREPARER

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

XV. CERTIFICATION

The Public Health Assessment*, Evaluation of Cancer Incidence, 1982-2000, and Environmental Concerns Related to the Bird Landfill in Walpole, Norfolk 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, SPAB, DHAC, ATSDR

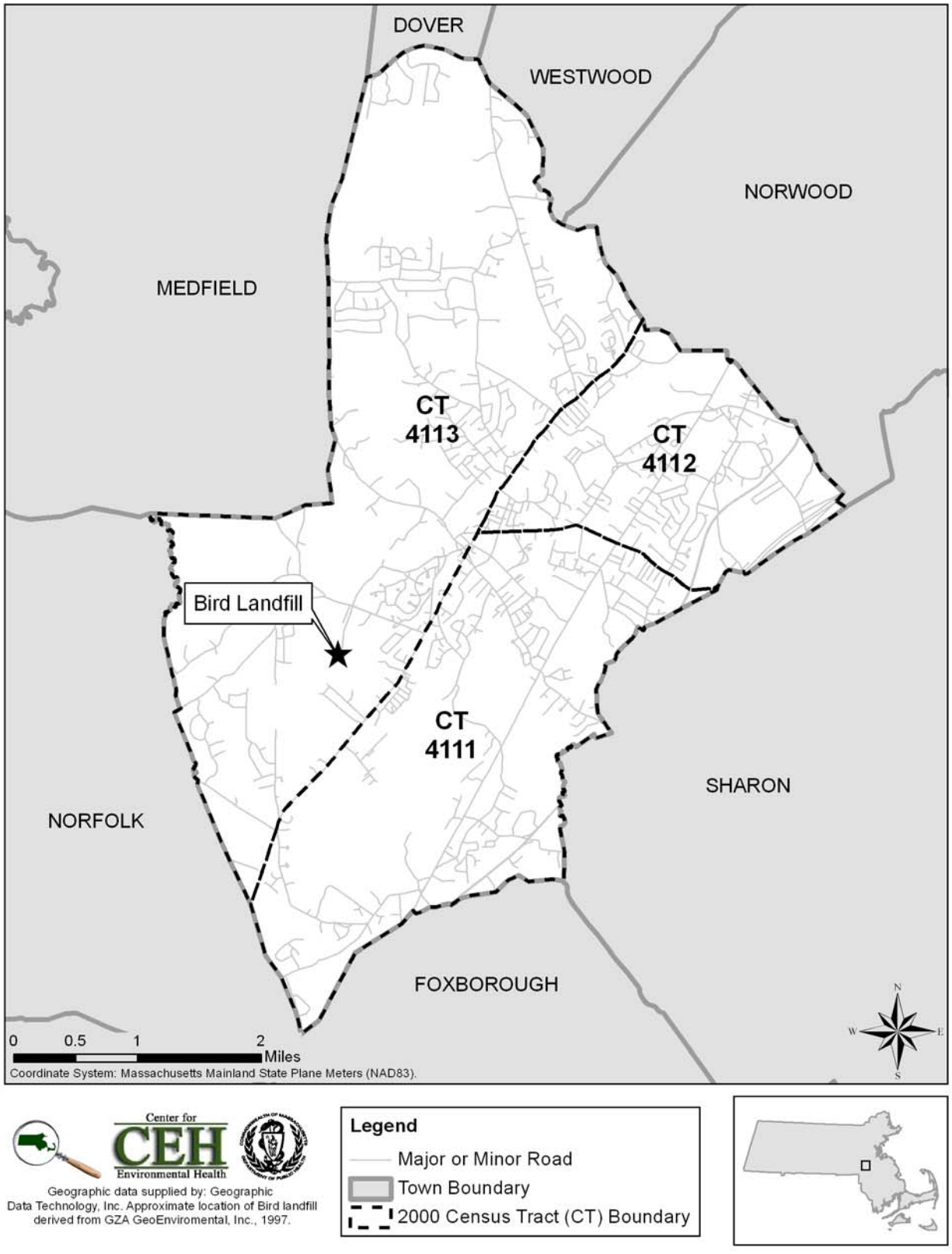
The Division of Health Assessment and Consultation, ATSDR, has reviewed this Public Health Assessment and concurs with its findings.

_____

Team Lead, CAT, SPAB, DHAC

FIGURES

Figure 1
Location of 2000 Census Tracts (CT)
Walpole, Massachusetts



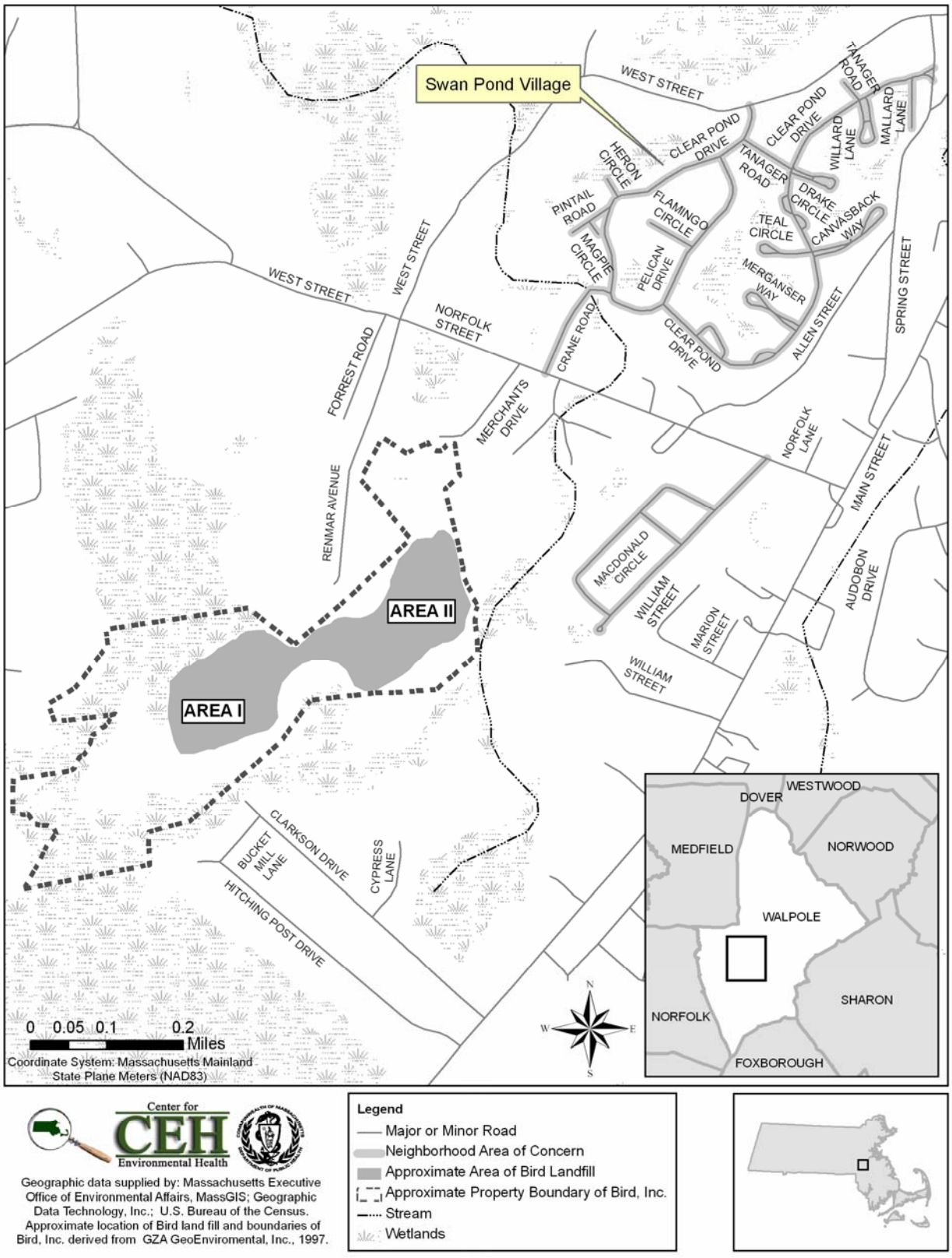
Center for CEH Environmental Health

Geographic data supplied by: Geographic Data Technology, Inc. Approximate location of Bird landfill derived from GZA GeoEnviromental, Inc., 1997.

Legend

- Major or Minor Road
- Town Boundary
- 2000 Census Tract (CT) Boundary

Figure 2
Location of Bird Landfill in Relation to Neighborhood Areas of Concern
Walpole, Massachusetts

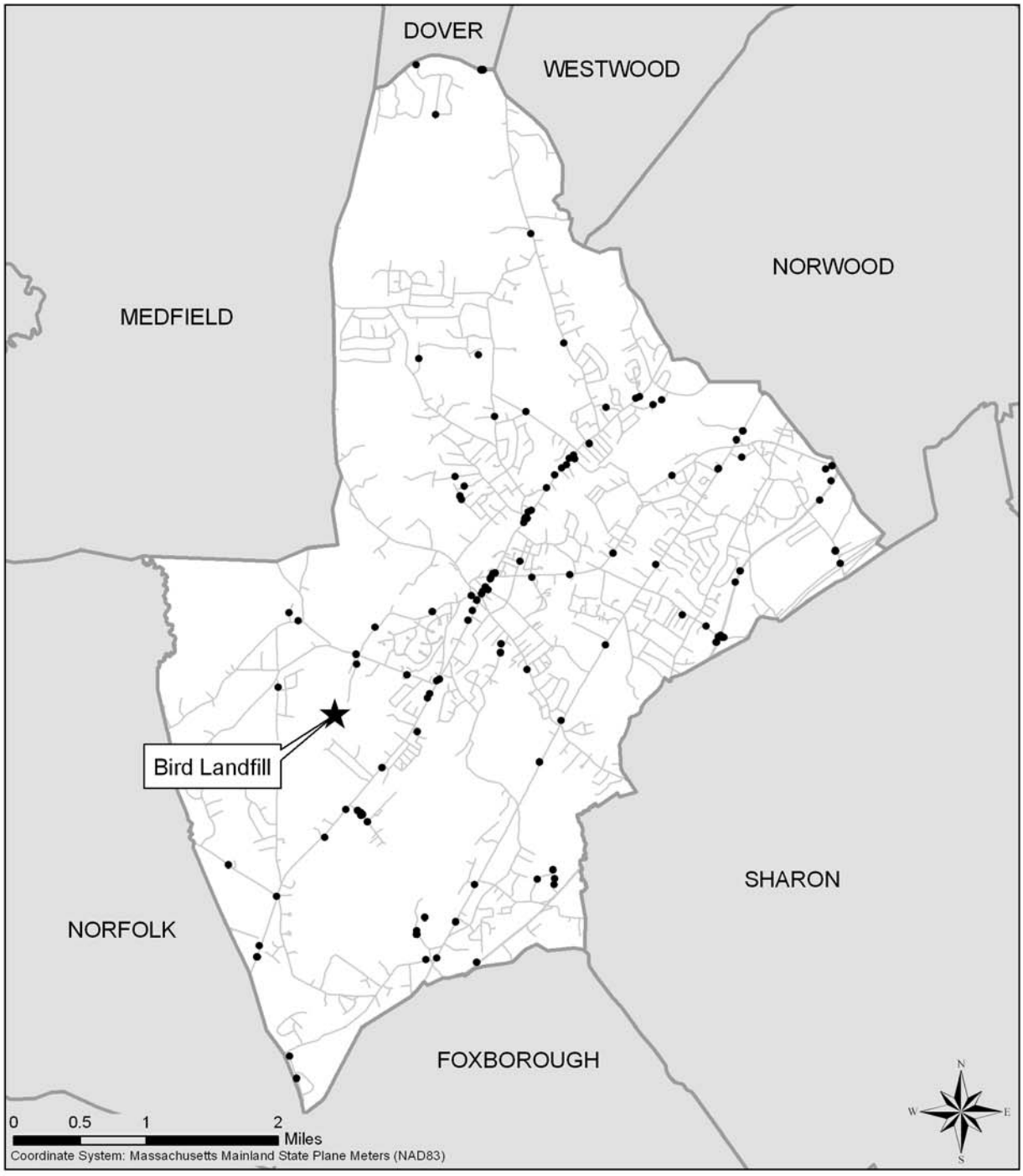


Center for CEH Environmental Health

Geographic data supplied by: Massachusetts Executive Office of Environmental Affairs, MassGIS; Geographic Data Technology, Inc.; U.S. Bureau of the Census. Approximate location of Bird land fill and boundaries of Bird, Inc. derived from GZA GeoEnvironmental, Inc., 1997.

Legend
- Major or Minor Road
- Neighborhood Area of Concern
- Approximate Area of Bird Landfill
- Approximate Property Boundary of Bird, Inc.
- Stream
- Wetlands

Figure 3
Location of MDEP 21E Hazardous Material and Oil Releases
Walpole, Massachusetts

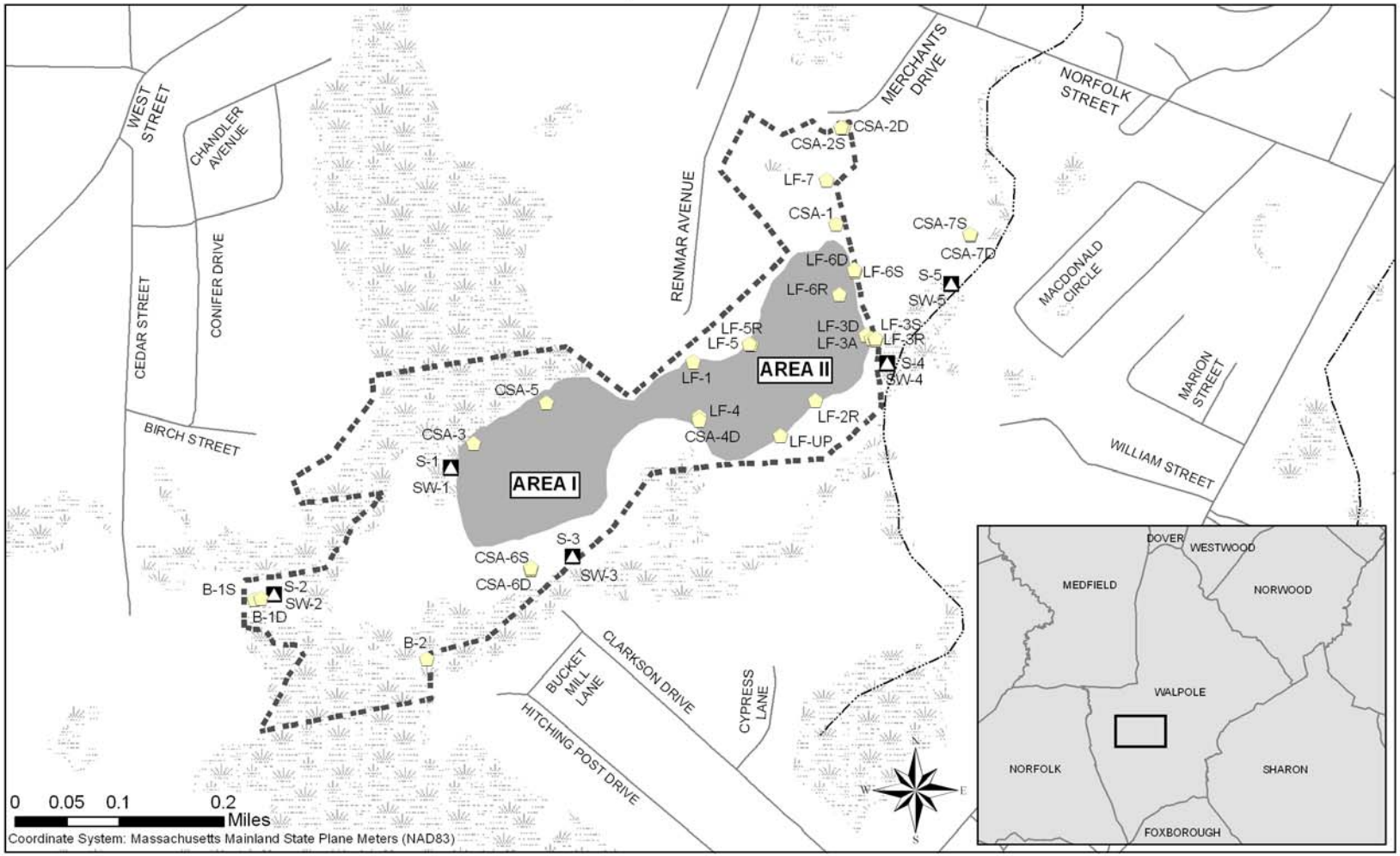


Center for CEH Environmental Health

Geographic data supplied by: Geographic Data Technology, Inc. Approximate location of Bird landfill derived from GZA GeoEnvironmental, Inc., 1997.

Legend
- MDEP 21E Sites
- Major or Minor Road
- Town Boundary

Figure 4
Approximate Groundwater, Surface Water, and Sediment Sampling Locations at the Bird Landfill
Walpole, Massachusetts



Center for CEH Environmental Health

Geographic data supplied by: Massachusetts Executive Office of Environmental Affairs, MassGIS; Geographic Data Technology, Inc. Approximate location of Bird landfill, boundaries of Bird, Inc. and sample locations derived from GZA GeoEnviromental, Inc. 1997.

Legend

- Surface Water Samples
- Sediment or Wetland Soil Samples
- Groundwater Monitoring Locations
- Wetlands
- Stream
- Major or Minor Road
- Approximate Area of Bird Landfill
- Approximate Property Boundary of Bird, Inc.

TABLES

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

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	N84-0434	UNKNOWN	NEPONSET ST. S.WALPOLE	7/12/1984	UNKNOWN	UNKNOWN	UNKNOWN
MAPPED	N87-1791	UNKNOWN	ENDEON ESTATE MYLOD RD	12/22/1987	#2 FUEL OIL	UST	UNKNOWN
MAPPED	N89-1718	UNKNOWN	ALBANY RD	10/13/1989	HYDRAULIC FLUID	MACHINERY	UNKNOWN
MAPPED	N89-2106	UNKNOWN	985 PROVIDENCE HIGHWAY	12/11/1989	GASOLINE	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N91-0515	L H JOHNSON CO	COBBLE KNOLL DR	4/18/1991	HYDRAULIC FLUID	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N92-1120	FOUR SEASONS ICE RINK	600 PROVIDENCE HWY	9/2/1992	WASTE OIL	DRUM	UNKNOWN
MAPPED	N92-1527	SKATING RINK	600 PROVIDENCE HWY	11/18/1992	AMMONIA	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N93-0024	POLE HOLE	SLEEPY HOLLOW RD	1/6/1993	UNKNOWN	UNKNOWN	UNKNOWN
MAPPED	N83-0083	UNKNOWN	95 WEST ST.	5/16/1983	#6 FUEL OIL	UNKNOWN	UNKNOWN
MAPPED	N83-0128	UNKNOWN	740 MAIN ST.	6/2/1983	TRANSFORMER OIL	UNKNOWN	UNKNOWN
MAPPED	N84-0182	UNKNOWN	RT.1 & RT.27	3/17/1984	GASOLINE	UNKNOWN	UNKNOWN
MAPPED	N85-0898	UNKNOWN	KENDALL TO 95 WEST ST.	11/25/1985	HYDRAULIC OIL	UNKNOWN	UNKNOWN
MAPPED	N85-0994	UNKNOWN	18 INDUSTRIAL RD.	12/30/1985	METHYLENE CHLORIDE	UNKNOWN	UNKNOWN
MAPPED	N86-0752	UNKNOWN	MILL POND/112 WASHINGTON ST.	8/18/1986	MISCELLANEOUS OIL	UNKNOWN	UNKNOWN
MAPPED	N86-1176	BECO	740 MAIN ST	UNKNOWN	MISCELLANEOUS OIL	UNKNOWN	UNKNOWN
MAPPED	N86-1227	UNKNOWN	112 WASHINGTON STREET	12/2/1986	NON PCB TRANS. OIL	UNKNOWN	UNKNOWN
MAPPED	N86-1254	UNKNOWN	100 NEPONSET STREET	12/9/1986	NO. 6 FUEL OIL	UST	UNKNOWN
MAPPED	N86-5003	UNKNOWN	333 CONEY ST.	4/10/1986	#2 FUEL OIL	UNKNOWN	UNKNOWN
MAPPED	N87-0149	UNKNOWN	21 SUMMER STREET	2/10/1987	UNKNOWN	UNKNOWN	UNKNOWN
MAPPED	N87-0355	UNKNOWN	95 WEST ST	7/18/1989	#2 FUEL OIL	UNKNOWN	UNKNOWN
MAPPED	N87-0696	UNKNOWN	2415 SOUTH MAIN ST	5/27/1987	#6 FUEL OIL	UNKNOWN	UNKNOWN
MAPPED	N87-0890	WALPOLE MA	CORNER RT.1 & RT 27	6/25/1987	UNKNOWN	DRUM	UNKNOWN
MAPPED	N87-1031	SOUTH WALPOLE	100 NEPONSET ST.	7/29/1987	MISCELLANEOUS OIL	UNKNOWN	UNKNOWN
MAPPED	N87-1091	SHAMROCK ROOFING	1724 WASHINGTON ST	UNKNOWN	ASPHALT	UNKNOWN	UNKNOWN
MAPPED	N87-1767	UNKNOWN	CORNER OF WINTER & MAIN STS	12/16/1987	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
MAPPED	N88-0498	UNKNOWN	WALPOLE STATE PRISON	UNKNOWN	TRANSFORMER OIL	TRANSFORMER	UNKNOWN
MAPPED	N88-0542	MOBIL STATION	751 MAIN STREET	UNKNOWN	GASOLINE	UST	UNKNOWN
MAPPED	N88-0856	UNKNOWN	HEMLOCK & CHERRY STREET	6/14/1988	HERBICIDES	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N88-0946	UNKNOWN	MCI	UNKNOWN	TRANSFORMER OIL	TRANSFORMER	UNKNOWN
MAPPED	N88-1174	MCI DIV OF CAPITAL PLAN AND OP	CEDAR JUNCTION	8/5/1988	WASTE OIL	UNKNOWN	UNKNOWN
MAPPED	N88-1188	UNKNOWN	RTES 1 & 27	8/8/1988	SOLVENTS	UNKNOWN	UNKNOWN
MAPPED	N88-1316	UNKNOWN	6 DUNBAR CT	UNKNOWN	#2 FUEL OIL	VEH. FUEL TANK	UNKNOWN
MAPPED	N88-1514	UNKNOWN	3 DONNER DRIVE	9/28/1988	UNKNOWN	UNKNOWN	UNKNOWN
MAPPED	N88-1823	KW ZION (JUNKYARD)	1700 MAIN STREET	UNKNOWN	GASOLINE	UNKNOWN	UNKNOWN
MAPPED	N89-****	UNKNOWN	923 MAIN ST	7/6/1989	PERCHLORETHYLENE	UNKNOWN	UNKNOWN
MAPPED	N89-0353	UNKNOWN	42 CEDAR ST	3/14/1989	MISCELLANEOUS OIL	ODOR	UNKNOWN

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	N89-0608	UNKNOWN	1171R MAIN ST.	UNKNOWN	WASTE OIL	DRUM	UNKNOWN
MAPPED	N89-0619	UNKNOWN	ALBANY ROAD/PILOTS WAY	UNKNOWN	PAINT	UNKNOWN	UNKNOWN
MAPPED	N89-0861	UNKNOWN	481 MAIN ST	5/25/1989	GASOLINE	AST	UNKNOWN
MAPPED	N89-0883	UNKNOWN	153 WASHINGTON ST	5/30/1989	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
MAPPED	N89-0982	UNKNOWN	953 MAIN ST.	UNKNOWN	PHOPLEX AC-261	DRUM	UNKNOWN
MAPPED	N89-1118	UNKNOWN	923 MAIN ST.	7/5/1989	PERCHLOROETHYLENE	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N89-1484	UNKNOWN	16 COUNTY ST	9/3/1989	#2 FUEL OIL	AST	UNKNOWN
MAPPED	N89-1647	UNKNOWN	180 MAIN ST.	9/29/1989	DIESEL FUEL	DRUM	UNKNOWN
MAPPED	N89-1856	UNKNOWN	100 NEPONSET ST.	10/30/1989	MISC	UST	UNKNOWN
MAPPED	N89-2044	BIRD MACHINE	100 NEPONSET ST	12/6/1989	HYDRAULIC FLUID	DRUM	UNKNOWN
MAPPED	N90-0025	UNKNOWN	187 GOULD ST.	1/4/1990	#2 FUEL OIL	AST	UNKNOWN
MAPPED	N90-1000	UNKNOWN	1171 MAIN ST.	6/21/1990	HYDRAULIC FLUID	HYDRAULIC LINE	UNKNOWN
MAPPED	N90-1090	UNKNOWN	740 MAIN ST.	7/9/1990	GASOLINE	PIPE/HOSE/LINE	UNKNOWN
MAPPED	N90-1593	MCI CEDAR JUNCTION	RTE 1A/MAIN ST	9/25/1990	MISC DETERGENTD	DRUM	UNKNOWN
MAPPED	N90-2035	UNKNOWN	740 MAIN ST.	12/17/1990	DIESEL FUEL	UNKNOWN	UNKNOWN
MAPPED	N91-0050	TRUCK TANK SPILL	460 MAIN ST	1/15/1991	DIESEL FUEL	TANK	UNKNOWN
MAPPED	N91-0378	ZION AUTOMOTIVE	1700 MAIN ST	UNKNOWN	MISCELLANEOUS OIL	UNKNOWN	UNKNOWN
MAPPED	N91-0412	CIBA CORNING	333 CONY ST	3/26/1991	MERCURY	UNKNOWN	UNKNOWN
MAPPED	N92-0552	MCI CEDAR JUNCTION	DEDHAM ST	5/1/1992	PAINT	DRUMS	UNKNOWN
MAPPED	N92-0827	RESIDENTIAL	15 CHAPMAN ST	6/30/1992	#2 FUEL OIL	AST	UNKNOWN
MAPPED	N92-1041	U-HAUL	1 PRODUCTION RD	8/17/1992	PAINTS,SOLVENTS,WASTE OIL	DRUM	UNKNOWN
MAPPED	N92-1156	REFUELING SPILL	25 INDUSTRIAL RD	9/10/1992	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
MAPPED	N92-1375	CIBA CONING DIAGNOSTICS	333 CONEY ST	11/19/1992	HYDROQUINONE	UNKNOWN	UNKNOWN
MAPPED	N93-0067	HEARTLAND PLAZA	514 MAIN ST	1/15/1993	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
MAPPED	N93-0853	STOP & SHOP	80 SOUTH ST	6/24/1993	HYDRAULIC FLUID	COMPACTOR	UNKNOWN
MAPPED	N93-1026	BIRD MACHINE CO	100 NEPONSET ST	8/1/1993	MACHINE COOLING OIL	STORAGE TANK	UNKNOWN
NOT MAPPED	N83-0114	UNKNOWN	WASHINGTON ST.	5/24/1983	UNKNOWN	UNKNOWN	UNKNOWN
NOT MAPPED	N83-0325	UNKNOWN	SOUTH ST.	10/11/1983	#6 FUEL OIL	UNKNOWN	UNKNOWN
NOT MAPPED	N84-0003	UNKNOWN	RT.1 S.WALPOLE	1/6/1984	GASOLINE	UNKNOWN	UNKNOWN
NOT MAPPED	N84-0179	UNKNOWN	WASHINGTON ST. E.WALPOLE	3/26/1984	GASOLINE	UST	UNKNOWN
NOT MAPPED	N84-0625	UNKNOWN	RT.1A	9/21/1984	GASOLINE	UST	UNKNOWN
NOT MAPPED	N85-0125	UNKNOWN	GAS STATION @ INTERSECTION RT.	2/22/1985	GASOLINE	UNKNOWN	UNKNOWN
NOT MAPPED	N85-0689	UNKNOWN	RT.1A	9/11/1985	#6 FUEL OIL	UNKNOWN	UNKNOWN
NOT MAPPED	N86-0101	UNKNOWN	TULMER ST.	2/15/1986	#2 FUEL OIL	UNKNOWN	UNKNOWN
NOT MAPPED	N86-0376	UNKNOWN	HEARTLAND PLAZA RTE.1A	5/16/1986	DIESEL FUEL	UNKNOWN	UNKNOWN
NOT MAPPED	N86-0506	UNKNOWN	RTE. 95N/BEFORE CONEY ST.EXIT	6/17/1986	DIESEL FUEL	UNKNOWN	UNKNOWN

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
NOT MAPPED	N86-1095	UNKNOWN	RTE.1 & UNION STREET	7/16/1986	WHITE MILKY SUBSTANCE	UNKNOWN	UNKNOWN
NOT MAPPED	N87-0526	UNKNOWN	RTE 1/STADIUM MOBIL	4/21/1987	GASOLINE	UNKNOWN	UNKNOWN
NOT MAPPED	N87-0575	UNKNOWN	SOUTH ST/FORMER KENDALL CO	4/29/1987	#6 FUEL OIL	UST	UNKNOWN
NOT MAPPED	N87-0676	UNKNOWN	OLD POST RD AT RT.1	5/19/1987	DIESEL FUEL	UNKNOWN	UNKNOWN
NOT MAPPED	N87-1137	UNKNOWN	RT 95N	UNKNOWN	#2 FUEL OIL	UNKNOWN	UNKNOWN
NOT MAPPED	N87-1639	WALPOLE/SHARON LINE	ROUTE 1 SOUTHBOUND	11/18/1987	GASOLINE	VEH. FUEL TANK	UNKNOWN
NOT MAPPED	N88-0412	UNKNOWN	RT 1 & UNION ST	UNKNOWN	GASOLINE	VEH. FUEL TANK	UNKNOWN
NOT MAPPED	N88-0887	POLE # 43/126-1	NORTH STREET	6/19/1900	TRANSFORMER OIL	TRANSFORMER	UNKNOWN
NOT MAPPED	N88-1111	ROUTE 1A	MAIN ST	7/29/1988	GASOLINE	PIPE/HOSE/LINE	UNKNOWN
NOT MAPPED	N88-1629	ROUTE 95 OFFRAMP	ROUTE 1 SOUTH BY	UNKNOWN	UNKNOWN	DRUM	UNKNOWN
NOT MAPPED	N88-2013	POLE #33-171	WASHINGTON STREET	12/21/1988	TRANSFORMER OIL	TRANSFORMER	UNKNOWN
NOT MAPPED	N89-0102	BICKFORDS	RTES 1 & 95	1/23/1989	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
NOT MAPPED	N89-1125	UNKNOWN	RTE # 1	7/7/1989	GARBAGE	INCINERATION AS	UNKNOWN
NOT MAPPED	N89-1212	KUHN TRANSPORTATION	WALPOLE H2O SUPPLY	8/1/1989	DIESEL FUEL	TANK	UNKNOWN
NOT MAPPED	N90-1235		RTE #1	7/27/1990	DIESEL FUEL	VEH. FUEL TANK	UNKNOWN
NOT MAPPED	N92-0180	PURITY SUPREME	RTE 1A - MAIN ST	2/14/1992	#2 FUEL OIL	PIPE/HOSE/LINE	UNKNOWN
NOT MAPPED	N92-0985	CONTAINER	WASHINGTON & SLAVE	8/4/1992	SULFURIC ACID	CONTAINER	UNKNOWN
NOT MAPPED	N92-1124	US POST OFFICE	COMMON ST @ CENTER	9/3/1992	#2 FUEL OIL	AST	UNKNOWN
NOT MAPPED	N92-1137	UNKNOWN		UNKNOWN	UNKNOWN	UNKNOWN	UNKNOWN
NOT MAPPED	N92-1306	POND SCUM	WILLWT POND	10/9/1992	GREEN MATERIAL	DRUM	UNKNOWN
MAPPED	3-0000024	FISH CHEMICAL & EQUIPMENT (INDUSTRIAL)	18 INDUSTRIAL RD	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	PIPE	WCSPRM
MAPPED	3-0000446	OLD COLONY GAS0LINE (FORMER, GAS STATION)	21 PROVIDENCE HWY	7/23/1986	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	DEPNDS
MAPPED	3-0000603	BLACKBURN & UNION PRIVILEGES (FORMER, MANUFACTURING)	SOUTH ST	4/15/1987	ASBESTOS	LAGOON, UNCONTAIN, UST	TIER1A
MAPPED	3-0001034	MCI WALPOLE (MUNICIPAL)	MAIN ST	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	TRANSFORM	TIERII
MAPPED	3-0001382	WIMBLETON TENNIS COURTS (COMMERCIAL)	20 COUNTY ST RTE 109	11/24/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	PENNFA
MAPPED	3-0001603	WOODWORKERS INC (COMMERCIAL)	767-777 EAST ST	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	RAO

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0001721	HOLLINGSWORTH & VOSE CO	112 WASHINGTON ST	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	DEPNFA
MAPPED	3-0001722	SEWER EXCAVATION	MAIN ST SPEAR AVE	1/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	TIER1D
MAPPED	3-0001723	BIRD AND SONS FMR	WASHINGTON ST	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	RAO
MAPPED	3-0001779	MAJORS SERVICE STATION FMR	745 MAIN ST	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	DEPNDS
MAPPED	3-0001801	HM GOULD & MOBIL OIL CO	295 UNION ST	1/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	TIERII
MAPPED	3-0001875	MOBIL STATION (GAS STATION)	751 MAIN ST	11/15/1988	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0001944	SPECIALTY CONVERTORS (INDUSTRIAL, WETLANDS)	2000 MAIN ST	1/15/1989	PETROLEUM BASED OIL, UNKNOWN CHEMICAL OF TYPE - OIL	DRUMS, LEACHFIELD, PIPE, SEPTIc	TIER1D
MAPPED	3-0002053	KENDALL MILL FMR (FORMER, INDUSTRIAL)	STATION ST W ST	10/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0002469	BIRD MACHINE CO CART PATH (FORMER, INDUSTRIAL, OPEN SPACE)	100 NEPONSET ST	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	LANDFILL	WCSPRM
MAPPED	3-0002494	J CONNOLLY & SONS (COMMERCIAL)	609 MAIN ST	1/15/1990	DIESEL FUEL	UNKNOWN	RAO
MAPPED	3-0002628	BIRD JOHNSON COMPANY (INDUSTRIAL, MANUFACTURING)	110 NORFOLK ST	3/28/1989	PETROLEUM BASED OIL	UST	RAO
MAPPED	3-0002742	PROPERTY (FARM, RESIDENTIAL)	1018 NORTH ST	10/23/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0002751	CYR OIL CO (GAS STATION)	MAIN ST N ST	1/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0003029	EXXON STATION (GAS STATION)	985 PROVIDENCE HWY	4/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	RAO
MAPPED	3-0003067	COMMERCIAL PROPERTY	28 INDUSTRIAL WAY	12/18/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	DEPNDS
MAPPED	3-0003190	SHELL STATION (GAS STATION)	920 MAIN ST	7/15/1990	UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	REMOPS
MAPPED	3-0003244	AMERICAN AUTO AUCTIONS FMR (AUTO DEALER, FORMER)	600 PROVIDENCE HWY	7/15/1993	UNKNOWN CHEMICAL OF TYPE - OIL, WASTE OIL	DRYWELL, LEACHFIELD	PENNFA
MAPPED	3-0003310	BIRD MACHINE OIL SPILL (MANUFACTURING)	100 NEPONSET ST	10/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UST	WCSPRM
MAPPED	3-0003356	BOSTON EDISON SUBSTATION (UTILITY)	MAIN ST	10/15/1990	UNKNOWN CHEMICAL OF UNKNOWN TYPE	TRANSFORM	RAO
MAPPED	3-0003544	MOBIL STATION 01 137	PROVIDENCE HWY HIGH PLN	4/15/1991	UNKNOWN CHEMICAL OF UNKNOWN TYPE		PENNFA

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0003574	RODMAN FORD TRUCK CENTER (REPAIR YARD)	PROVIDENCE HWY	4/15/1991	WASTE OIL	LEACHFIELD, UST	RAO
MAPPED	3-0003642	GETTY PETROLEUM (GAS STATION)	571 MAIN ST	7/15/1991	PETROLEUM BASED OIL	UST	RAO
MAPPED	3-0003645	BEACH PROPERTY (FORMER)	ELM ST	7/15/1991	PETROLEUM BASED OIL	UNKNOWN	WCSPRM
MAPPED	3-0004055	UNDEVELOPED PROPERTY (OPEN SPACE)	TURCO DR	10/15/1992	UNKNOWN CHEMICAL OF UNKNOWN TYPE	UNKNOWN	TIER1D
MAPPED	3-0004452	SUNOCO SERVICE STATION (GAS STATION)	1041 MAIN ST	9/29/1992	PETROLEUM BASED OIL	UST	TIERII
MAPPED	3-0004812	CUMBERLAND FARMS (COMMERCIAL, GAS STATION)	1185 WASHINGTON ST	10/1/1993	GASOLINE	PIPE, UST	REMOPS
MAPPED	3-0010765	WALPOLE FIRE DEPT (MUNICIPAL)	20 STONE ST	3/29/1994	UNKNOWN CHEMICAL OF UNKNOWN TYPE (15 GAL)	PIPE	RAO
MAPPED	3-0010789	NO LOCATION AID	920 MAIN ST	3/18/1994	WASTE OIL (980 PPM)	UNKNOWN	RAO
MAPPED	3-0010863	FMR GAS STATION (COMMERCIAL)	739 MAIN ST	4/14/1994	GASOLINE (140 PPMV), PETROLEUM BASED OIL, TOTAL PETROLEUM HYDROCARBONS (TPH) (50 PPM)	UST	RAONR
MAPPED	3-0011220	OFF ROUTE ONE (PARKING LOT, ROADWAY)	25 WALPOLE SOUTH PARK RD	6/30/1994	DIESEL FUEL (100 GAL), DIESEL FUEL (125 GAL)	FUELTANK	RAO
MAPPED	3-0011271	NO LOCATION AID (COMMERCIAL)	215 MAIN ST	7/12/1994	BENZENE (1783 PPMV), GASOLINE (1800 PPMV)	UST	RAO
MAPPED	3-0011445	NO LOCATION AID (ROADWAY)	OLD FISHER LN	8/10/1994	DIESEL FUEL (10 GAL), DIESEL FUEL (15 GAL)	FUELTANK, VEHICLE	RAO
MAPPED	3-0011467	FRONT OF 595WASHINGTON ST/POLE #78/24 (RESIDENTIAL, ROADWAY)	WASHINGTON ST	8/14/1994	MINERAL OIL, UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL)	TRANSFORM	RAO
MAPPED	3-0011674	NO LOCATION AID (COMMERCIAL)	985 PROVIDENCE HWY	9/30/1994	GASOLINE (15 GAL), GASOLINE (31.2 GAL)	VEHICLE	RAO
MAPPED	3-0011865	NO LOCATION AID (RESIDENTIAL)	377 NORTH ST	11/17/1994	FUEL OIL #2 (100 GAL), FUEL OIL #2 (75 GAL)	PIPE	RAO
MAPPED	3-0011931	NO LOCATION AID	1 PRODUCTION RD	12/6/1994	GASOLINE, GASOLINE (300 PPMV)	UST	RAO
MAPPED	3-0011958	NO LOCATION AID (RESIDENTIAL)	173 MYLOD ST	12/13/1994	TOTAL PETROLEUM HYDROCARBONS (TPH) (2400 PPM), TOTAL PETROLEUM HYDROCARBONS (TPH) (5570 MG/KG)	UST	RAO
MAPPED	3-0011999	POLE #91/2 @16 HARDING ST (RESIDENTIAL)	HARDING ST	12/24/1994	UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL)	TRANSFORM	RAO
MAPPED	3-0012079	REAR OF FACILITY AT LOADING DOCK (INDUSTRIAL)	333 CONEY ST	1/20/1995	DIESEL FUEL (10 GAL), DIESEL FUEL (50 GAL)	VEHICLE	RAO
MAPPED	3-0012120	MCCARTHY WAREHOUSE ROUTE 1 (COMMERCIAL)	295 UNION ST	1/31/1995	FUEL OIL #2 (100 GAL), FUEL OIL #2 (70 GAL)	TANKER	RAO

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0012636	WALPOLE HIGH SCHOOL	257 COMMON ST	6/29/1995	FUEL OIL #2 (1500 PPMV), FUEL OIL #4 (1500 PPMV)	UST	RAO
MAPPED	3-0012692	NO LOCATION AID	110 NORFOLK ST	7/14/1995	ETHANE, 1,1-DICHLORO- (1900 UG/L)	UNKNOWN	RAO
MAPPED	3-0013087	PEGGY LAWTON KITCHENS INC (COMMERCIAL)	255 WASHINGTON ST	10/26/1995	FUEL OIL #2 (10 GAL), FUEL OIL #2 (330 PPMV)	UST	RAO
MAPPED	3-0013186	TEXACO STATION RTE1/RTE 27 (COMMERCIAL)	985 PROVIDENCE HWY RTE 1	11/28/1995	OIL, UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL)	TANKER	STMRET
MAPPED	3-0013236	NO LOCATION AID	935 EAST ST	12/7/1995	TOTAL PETROLEUM HYDROCARBONS (TPH) (2.7 MG/L)	UNKNOWN	DPS
MAPPED	3-0013242	NEAR NORFOLK TOWNLINE (ROADWAY)	WINTER ST	12/10/1995	PETROLEUM BASED OIL (23 GAL)	VEHICLE	RAO
MAPPED	3-0013321	SCHOOL MEADOW AT BROOK WELLFIELD	1303 WASHINGTON ST	1/11/1996	FUEL OIL #2 (17 MG/L), FUEL OIL #2 (9200 MG/KG)	UNKNOWN	RAO
MAPPED	3-0013338	PAD MOUNTED TRANSFORMER PMH9655 (COMMERCIAL, INDUSTRIAL, ROADWAY)	412 HIGH PLAINS ST	1/17/1996	MINERAL OIL (165 GAL)	TRANSFORM	RAO
MAPPED	3-0013681	NO LOCATION AID (COMMERCIAL)	80 SOUTH ST	4/19/1996	DIESEL FUEL (30 GAL)	VEHICLE	RAO
MAPPED	3-0014101	BLESSED CHURCH	10 DIAMOND ST	8/8/1996	FUEL OIL #2 (101 PPMV), FUEL OIL #2 (101 PPMV)	UST	RAO
MAPPED	3-0014118	RTE 1 (COMMERCIAL)	985 BOSTON PROV HWY	8/13/1996	BENZENE, DIMETHYL (24000 UG/L), BENZENE, ETHYL- (990 UG/L), BENZENE, METHYL- (6000 UG/L), GASOLINE	UST	RAONR
MAPPED	3-0014220	NEXT TO 908 & 920 MAIN ST (COMMERCIAL)	935 EAST ST	9/12/1996	LUBRICATING OIL (36 INCH), UNKNOWN CHEMICAL OF UNKNOWN TYPE (45.6 INCH)	UNKNOWN	DPS
MAPPED	3-0014259	NO LOCATION AID	514 HIGH PLAIN ST	9/18/1996	BENZENE (290 PPB)	UNKNOWN	DPS
MAPPED	3-0014291	NO LOCATION AID (COMMERCIAL)	112 WASHINGTON ST	10/2/1996	UNKNOWN CHEMICAL OF TYPE - OIL (100 GAL)	MACHINERY	RAO
MAPPED	3-0014490	PIONEER FARMS GREENHOUSE (COMMERCIAL)	505 FISHER ST	11/11/1996	FUEL OIL #2 (175 GAL), FUEL OIL #2 (200 GAL)	PIPE	RAO
MAPPED	3-0014684	REAR ALLEY	943 MAIN ST	1/2/1997	FUEL OIL #2, FUEL OIL #2 (150 GAL)	AST	RAO
MAPPED	3-0014695	NO LOCATION AID (COMMERCIAL)	1340 MAIN ST	1/6/1997	GASOLINE (115 PPM), GASOLINE (115 PPMV), GASOLINE (464 PPM)	UST	REMOPS
MAPPED	3-0014755	NO LOCATION AID	506 HIGH PLAIN ST	1/23/1997	BENZENE	UNKNOWN	DPS
MAPPED	3-0014756	NO LOCATION AID	642 BOSTON PROVIDENCE HWY	1/23/1997	BENZENE	UNKNOWN	DPS
MAPPED	3-0014831	POLE 74/59 (RESIDENTIAL, RIGHT OF WAY)	FOREST AND WEST ST	2/17/1997	UNKNOWN CHEMICAL OF TYPE - OIL (10 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (25 GAL)	TRANSFORM	RAO

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0014992	NO LOCATION AID (COMMERCIAL)	1439 MAIN ST	4/11/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE, UNKNOWN CHEMICAL OF UNKNOWN TYPE (3 INCH)	FORMER, UST	RAO
MAPPED	3-0015034	RAMP (ROADWAY)	RTE 95 SOUTH @ CONEY ST	4/20/1997	GASOLINE (15 GAL)	VEHICLE	RAO
MAPPED	3-0015135	DAY CARE CENTER/SUNOCO SERVICE STATION (SCHOOL)	62 FRONT ST	5/23/1997	UNKNOWN CHEMICAL OF TYPE - OIL (7.7 MG/L), UNKNOWN CHEMICAL OF TYPE - OIL (7.73 PPM)	UST	RAONR
MAPPED	3-0015417	BECO/POLE #446/1 (INDUSTRIAL, POLE #1)	SADDLE WAY	8/14/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (25 GAL)	PIPE	RAO
MAPPED	3-0015444	POLE #451/3 (ROADWAY)	DOVER RD	8/19/1997	UNKNOWN CHEMICAL OF UNKNOWN TYPE (15 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (30 GAL)	PIPE	RAO
MAPPED	3-0015688	NO LOCATION AID	24 INDUSTRIAL RD	11/4/1997	ETHANE, 1,1,2,2-TETRACHLORO- (2 UG/L), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (5 UG/L)	UNKNOWN	RAO
MAPPED	3-0015734	MCI (STATE)	MAIN ST (RTE 1A)	11/20/1997	DIESEL FUEL, UNKNOWN CHEMICAL OF UNKNOWN TYPE (1 GAL/HR)	UST	RAO
MAPPED	3-0016685	MCI (MUNICIPAL)	CEDAR JCT	4/9/1998	GASOLINE (1980 PPMV)	UST	RAO
MAPPED	3-0016820	WEBSTER TRUCKING (COMMERCIAL)	25 INDUSTRIAL RD	5/20/1998	DIESEL FUEL (241 PPMV), DIESEL FUEL (247 PPM)	UST	RAO
MAPPED	3-0016915	NO LOCATION AID (INDUSTRIAL)	24 INDUSTRIAL RD	6/15/1998	BENZENE, METHYL- (60 GAL)	AST	RAO
MAPPED	3-0016990	SUMMER ST (RESIDENTIAL)	10 COBBLE KNOLL DR	6/29/1998	PETROLEUM BASED OIL, PETROLEUM BASED OIL (10.3 MG/L)	UNKNOWN	RAO
NOT MAPPED	3-0016994	LOTS 4 AND 5 POLE 25 WEST SIDE RTE 1	BOSTON PROVIDENCE HWY	6/30/1998	BENZENE, DIMETHYL (7.4 PPM)	UNKNOWN	DPS
MAPPED	3-0017188	STADIUM MOBIL (COMMERCIAL)	2285 PROVIDENCE HWY	8/20/1998	BENZENE, DIMETHYL (7400 PPB), BENZENE, METHYL- (9500 UG/L), UNKNOWN CHEMICAL OF UNKNOWN TYPE (4700 PPB)	UNKNOWN	TIERII
MAPPED	3-0017224	PETES DREAM (COMMERCIAL)	1065 MAIN ST	8/27/1998	FUEL OIL #2 (297 PPMV), FUEL OIL #2 (297 PPMV)	UST	TIERII
MAPPED	3-0017485	AT WILLOW ST (INDUSTRIAL)	100 NEPONSET ST	10/27/1998	CUTTING OIL (100 PPMV), OIL (100 PPMV)	UST	RAO
MAPPED	3-0017705	CORNER OF NORTON AVE AND SCHOOL ST	50 SCHOOL ST	12/7/1998	[illegible] MG/KG), 1,2-BENZENEDICARBOXYLIC ACID, BIS(2-ETHYLHEXYL) ESTER (24 UG/L), ACENAPHTHYLENE, 1,2-DIHYDRO (250 MG/KG), BENZ[A]ANTHRACENE (500 MG/KG), BENZ[E]ACEPHENANTHRYLENE (610 MG/KG), BENZO(K)FLUORANTHENE (240 MG/KG), BEN	UNKNOWN	RAO

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0017709	BECO TRANSMISSION LINE RIGHT OF WAY	NEAR 2 TAFT ST OFF RTE 1A	12/9/1998	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL, UNKNOWN CHEMICAL OF UNKNOWN TYPE, WASTE OIL	DRUMS	RAO
MAPPED	3-0017751	NO LOCATION AID (RESIDENTIAL)	291 PLIMPTON ST	12/17/1998	FUEL OIL #2, FUEL OIL #2 (8.8 GAL)	AST, PIPE, TANKER	RAO
MAPPED	3-0017796	NORTH STREET (COMMERCIAL)	745 MAIN ST	12/29/1998	GASOLINE (175 PPMV)	UST	REMOPS
MAPPED	3-0018762	BETWEEN 11 AND 15 APPLE TREE LANE (ROADWAY)	APPLE TREE LN	9/18/1999	UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL), UNKNOWN CHEMICAL OF TYPE - OIL (20 GAL)	TRANSFORM	RAO
MAPPED	3-0018831	NO LOCATION AID (ROADWAY)	331 WEST ST	10/7/1999	UNKNOWN	UNKNOWN	TIER1D
MAPPED	3-0018926	RESIDENCE	1611 WASHINGTON ST	11/3/1999	FUEL OIL #2 (100 GAL), OIL (100 GAL)	UNKNOWN	TIER1C
MAPPED	3-0019121	NO LOCATION AID	1075 MAIN ST	12/27/1999	2-METHYLNAPHTHALENE (34 UG/L), BENZENE (75 UG/L), NAPHTHALENE (185 UG/L)	UNKNOWN	TIERII
MAPPED	3-0019146	BOSTON EDISON (COMMERCIAL, ROADWAY)	740 MAIN ST AKA RTE 1A	1/8/2000	UNKNOWN CHEMICAL OF TYPE - OIL (70 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (75 GAL)	REGULATOR, VOLTAGE	RAO
MAPPED	3-0019565	NO LOCATION AID (COMMERCIAL)	1075 MAIN ST	5/23/2000	UNKNOWN CHEMICAL OF UNKNOWN TYPE, UNKNOWN CHEMICAL OF UNKNOWN TYPE (.5 INCH)	UST	RAONR
MAPPED	3-0019617	RTE 1A (COMMERCIAL)	1333 MAIN ST	6/12/2000	FUEL OIL #2, FUEL OIL #2 (3600 INCH)	UST	RAO
MAPPED	3-0019692	NO LOCATION AID (COMMERCIAL)	333 CONEY ST	7/6/2000	DIESEL FUEL (45 GAL), UNKNOWN CHEMICAL OF UNKNOWN TYPE (20 GAL)	VEHICLE	RAO
MAPPED	3-0019859	RTE 1 AND PINE ST	WALPOLE PARK S	8/9/2000	METHANE, BROMODICHLORO- (6 UG/L), METHANE, TRICHLORO- (9 UG/L)	UNKNOWN	RAO
MAPPED	3-0019925	NO LOCATION AID	541 THRU 571 MAIN ST	9/1/2000	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (6.1 UG/L)	UNKNOWN	RAO
NOT MAPPED	3-0020110	EXIT 10 (ROADWAY)	RTE 95 N	11/13/2000	UNKNOWN CHEMICAL OF TYPE - OIL, UNKNOWN CHEMICAL OF TYPE - OIL, UNKNOWN CHEMICAL OF UNKNOWN TYPE (1 GAL)	VEHICLE	RAO
MAPPED	3-0020444	NO LOCATION AID (ROADWAY)	900 WASHINGTON ST	3/5/2001	UNKNOWN CHEMICAL OF UNKNOWN TYPE (50 GAL)	VEHICLE	RAO
NOT MAPPED	3-0020549	NEAR PINE ST (COMMERCIAL)	VACANT PARCEL 54-21 OFF RTE 1	3/30/2001	BENZ[A]ANTHRACENE (1.09 MG/KG), BENZ[E]ACEPHENANTHRYLENE (1.43 MG/KG), BENZO[A]PYRENE (.99 MG/KG), DIBENZ[A,H]ANTHRACENE (.888 MG/KG), INDENO(1,2,3-CD)PYRENE (.888 MG/KG), LEAD (730 MG/KG), LEAD (730 PPM)	UNKNOWN	RAO

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0020605	NO LOCATION AID (COMMERCIAL)	2285 PROVIDENCE HWY	4/19/2001	GASOLINE (.6 INCH), UNKNOWN CHEMICAL OF UNKNOWN TYPE (.1 INCH)	UNKNOWN	RAONR
MAPPED	3-0020712	NO LOCATION AID (COMMERCIAL)	1340 MAIN ST	5/16/2001	GASOLINE (30 GAL), GASOLINE (40 GAL)	PIPE	RAONR
MAPPED	3-0020901	MCI CEDAR JUNCTION (STATE)	MAIN ST	7/12/2001	OIL, OIL (30 PPM)	UNKNOWN	RAO
MAPPED	3-0021359	NO LOCATION AID (COMMERCIAL)	920 MAIN ST	12/28/2001	BENZENE (63.6 UG/L), BENZENE, DIMETHYL (12330 UG/L), BENZENE, ETHYL- (700 UG/L), BENZENE, METHYL- (3620 UG/L), BTEX (5 MG/L), NAPHTHALENE (515 UG/L), UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (149 UG/L)	UNKNOWN	RAONR
NOT MAPPED	3-0021454	NEAR NO 1303 (COMMERCIAL, RESIDENTIAL, ROADWAY)	WASHINGTON ST	2/1/2002	UNKNOWN CHEMICAL OF UNKNOWN TYPE (10 GAL), WASTE OIL (.25 GAL)	HYDRAULIC, PIPE, VEHICLE	RAO
MAPPED	3-0021458	BEHIND BUILDINGS	961 MAIN ST	2/4/2002	NAPHTHALENE (7.3 MG/KG)	UNKNOWN	RAO
MAPPED	3-0021561	SHELL STATION 137870	920 MAIN ST	10/10/2001	2-METHYLNAPHTHALENE (5.35 MG/KG), BENZ[A]ANTHRACENE (3.53 MG/KG), CHRYSENE (16.2 MG/KG)	UNKNOWN	RAONR
MAPPED	3-0021915	ROUTE 1 AND PINE ST	15 WALPOLE PARK S	7/2/2002	LEAD (.046 MG/L)	UNKNOWN	TIER1B
NOT MAPPED	3-0022095	UTILITY POLE 4/9DA STATION 447 (RIGHT OF WAY, UTILITY)	MAIN ST NEAR 1491	9/12/2002	MINERAL OIL, MINERAL OIL (30 GAL)	TRANSFORM	RAO
MAPPED	3-0022209	NO LOCATION AID (COMMERCIAL)	743 MAIN ST	10/15/2002	FUEL OIL #2 (175 GAL), FUEL OIL #2 (200 GAL), FUEL OIL #2 (275 GAL)	AST	RAONR
MAPPED	3-0022307	NO LOCATION AID (COMMERCIAL, INDUSTRIAL)	18 RENMER AVE	11/14/2002	GASOLINE (20 GAL)	VEHICLE	RAO
MAPPED	3-0022399	WALPOLE MALL PARKING LOT (COMMERCIAL)	70-90 PROVIDENCE HWY	12/16/2002	DIESEL FUEL (20 GAL), DIESEL FUEL (5 GAL)	FUEL TANK, VEHICLE	RAO
MAPPED	3-0022577	VACANT LAND	1425 MAIN ST	2/19/2003	ETHENE, TETRACHLORO- (12.9 UG/L)	UNKNOWN	DPS
MAPPED	3-0022935	BIRD MACHINE CO (INDUSTRIAL)	100 NEPONSET ST	6/17/2003	PETROLEUM BASED OIL, UNKNOWN CHEMICAL OF TYPE - OIL	UNKNOWN	RAO
MAPPED	3-0023506	NSTAR SERVICE CENTER (COMMERCIAL)	760 MAIN ST	1/14/2004	GASOLINE (.05 GAL/HR), GASOLINE (5 GAL)	PIPE, UST	RAO
MAPPED	3-0023513	BIRD MACHINE	100 NEPONSET ST	1/14/2004	LEAD (4500 MG/KG)	UNKNOWN	TIERII
MAPPED	3-0023575	BIRD MACHINE CO (COMMERCIAL)	100 NEPONSET ST	1/23/2004	OIL	UNKNOWN	TIERII
MAPPED	3-0023589	HOLLINGSWORTH AND VOSE COMPANY	112 WASHINGTON ST	1/20/2004	CHROMIUM (2110 MG/KG), COPPER (2540 MG/KG), LEAD (372 MG/KG)	UNKNOWN	RAO
MAPPED	3-0023619	BOYDEN SCHOOL	1852 WASHINGTON ST	2/20/2004	GAL)	TANKER	RAO
NOT MAPPED	3-0023787	BEACH PROPERTY KENDALL COMPANY	ELM ST	4/21/2004	BENZ[A]ANTHRACENE (31.9 MG/KG), BENZ[E]ACEPHENANTHRYLENE (24.3 MG/KG)	UNKNOWN	TIER1D

Table 1 (Continued)

MAPPED/ NOT MAPPED	SPILL ID/RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCE	STATUS
MAPPED	3-0024105	BIRD MACHINE COMPANY-DEMOLITION DEBRIS	100 NEPONSET ST	7/30/2004	LEAD (.0331 MG/L), NICKEL (1.426 MG/L), UNKNOWN CHEMICAL OF UNKNOWN TYPE (.00000021 MG/KG), ZINC (1.218 MG/L)	UNKNOWN	UNCLSS
MAPPED	3-0020901	MCI CEDAR JUNCTION (STATE)	MAIN ST	7/12/2001	OIL, OIL (30 PPM)	UNKNOWN	RAO
MAPPED	3-0024107	SHELL SERVICE STATION #137870	920 MAIN ST	8/2/2004	UNKNOWN CHEMICAL OF TYPE - HAZARDOUS MATERIAL (.911 UG/G)	UNKNOWN	RAONR
MAPPED	3-0024222	BAKER HUGHES INC FMLY BIRD MACHINE CO	100 NEPONSET ST	9/8/2004	NICKEL (.14 MG/L)	UNKNOWN	UNCLSS
MAPPED	3-0024400	ROUTE 1 (COMMERCIAL)	985 BOSTON PROVIDENCE HWY	11/10/2004	GASOLINE (100 PPMV), UNKNOWN CHEMICAL OF UNKNOWN TYPE (100 PPMV)	UST	TIERII
MAPPED	3-0024474	CALLAHAN COMPANY 42-07-15N 71-15-57W (INDUSTRIAL)	18 INDUSTRIAL RD	12/8/2004	2-PROPANONE (1300 GAL), 2-PROPANONE (1300 LBS)	PIPE, TANKER	UNCLSS
MAPPED	3-0024498	BIRD PARK (WATERBODY)	RHOADES AVE	12/17/2004	PETROLEUM BASED OIL	UNKNOWN	UNCLSS
MAPPED	3-0024542	SUNOCO SERVICE STATION	1041 MAIN ST	1/4/2005	UNKNOWN	UNKNOWN	UNCLSS
MAPPED	3-0024565	CALLAHAN CO (INDUSTRIAL)	18 INDUSTRIAL RD	1/14/2005	2-PROPANONE (200 GAL)	UNKNOWN	RAO
MAPPED	3-0024608	CORNER OF JUNE STREET (RESIDENTIAL)	259-261 WASHINGTON ST	2/1/2005	FUEL OIL #2	PIPE	UNCLSS
MAPPED	3-0024703	NO LOCATION AID (RESIDENTIAL)	141 LINCOLN RD	3/16/2005	GAL)	AST, PIPE	UNCLSS
MAPPED	3-0024778	CALLAHAN COMPANY	18 INDUSTRIAL RD	4/6/2005	2-BUTANONE (17900 UG/L), ETHENE , 1,2-DICHLORO- (1370 UG/L), ETHENE, CHLORO- (184 UG/L)	UNKNOWN	UNCLSS
MAPPED	3-0024804	MOBIL STATION 01-137 (COMMERCIAL)	980 PROVIDENCE HWY	4/26/2005	UNKNOWN CHEMICAL OF UNKNOWN TYPE (400 GAL)	PIPE	UNCLSS

Source: Massachusetts Deparment of Environmental Protection (MDEP) Bureau of Waste Site Cleanup. 2004. Downloadable Site Lists. http://www.state.ma.us/dep/bwsc/sites/sdown.htm

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

Table 2
Maximum concentrations of contaminants detected in on-site groundwater samples at the Bird Landfill that exceeded comparison values (samples taken from 1984 - 2005)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Arsenic	Nov-94	LF-UP	189	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3 Chronic EMEG (Adult), RMEG (adult) = 10 U.S. EPA MCL = 10
Barium	Nov-94	LF-UP	986	RMEG (child) = 700 RMEG (adult) = 2000 MDEP MMCL = 2000
Benzene	Nov-04	CSA-3	21	CREG = 0.6 MDEP MMCL = 5
Cadmium	Jun-02	CSA-2D	16.4	Chronic EMEG (child) = 2 Chronic EMEG (adult) = 7 MDEP MMCL = 5
Chloroethane	May-87	LF-3A	250	EPA RBC = 3.6
Chromium (total)	Nov-94	LF-UP	506	Hexavalent Chromium: RMEG (child) = 30 Hexavalent Chromium: RMEG (adult) = 300 MDEP MMCL = 100
Chromium (VI)	May-99	CSA-7S	580	Hexavalent Chromium: RMEG (child) = 30 Hexavalent Chromium: RMEG (adult) = 300 MDEP MMCL = 100
Copper	Nov-04	CSA-6S	818	Intermediate EMEG (child) = 100 Intermediate EMEG (adult) = 400 MDEP MMCL (Action Level) = 1300
1,2-Dichloroethylene, cis-	Nov-94	LF-3S	110	Intermediate EMEG (child) = 3,000 Intermediate EMEG (adult) = 10,000 EPA RBC = 61 MDEP MMCL = 70
Iron	Nov-94	LF-3D	898,000	EPA RBC = 11,000
Lead	Nov-94	LF-UP	230	MCLG = 0 MDEP MMCL = 15

Table # 2 (Continued)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Manganese	May-92	LF-1	26,000	RMEG (child) = 500 RMEG (adult) = 2000 EPA RBC = 730
Silver	May-97	LF-1	250	RMEG (child) = 50 RMEG (adult) = 200 EPA RBC = 180
Tetrachlorethylene	Nov-99	LF-6D	41	RMEG (child) = 100 RMEG (adult) = 400 EPA RBC (residential) = 1.2 MDEP MMCL = 5
Trichloroethylene	Nov-99	LF-6D	34	EPA RBC (residential) = 1.6 MCLG = 0 MDEP MMCL = 5
Vinyl Chloride	Nov-95	LF-3S	24	CREG = 0.03 Chronic EMEG (child) = 0.2 Chronic EMEG (adult) = 0.7 MCLG = 0 MDEP MMCL = 2

Data sources:

GZA GeoEnvironmental, Inc. 1997. Bird Landfill: Draft Comprehensive Site Assessment.

GZA GeoEnvironmental, Inc. 1998. Addendum to the Comprehensive Site Assessment (CSA), Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2000. 2000 Semi-Annual Groundwater Monitoring Report, Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2001. 2001 Semi-Annual Groundwater Monitoring Report. Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2002. 2002 Semi-Annual Groundwater Monitoring Report, Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2005. Semi-Annual Groundwater Monitoring Report (Spring 2005), Bird Inc. Landfill.

Comparison values (source organization, reference):

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

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

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

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 2005b)

Table # 2 (Continued)

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 2005b)

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

MCLG = Maximum Contaminant Level Goal for drinking water (U.S. EPA, ATSDR 2005b)

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

Table 3
Maximum concentrations of contaminants detected in on-site surface water samples at the Bird Landfill that exceeded comparison values (samples taken from May 1996 - November 2005)

Contaminant	Date of sample	Descriptive location of sample	Maximum concentration (ppb)	Drinking water comparison value (ppb)
Arsenic	May-96	SW-5	6	CREG = 0.02 Chronic EMEG (child); RMEG (child) = 3 Chronic EMEG (Adult), RMEG (adult) = 10 U. S. EPA MCL = 10
Barium	May-96	SW-1	1080	RMEG (child) = 700 RMEG (adult) = 2000 MDEP MMCL = 2000
Benzene	May-96	SW-1	0.97	CREG = 0.6 MDEP MMCL = 5
Cadmium	May-99	SW-4	8.6	Chronic EMEG (child) = 2 Chronic EMEG (adult) = 7 MDEP MMCL = 5
Iron	May-96	SW-1	52,000	EPA RBC = 11,000
Lead	May-96	SW-4	85	MCLG = 0 MDEP MMCL = 15
Manganese	May-96	SW-1	1,530	RMEG (child) = 500 RMEG (adult) = 2000
Silver	May-96	SW-4	85	RMEG (child) = 50 RMEG (adult) = 200

Data sources:

GZA GeoEnvironmental, Inc. 1997. Bird Landfill: Draft Comprehensive Site Assessment.

GZA GeoEnvironmental, Inc. 1998. Addendum to the Comprehensive Site Assessment (CSA), Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2000. 2000 Semi-Annual Groundwater Monitoring Report, Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2001. 2001 Semi-Annual Groundwater Monitoring Report. Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2002. 2002 Semi-Annual Groundwater Monitoring Report, Bird, Inc. Landfill.

GZA GeoEnvironmental, Inc. 2005. Semi-Annual Groundwater Monitoring Report (Spring 2005), Bird Inc. Landfill.

Comparison values (source organization, reference):

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

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

Table #3 (Continued)

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

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 2005b)

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 2005b)

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

MCLG = Maximum Contaminant Level Goal for drinking water (U.S. EPA, ATSDR 2005b)

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

Table 4
Summary of Possible Exposure Pathways for the Bird Landfill
Walpole, Massachusetts

Environmental Medium	Exposure Pathway	Contaminant(s)	Point of Exposure	Route of Exposure	Receptor Population	Time Frame	Type of Pathway	Notes
Groundwater	Groundwater contamination	Metals, VOCs, PAHs	Off-site municipal/ private wells	Ingestion, Inhalation	Residents	Past, Present	Eliminated	Municipal water tested and treated. One private well not used, other private well has a filter and is located outside estimated extent of contaminated groundwater.
	Groundwater contamination	Metals, VOCs, PAHs	Off-site municipal/ private wells	Ingestion, Inhalation	Residents	Future	Potential	Exposures are possible if new private wells are installed down gradient of the landfill. Potential impacts to municipal wells are unlikely.
	Volatilization of shallow groundwater to indoor air	VOCs (e.g. Vinyl Chloride)	Off-site residences with basements	Inhalation	Residents	Past, Present, Future	Potential	Off-site concentrations of VOCs in groundwater are expected to be below a level of health concern for possible indoor air exposure in down-gradient homes.
Surface Water	Surface water	VOCs, Metals	Stream, pond, wetland	Ingestion	Trespassers	Past, Present, Future	Potential	Concentrations detected in surface water are unlikely to result in adverse health effects for recreational exposure scenario.
Soil/Sediment	Soil/sediment	Metals	Stream, pond, wetland	Ingestion	Trespassers	Past, Present, Future	Potential	Concentrations detected in wetland soils and sediment are below comparison values.

TABLE 6a
Bladder Cancer Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	31	26.2	119	81 -- 168	24	19.8	121	77 -- 180	7	6.3	111	44 -- 228
4112	22	18.2	121	76 -- 183	15	13.3	113	63 -- 186	7	4.9	143	57 -- 296
4113	19	21.3	89	54 -- 139	10	15.8	63	30 -- 116	9	5.5	164	75 -- 311
Town Total[†]	73	65.5	112	87 -- 140	50	48.8	102	76 -- 135	23	16.6	138	88 -- 207

[†] One case for which census tract designation was not possible was included in the town total.

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

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

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

TABLE 6b
Bladder Cancer Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	10	7.2	139	67 -- 256	8	5.4	148	64 -- 292	2	1.8	NC	NC -- NC
4112	5	6.0	84	27 -- 196	5	4.4	114	37 -- 265	0	1.6	NC	NC -- NC
4113	2	5.6	NC	NC -- NC	1	4.1	NC	NC -- NC	1	1.4	NC	NC -- NC
Town Total[†]	18	18.7	96	57 -- 152	15	13.9	108	60 -- 178	3	4.8	NC	NC -- NC

[†] One case for which census tract designation was not possible was included in the town total.

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

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

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

TABLE 6c
Bladder Cancer Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	10	8.1	123	59 -- 227	6	6.2	97	36 -- 212	4	2.0	NC	NC -- NC
4112	6	5.7	105	38 -- 229	4	4.2	NC	NC -- NC	2	1.5	NC	NC -- NC
4113	10	6.2	161	77 -- 296	4	4.6	NC	NC -- NC	6	1.6	382	* 139 -- 831
Town Total	26	19.9	130	85 -- 191	14	14.9	94	51 -- 158	12	5.0	239	* 124 -- 418

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

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

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

TABLE 6d
Bladder Cancer Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	11	9.7	114	57 -- 203	10	7.2	138	66 -- 254	1	2.4	NC	NC -- NC
4112	11	6.8	163	81 -- 291	6	4.9	121	44 -- 264	5	1.8	274	88 -- 640
4113	7	10.9	64	26 -- 132	5	7.9	64	20 -- 148	2	3.0	NC	NC -- NC
Town Total	29	27.3	106	71 -- 153	21	20.0	105	65 -- 160	8	7.3	110	47 -- 216

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

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

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

TABLE 7a
Hodgkin's Disease Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	5	5.4	92	30 -- 214	2	3.4	NC	NC -- NC	3	2.0	NC	NC -- NC
4112	6	4.0	150	55 -- 327	3	2.2	NC	NC -- NC	3	1.8	NC	NC -- NC
4113	8	5.0	161	69 -- 317	3	2.8	NC	NC -- NC	5	2.1	233	75 -- 543
Town Total	19	14.2	134	81 -- 209	8	8.2	98	42 -- 192	11	6.0	184	92 -- 330

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

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

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

TABLE 7b
Hodgkin's Disease Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	0	1.5	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.6	NC	NC -- NC
4112	1	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
4113	2	1.5	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.6	NC	NC -- NC
Town Total	3	4.2	NC	NC -- NC	2	2.5	NC	NC -- NC	1	1.7	NC	NC -- NC

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

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

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

TABLE 7c
Hodgkin's Disease Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	4	1.8	NC	NC -- NC	1	1.1	NC	NC -- NC	3	0.7	NC	NC -- NC
4112	2	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC
4113	3	1.6	NC	NC -- NC	1	0.9	NC	NC -- NC	2	0.7	NC	NC -- NC
Town Total	9	4.6	194	89 -- 368	3	2.6	NC	NC -- NC	6	2.0	301	* 110 -- 656

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

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

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

TABLE 7d
Hodgkin's Disease Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	1	1.8	NC	NC -- NC	1	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC
4112	3	1.4	NC	NC -- NC	1	0.8	NC	NC -- NC	2	0.6	NC	NC -- NC
4113	3	2.1	NC	NC -- NC	1	1.3	NC	NC -- NC	2	0.9	NC	NC -- NC
Town Total	7	5.3	131	53 -- 270	3	3.1	NC	NC -- NC	4	2.3	NC	NC -- NC

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

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

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

TABLE 8a
Kidney Cancer Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	21	15.8	133	82 -- 203	10	10.1	99	47 -- 182	11	5.7	191	95 -- 343
4112	12	11.6	103	53 -- 180	7	7.2	98	39 -- 202	5	4.5	111	36 -- 260
4113	15	14.1	106	59 -- 175	6	8.9	67	24 -- 146	9	5.2	173	79 -- 329
Town Total	48	41.5	116	85 -- 153	23	26.2	88	56 -- 132	25	15.2	164	* 106 -- 242

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

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

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

TABLE 8b
Kidney Cancer Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	6	3.3	182	66 -- 396	2	2.0	NC	NC -- NC	4	1.3	NC	NC -- NC
4112	2	2.7	NC	NC -- NC	2	1.6	NC	NC -- NC	0	1.1	NC	NC -- NC
4113	3	2.7	NC	NC -- NC	2	1.7	NC	NC -- NC	1	1.0	NC	NC -- NC
Town Total	11	8.7	126	63 -- 225	6	5.4	111	41 -- 243	5	3.4	149	48 -- 347

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

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

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

TABLE 8c
Kidney Cancer Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	9	5.2	173	79 -- 329	6	3.3	179	65 -- 390	3	1.8	NC	NC -- NC
4112	1	3.8	NC	NC -- NC	0	2.4	NC	NC -- NC	1	1.5	NC	NC -- NC
4113	9	4.4	204	93 -- 387	3	2.8	NC	NC -- NC	6	1.6	381	* 139 -- 830
Town Total	19	13.4	142	85 -- 222	9	8.6	105	48 -- 199	10	4.8	208	100 -- 382

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

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

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

TABLE 8d
Kidney Cancer Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	6	6.9	87	32 -- 189	2	4.4	NC	NC -- NC	4	2.5	NC	NC -- NC
4112	9	5.1	176	80 -- 334	5	3.2	158	51 -- 369	4	2.0	NC	NC -- NC
4113	3	7.9	NC	NC -- NC	1	4.9	NC	NC -- NC	2	3.0	NC	NC -- NC
Town Total	18	19.9	90	54 -- 143	8	12.5	64	28 -- 127	10	7.4	134	64 -- 247

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

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

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

TABLE 9a
Leukemia Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	12	13.6	88	45 -- 154	6	8.2	73	27 -- 159	6	5.4	111	40 -- 241
4112	12	9.9	122	63 -- 212	4	5.6	NC	NC -- NC	8	4.3	186	80 -- 367
4113	16	12.0	134	76 -- 217	10	6.9	145	69 -- 267	6	5.1	118	43 -- 257
Town Total	40	35.3	113	81 -- 154	20	20.5	97	59 -- 150	20	14.7	136	83 -- 209

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

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

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

TABLE 9b
Leukemia Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	2	3.3	NC	NC -- NC	1	2.0	NC	NC -- NC	1	1.3	NC	NC -- NC
4112	3	2.7	NC	NC -- NC	3	1.5	NC	NC -- NC	0	1.2	NC	NC -- NC
4113	1	2.7	NC	NC -- NC	1	1.6	NC	NC -- NC	0	1.1	NC	NC -- NC
Town Total	6	8.7	69	25 -- 150	5	5.1	98	32 -- 230	1	3.6	NC	NC -- NC

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

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

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

TABLE 9c
Leukemia Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	4	3.8	NC	NC -- NC	4	2.3	NC	NC -- NC	0	1.5	NC	NC -- NC
4112	5	2.8	180	58 -- 420	1	1.6	NC	NC -- NC	4	1.2	NC	NC -- NC
4113	5	3.2	155	50 -- 362	4	1.9	NC	NC -- NC	1	1.3	NC	NC -- NC
Town Total	14	9.8	143	78 -- 241	9	5.8	155	71 -- 293	5	3.9	127	41 -- 296

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

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

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

TABLE 9d
Leukemia Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	6	6.1	98	36 -- 214	1	3.5	NC	NC -- NC	5	2.6	195	63 -- 454
4112	4	4.5	NC	NC -- NC	0	2.5	NC	NC -- NC	4	2.0	NC	NC -- NC
4113	10	7.1	141	67 -- 259	5	3.9	128	41 -- 298	5	3.2	156	50 -- 365
Town Total	20	17.7	113	69 -- 175	6	9.9	60	22 -- 132	14	7.8	181	99 -- 303

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

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

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

TABLE 10a
Liver Cancer Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	3	3.9	NC	NC -- NC	2	2.9	NC	NC -- NC	1	1.0	NC	NC -- NC
4112	3	2.8	NC	NC -- NC	3	2.1	NC	NC -- NC	0	0.7	NC	NC -- NC
4113	1	3.4	NC	NC -- NC	1	2.6	NC	NC -- NC	0	0.9	NC	NC -- NC
Town Total	7	10.1	69	28 -- 143	6	7.6	79	29 -- 173	1	2.5	NC	NC -- NC

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

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

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

TABLE 10b
Liver Cancer Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
4112	1	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
4113	0	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	1	1.9	NC	NC -- NC	1	1.3	NC	NC -- NC	0	0.5	NC	NC -- NC

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

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

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

TABLE 10c
Liver Cancer Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	1	1.1	NC	NC -- NC	0	0.8	NC	NC -- NC	1	0.3	NC	NC -- NC
4112	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
4113	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	2	2.8	NC	NC -- NC	1	2.1	NC	NC -- NC	1	0.7	NC	NC -- NC

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

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

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

TABLE 10d
Liver Cancer Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	2	2.0	NC	NC -- NC	2	1.5	NC	NC -- NC	0	0.5	NC	NC -- NC
4112	2	1.5	NC	NC -- NC	2	1.1	NC	NC -- NC	0	0.4	NC	NC -- NC
4113	0	2.3	NC	NC -- NC	0	1.7	NC	NC -- NC	0	0.6	NC	NC -- NC
Town Total	4	5.7	NC	NC -- NC	4	4.3	NC	NC -- NC	0	1.4	NC	NC -- NC

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

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

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

TABLE 11a
Lung & Bronchus Cancer Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	99	99.0	100	81 -- 122	58	60.4	96	73 -- 124	41	38.7	106	76 -- 144
4112	69	73.8	94	73 -- 118	41	43.6	94	67 -- 128	28	30.2	93	62 -- 134
4113	81	86.7	93	74 -- 116	48	52.5	91	67 -- 121	33	34.2	97	66 -- 136
Town Total	249	258.6	96	85 -- 109	147	157.0	94	79 -- 110	102	101.6	100	82 -- 122

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

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

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

TABLE 11b
Lung & Bronchus Cancer Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	30	24.4	123	83 -- 175	22	16.0	137	86 -- 208	8	8.4	95	41 -- 188
4112	21	20.4	103	64 -- 157	11	13.2	83	42 -- 149	10	7.2	138	66 -- 254
4113	21	20.0	105	65 -- 161	12	12.8	94	48 -- 163	9	7.1	126	58 -- 239
Town Total	72	65.0	111	87 -- 140	45	42.3	106	78 -- 142	27	22.6	119	79 -- 174

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

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

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

TABLE 11c
Lung & Bronchus Cancer Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	33	30.9	107	73 -- 150	18	19.0	95	56 -- 150	15	11.9	126	70 -- 207
4112	12	23.4	51	* 27 -- 90	7	14.0	50	20 -- 103	5	9.3	54	17 -- 125
4113	19	26.1	73	44 -- 114	10	16.0	62	30 -- 115	9	10.0	90	41 -- 170
Town Total	64	80.1	80	62 -- 102	35	49.3	71	* 49 -- 99	29	30.8	94	63 -- 135

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

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

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

TABLE 11d
Lung & Bronchus Cancer Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	36	40.9	88	62 -- 122	18	23.2	78	46 -- 123	18	17.8	101	60 -- 160
4112	36	30.0	120	84 -- 166	23	16.5	139	88 -- 209	13	13.5	97	51 -- 165
4113	41	45.3	90	65 -- 123	26	25.3	103	67 -- 150	15	20.0	75	42 -- 124
Town Total	113	116.1	97	80 -- 117	67	65.1	103	80 -- 131	46	51.0	90	66 -- 120

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

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

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

TABLE 12a
Mesothelioma Incidence
Walpole, Massachusetts
1982–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	2	2.0	NC	NC -- NC	2	1.7	NC	NC -- NC	0	0.3	NC	NC -- NC
4112	3	1.4	NC	NC -- NC	1	1.1	NC	NC -- NC	2	0.3	NC	NC -- NC
4113	1	1.6	NC	NC -- NC	1	1.3	NC	NC -- NC	0	0.3	NC	NC -- NC
Town Total	6	5.1	119	43 -- 259	4	4.2	NC	NC -- NC	2	0.9	NC	NC -- NC

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

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

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

TABLE 12b
Mesothelioma Incidence
Walpole, Massachusetts
1982–1987

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
4112	1	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	1	0.1	NC	NC -- NC
4113	0	0.3	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
Town Total	1	1.1	NC	NC -- NC	0	0.9	NC	NC -- NC	1	0.2	NC	NC -- NC

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

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

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

TABLE 12c
Mesothelioma Incidence
Walpole, Massachusetts
1988-1993

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	1	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.1	NC	NC -- NC
4112	1	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.1	NC	NC -- NC
4113	1	0.5	NC	NC -- NC	1	0.4	NC	NC -- NC	0	0.1	NC	NC -- NC
Town Total	3	1.6	NC	NC -- NC	2	1.3	NC	NC -- NC	1	0.3	NC	NC -- NC

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

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

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

TABLE 12d
Mesothelioma Incidence
Walpole, Massachusetts
1994–2000

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
4111	1	0.9	NC	NC -- NC	1	0.8	NC	NC -- NC	0	0.1	NC	NC -- NC
4112	1	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.1	NC	NC -- NC
4113	0	1.0	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.2	NC	NC -- NC
Town Total	2	2.5	NC	NC -- NC	2	2.1	NC	NC -- NC	0	0.4	NC	NC -- NC

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

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

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

APPENDIX A

Cancer Incidence Coding Definitions

Appendix A:
Coding Definitions of Cancer Site/Type*

Cancer Site / Type	*ICD-O-1 and Other Pre-ICD-O-2 Codes*		*ICD-O-2 Codes*		*ICD-O-3 Codes*	
	Site code	***Histology code***	***Site code***	***Histology code***	***Site code***	***Histology code***
Bladder	188.0-188.9	except 9590-9980	C67.0-C67.9	except 9590-9989	C67.0-C67.9	except 9590-9989
Hodgkin's Disease	140.0-199.9	includes O9650-O9667, P9653-P9683, B9653-B9658	C00.00-C80.9	includes 9650-9667	C00.00-C80.9	includes 9650-9667
Kidney & Renal Pelvis	189.0, 189.1	except 9590-9980	C64.9, C65.9	except 9590-9989	C64.9, C65.9	except 9590-9989
Leukemia	140.0-199.9	includes O9800-O9943, O9951, P9803-P9943, B9803-B9943	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9800-9822, 9824-9826, 9828-9941 2. includes 9823, 9827	1. C00.0-C80.9 AND 2. C42.0, C42.1, C42.4	1. includes 9733, 9742, 9800-9820, 9826, 9831-9948, 9963-9964 2. includes 9823, 9827
Liver	155.0	except 9590-9980	C22.0	except 9590-9989	C22.0	except 9590-9989
Lung & Bronchus	162.2-162.9	except 9050-9053, 9590-9980	C34.0-C34.9	except 9590-9989	C34.0-C34.9	except 9590-9989
Mesothelioma	140.0-199.9	includes O9050-O9053, P9051, P9053	C00.0-C80.9	includes 9050-9053	C00.0-C80.9	includes 9050-9055

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

APPENDIX B

Risk Factor Information for Selected Cancer Types

Bladder Cancer

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

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

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

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

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

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

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Hodgkin's Disease

Hodgkin's disease (or Hodgkin's lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin's lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,350 new cases of this disease in the U.S. in 2005, accounting for less than 1% of all cancer types, and approximately 1,410 deaths (ACS, 2005). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS, 1999).

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

No major risk factors for Hodgkin's disease have been found (ACS, 1999). However, the clinical and cellular features of Hodgkin's disease suggest a chronic infectious process (Mueller, 1996). The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin's disease may be involved for each group. Researchers have proposed that among young adults, Hodgkin's disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is probably similar to those of other lymphomas (Mueller, 1996). The virus that has been linked most specifically to this disease is the Epstein-Barr virus (EBV). EBV, a herpesvirus, is common in the general population and causes mononucleosis or "mono." Approximately 40% to 50% of Hodgkin's disease cases are associated with EBV (Weiss, 2000). In addition, several studies have also shown that young adults who have developed infectious mononucleosis have a significantly higher risk of developing Hodgkin's disease (ACS, 1999). However, the absence of EBV infection in about half the cases and the high prevalence of EBV in the general population suggest that EBV may be only one of several factors in the development of this cancer. Although cytomegalovirus (CMV) and the more recently identified human herpesvirus type 6 have been considered as possible factors in the development of Hodgkin's disease, results of antibody studies are inconsistent and these viruses do not appear to be related to risk of Hodgkin's disease (Mueller, 1996).

Slightly higher rates of Hodgkin's disease occur among people with reduced immunity, such as those with AIDS, people with congenital immune deficiencies, and individuals on immunosuppressant medication following organ transplants. However, Hodgkin's disease occurs at a much lower rate than non-Hodgkin's lymphomas among this group of individuals (ACS, 1999).

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

Hodgkin's disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin's disease is associated with an infectious agent (Mueller, 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin's disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller, 1996). Based on an examination of medical and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin's disease does not seem to be caused by genetic, lifestyle (e.g., dietary), or environmental factors (ACS, 1999).

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Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

Kidney Cancer

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

Since 1970, 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, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 30% to 100% (ACS, 2001). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of those in women may be caused by cigarette smoking (ACS, 2001).

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

(and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (ACS, 2001).

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

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

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

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Leukemia

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

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

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

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell, 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2000b). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

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

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

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

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

development of chronic leukemia remains controversial (ACS, 1999). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al., 1997).

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

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Liver Cancer

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

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

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

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan, 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

(London and McGlynn, 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands, 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan, 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS, 2001; London and McGlynn, 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS, 2001). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS, 2001; ATSDR, 2001).

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

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Lung Cancer

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

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

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

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS, 2000). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (ACS, 2000; Samet and Eradze, 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2005.

Fraumeni, 1996; Pohlablen et al., 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS, 2000; Blot and Fraumeni, 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al., 2001; Steenland and Sanderson, 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni, 1996).

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

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

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

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

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Mesothelioma

Mesothelioma is a malignant (cancerous) tumor arising in the mesothelial cells of the pleura, peritoneum or pericardium which are tissues lining the chest cavity, abdominal cavity, and cavity around the heart, respectively. Seventy-five percent of mesotheliomas start in the pleura (membranes surrounding the lungs), while 10%-20% start in the peritoneum (membranes of the abdomen). Very rarely, mesothelioma occurs in the pericardium (membranes surrounding the heart) (ACS, 2004). In the United States, an estimated 2,000 to 3,000 new cases of mesothelioma are diagnosed every year. While the incidence of mesothelioma increased steeply in the United States during the 1970's to the mid-1990's, more recent data indicate that incidence rates appear to have stabilized and may now be decreasing (ACS, 2004; Price and Ware, 2004). Men are five times more likely to develop mesothelioma than women and the disease is more common in whites than blacks (ACS, 2004). The latency period (i.e., the interval between first exposure to a disease-causing agent and the appearance of symptoms of the disease [Last 1995]) ranges from 20 to 50 years and most individuals diagnosed with mesothelioma are over 65 years of age (ACS, 2004).

Asbestos exposure (via inhalation or ingestion) is the most well-established risk factor associated with mesothelioma and occupational exposure to asbestos accounts for most cases of this disease (ACS, 2006). In case-control studies, up to 75% of individuals with mesothelioma had been exposed to asbestos (Antman et al., 1997). Asbestos is a naturally occurring fibrous mineral that has been used historically as an insulating material (ACS, 2006). The carcinogenic effect of these fibers is believed to be related to the physical properties of the fibers rather than the chemical make-up. Therefore, this type of cancer may be caused by the physical irritation of the cells by asbestos fibers (ACS, 2004). Also, the risk of mesothelioma is dose-dependent: it increases with duration and level of exposure (ACS, 2004).

Occupations such as insulation workers, asbestos factory workers, shipyard workers, asbestos miners and millers, and construction workers have been identified as being at high risk of developing mesothelioma due to asbestos exposure (ACS, 2004; Antman et al., 1997). Family members of workers exposed to asbestos are suspected to be at an increased risk of developing mesothelioma because they may breathe in asbestos fibers from the clothing of exposed workers (ACS, 2004, 2006). Exposure to asbestos-containing building material is also a concern, particularly in older buildings when these materials begin to decompose. Asbestos may be detected in a water supply as well through the corrosion of asbestos-cement pipes (ACS, 2006). It is estimated that one-third of mesothelioma cases in the U.S. are caused by non-occupational exposure to asbestos such as among family members of workers and residents living near asbestos factories and mines (ACS, 2006).

Other risk factors for mesothelioma include exposure to thorium dioxide, used in the past for certain x-ray tests, and exposure to chemicals related to asbestos (e.g., zeolite) (ACS, 2004,

Source: Community Assessment Program, Center for Environmental Health, Massachusetts Department of Public Health. March 2006.

Antman et al., 1997). More recently, the simian virus SV40 has been implicated in the etiology of some mesotheliomas but the role of this virus remains unclear (ACS, 2004, Rizzo et al., 2001).

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APPENDIX C

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Terms

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

General Terms

Absorption

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

Acute

Occurring over a short time [compare with chronic].

Acute exposure

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

Additive effect

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

Adverse health effect

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

Aerobic

Requiring oxygen [compare with anaerobic].

Ambient

Surrounding (for example, ambient air).

Anaerobic

Requiring the absence of oxygen [compare with aerobic].

Analyte

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

Analytic epidemiologic study

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

Antagonistic effect

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

Background level

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

Biodegradation

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

Biologic indicators of exposure study

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

Biologic monitoring

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

Biologic uptake

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

Biomedical testing

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

Biota

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

Body burden

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

CAP [see Community Assistance Panel.]

Cancer

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

Cancer risk

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

Carcinogen

A substance that causes cancer.

Case study

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

Case-control study

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

CAS registry number

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

Central nervous system

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

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

Chronic

Occurring over a long time [compare with acute].

Chronic exposure

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

Cluster investigation

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

Community Assistance Panel (CAP)

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

Comparison value (CV)

Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during

the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)

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

Concentration

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

Contaminant

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

Delayed health effect

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

Dermal

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

Dermal contact

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

Descriptive epidemiology

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

Detection limit

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

Disease prevention

Measures used to prevent a disease or reduce its severity.

Disease registry

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

DOD

United States Department of Defense.

DOE

United States Department of Energy.

Dose (for chemicals that are not radioactive)

The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a

measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)

The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship

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

Environmental media

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

Environmental media and transport mechanism

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

EPA

United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology

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

Exposure

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

Exposure assessment

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

Exposure-dose reconstruction

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

Exposure investigation

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

Exposure pathway

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

Exposure registry

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

Feasibility study

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

Geographic information system (GIS)

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

Grand rounds

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

Groundwater

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

Half-life ($t_{½}$)

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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