ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

For Public Comment

W.R. GRACE SUPERFUND SITE
ACTON, MIDDLESEX COUNTY, MASSACHUSETTS

EPA FACILITY ID: MAD001002252
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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

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THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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Prepared by:

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

ATSDR	Agency for Toxic Substances and Disease Registry
AWD	Acton Water District
CAEPA	California Environmental Protection Agency
CAP	Community Assessment Program
CEL	Cancer Effect Level
COC	contaminant of concern
CNS	central nervous system
CREG	cancer risk evaluation guide
CSF	cancer slope factor
CVs	comparison values
DHHS	Department of Health and Human Services
EMEG	environmental media evaluation guide
HOD	Health Outcome Data
IARC	International Agency of Research on Cancer
IRIS	integrated risk information system
LOAEL	lowest observed adverse effect level
MCL	maximum contaminant level
MDPH	Massachusetts Department of Public Health
MIEBK	methyl isobutyl ketone
	(also known as 4-methyl-2-pentanone)
mg/kg/day	milligrams per kilogram per day
mg/L	milligrams per liter
mg/m^3	micrograms per cubic meter
MRL	minimal risk level
NAS	National Academy of Sciences
NOAEL	no observed adverse effect level
NPL	National Priority List
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PHA	public health assessment

ppb	parts per billion
TEF	toxicity equivalent factor
RBC	risk-based concentration
RfD	reference dose
RI	Remedial Investigation
RMEG	reference dose media evaluation guide
SDWA	Safe Drinking Water Act
SVOC	semi-volatile organic compound
µg/L	micrograms per liter
USEPA	United States Environmental Protection Agency
VOC	volatile organic compound
WHO	World Health Organization

Summary

The Massachusetts Department of Public Health, Bureau of Environmental Health Assessment under cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) previously evaluated the public health significance of environmental contamination at the W.R. Grace and Company, Inc., Acton, Massachusetts site in an initial release public health assessment dated November 4, 1992. Subsequent to that evaluation, the U.S. Environmental Protection Agency (USEPA) and the W.R. Grace and Company remediated portions of the site and conducted additional sampling. Because of the changing site conditions and the availability of additional environmental data, the USEPA contacted ATSDR in 2003 and requested that the new environmental data be reviewed and evaluated.

In 1978 the Town of Acton detected Volatile Organic Compounds (VOCs) in two of its municipal wells (Assabet One and Two), and as a result the Town of Acton closed the two wells. Use of the municipal wells, Assabet One and Two, was restarted in 1982 after the well water was treated with an activated carbon purification system (air stripping process) to remove the VOC's. In September 1983 the W.R. Grace site was added to the National Priorities List (NPL). Since that time the affected municipal supply wells, associated groundwater plume, and other portions of the site have become the subject of ongoing investigations by the USEPA and the W.R. Grace and Company, Inc.

ATSDR reviewed the available data and information for the W.R. Grace site through August 5, 2004, and identified one completed exposure pathway and three potential exposure pathways. The completed pathway is exposure to contaminants from the municipal drinking water supply and the potential pathways of exposure are private well water, surface water, and sediment. In addition, ATSDR evaluated the potential for exposure from to site related contaminants through vapor intrusion and fish consumption and considers these pathways to be incomplete.

Persons who were supplied water by the Town of Acton's Assabet One and Two wells between 1970 and 1978* may have previously drank, bathed, and showered with groundwater contaminated with VOC's. There is very limited data for this time period and there is no information regarding quality control and quality assurance (QA/QC) practices for the available data set. However, in an effort to address community concern regarding past exposure to drinking water contaminants in these wells, ATSDR did evaluate the available historical data. *Based on ATSDR's evaluation of the data for Assabet wells One and Two, past exposure to trichloroethylene (TCE) may have slightly exceeded the current health guideline between 1970 and 1978. However, ATSDR concludes that adverse health effects are not likely based on past exposure to TCE in drinking water at this site because very conservative assumptions were used in the assessment and the duration of exposure was limited.*

Presently the municipal drinking water wells affected by VOC contamination are treated through an air stripping process to remove the VOC's. Recent sampling has indicated no VOC contamination (due to the treatment process); however, it has indicated the presence of arsenic and manganese. Based on the concentrations reported for the wells and toxicological evaluations, adverse health effects are not expected to occur. *Therefore, ATSDR considers current exposure*

*The period time from when the wells were opened until the time contamination was discovered and the wells were closed.

to VOC's, arsenic, and manganese in the municipal drinking water supply to be a no apparent public health hazard.

Six private irrigation wells have been identified in the vicinity of the W.R. Grace site that are used for non-drinking water purposes. One well contained elevated levels of vinyl chloride, four wells indicated no present VOC contamination, and one well had not been sampled. The un-sampled well was properly sealed and permanently closed. Based on concentrations reported for the VOC contaminated private well and toxicological evaluations, adverse health effects are not expected to occur. *Therefore, ATSDR concludes that exposure to groundwater from private irrigation wells for non-drinking water uses poses no apparent public health hazard.*

Trespassers who access the W.R. Grace site may have been exposed in the past, may be presently exposed, or may be exposed in the future to contaminants in surface water. However, based on the concentrations reported in surface water and toxicological evaluations, adverse health effects are not expected to occur. *Therefore, ATSDR concludes that occasional exposure to surface water poses no apparent public health hazard.*

Trespassers who access the W.R. Grace site may also have been exposed in the past, may be presently exposed, or may be exposed in the future to contaminants in soil and sediment. Based on the concentrations reported in sediment and toxicological evaluations, exposure to arsenic indicates elevated risk for trespassers. ATSDR's evaluation of potential health effects from exposure to contaminants in sediment incorporated conservative assumptions for health effects, which may have resulted in an over estimation of potential exposure to individuals. ATSDR considered adolescents to be the most likely age group to access the site and based it assumptions on this age group. *ATSDR concludes that exposure to the highest levels of arsenic in sediment at the W.R. Grace site may pose a public health hazard to adolescents via dermal contact and ingestion. However, adolescents should not be trespassing on the site or accessing the areas with elevated levels of arsenic as often as estimated by ATSDR. There is a moderate elevated excess lifetime theoretical cancer risk for adults, if they routinely trespass on the site and come into contact with contaminated sediment.* The site is partially fenced, but trespassers can gain access. The site does have no trespassing signage.

ATSDR also considered possible exposure to contaminants from the volatilization of VOC's from groundwater into buildings near the site. *Based on modeled concentrations of contaminants from the volatilization of VOC's into buildings, ATSDR concludes that no adverse health effects are expected to occur. Therefore, the vapor intrusion pathway is classified as posing no apparent public health hazard.*

ATSDR considered the consumption of fish from Sinking Pond. Based on information provided to ATSDR, edible size fish from Sinking Pond could not be caught after several attempts. Because edible size fish could not be caught for evaluation, it is unlikely that persons accessing the W.R. Grace site are catching and consuming fish on a regular basis. Therefore, *ATSDR presently considers fish consumption from Sinking Pond to be a no apparent public health hazard.* However, if future conditions change at Sinking Pond, and fish of edible size can be caught and consumed by anglers, then a reassessment of this pathway may be warranted.

Based on its evaluation of completed and potential exposure pathways ATSDR recommends: (1) continued periodic monitoring of the municipal drinking water wells used by the Acton Water District to ensure that the air strippers are adequately removing VOC contamination and that the

municipal drinking water supply meets all the requirements of the Safe Drinking Water Act; (2) that USEPA and the W.R. Grace and Company address areas of the site with elevated levels of arsenic and manganese (i.e., Sinking Pond, North Lagoon Wetland) in soil and sediment in a manner that would be protective of public health; (3) that the five remaining active private irrigation wells in the vicinity of the W.R. Grace that are used for non-drinking water purposes be monitored periodically by W.R. Grace to determine whether levels of contaminants are of public health significance; and, (4) that no new private wells be installed in the vicinity of the contaminated groundwater plume near the W.R. Grace site.

Purpose and Health Issues

The W.R. Grace Site was listed on the U.S. Environmental Protection Agency's National Priorities List for Uncontrolled Hazardous Waste Sites (NPL) on September 8, 1983. As mandated by Congress, the Agency for Toxic Substances and Disease Registry (ATSDR) has prepared this public health assessment for the W.R. Grace Site. The data available for this site have been reviewed and summarized in this document. The purpose of this public health assessment (PHA) is to evaluate and present information pertaining to whether exposures to site-related contaminants could have occurred in the past, are presently occurring, or may occur in the future, and whether health effects could result from these exposures, based on available data. In addition, ATSDR will make recommendations or identify actions needed to minimize exposure to contaminants, as warranted. This document has been drafted in accordance with the ATSDR Public Health Assessment Guidance Manual [1].

Background

Site Description and History

The W.R. Grace site is located in the towns of Acton and Concord, Massachusetts, off of Independence Road, and covers approximately 260 acres. The site is bounded in the north by Fort Pond Brook and to the east and south by the Assabet River. The site was the former location of the American Cyanimid Company and the Dewey & Almy Chemical Company. These companies produced sealant products for rubber containers, latex products, plasticizers, resins, and other products. Operations at the W.R.Grace facility included the production of materials used to make concrete, container sealing compounds, latex products, and paper and plastic battery separators. Effluent wastes from these operations flowed into several unlined lagoons (the primary lagoon, secondary lagoon, north lagoon, and emergency lagoon), and solid and hazardous wastes were buried in or placed onto an on-site industrial landfill and several other disposal areas. These other waste sites include battery separator lagoons, the battery separator chip pile, the boiler lagoon, and the tank car area. In addition, the by-products of some chemical processes were disposed of in the blowdown pit. Since 1973, residents in south Acton have filed complaints about periodic odors and irritants in the air around the W.R. Grace plant. Investigations in 1978 indicated that two municipal wells, Assabet One and Assabet Two, were contaminated. As a result of these findings, the town took precautionary action and closed the two wells. The Acton Water District (AWD) operates and maintains air strippers to remove any volatile organic compounds that may be present in groundwater pumped from Assabet One, Assabet Two, Scribner, Lawsbrook, and Christofferson town wells. The AWD routinely samples and treats the water they provide to users to assure that safe quality standards are met. Discharge to all lagoons and the battery separator area ceased in 1980. Operable Unit-1 for the site, which included soil remediation, was completed in 1997.

Demographics

According to U.S. Census 2000 data, approximately 8,307 people reside within a one mile radius of the W.R. Grace Site. The majority of this population is white or Caucasian. However, the data reports that there are also approximately 66 blacks, 10 American Indian or Alaska Natives, and 552 Asians in this one mile radius. The residents in the vicinity include 874 children aged 6 and

younger, and 933 adults aged 65 and older. More detailed demographic information is presented in Appendix B, Figure 1.

Land Use and Natural Resources

Industrial parks border the site to the south and residential housing borders the site to the northeast. The U.S. Census 2000 data indicates approximately 3,161 housing units within a one mile radius of the site.

Sinking Pond, located in the center of the site, is the only natural water body on the site (Figure 2). Recreational use of the pond is unlikely since it appears that the pond does not support aquatic life and lacks aesthetic quality for swimming. The Assabet River which borders the site on the east and south is used for boating and fishing, but is not actively used for swimming. Fort Pond Brook borders the site on the north. It has been reported by the community, that recreational uses of Fort Pond Brook include fishing, canoeing, and kayaking. The Massachusetts Department of Fish and Game stocks Fort Pond Brook with trout every spring.

The W.R. Grace site overlies the Sinking Pond Aquifer†. The town of Acton has six wells that draw water from the aquifer for the municipal water supply. Two wells, Assabet One and Assabet Two were closed in 1978 after monitoring by the Acton Water District detected levels of Volatile Organic Compound (VOCs) in the wells. There are no private wells on site; however, six private irrigation wells have been identified in the vicinity of the plume.

Currently the W.R. Grace site is partially fenced to restrict some access. Some areas have posted "No Trespassing" signs (Figure 4). A former guard station at the main gate is no longer manned (Figure 3). Trespassers access the site to dirt bike, hike or horse back ride (Figure 5). Evidence of other activity on the site included domestic trash and campfire remnants. The area supports a variety of trees, shrubs, wild flowers, and wildlife. It has been reported in the past, that children do access the site for play and recreation.

Discussion of Environmental Contamination

Evaluation Process

The process by which ATSDR evaluates the possible health impact of contaminants is summarized in the following section and described in more detail in Appendix C. There are two steps in the evaluation process. The first step involves screening the available data to determine the contaminants of concern (COCs) for each media. As part of the ATSDR screening process, maximum detected concentrations of contaminants are compared with Comparison Values (CVs) to determine which chemicals, if any, require additional evaluation. CVs are concentrations of chemicals in the environment, below which no adverse health effects are expected to occur. In the event that a contaminant concentration exceeds its respective CV, it does not necessarily indicate that health effects are expected to occur. Rather, it is indicated that further evaluation of the particular contaminant and the ways in which individuals might be exposed to it is necessary. It should also be noted that other contaminants may be evaluated further if concerns are received from the community regarding the presence of the particular contaminant(s).

† An aquifer is a geological formation that conducts water so as to provide sufficient quantities of water to wells.

During the second step of the evaluation process, several important factors are considered in order to determine whether chemicals and exposure situations could be a public health hazard. These factors include the sampling locations and data quality, specific exposure routes (ingestion, inhalation, or direct skin contact), contaminant concentration, exposure frequency, exposure duration, toxicity information, human susceptibility, and community health concerns. Therefore, the second step involves the calculation of child and adult exposure doses for the identified COCs, which incorporate site-specific (if available) information on these important factors. Exposure doses are estimated amounts of a contaminant that individuals come in contact with as a result of specific exposure situations. As part of its evaluation ATSDR considers chemical exposures that may result in cancer, as well as, non-cancer health impacts.

The calculated, site-specific exposure doses are then compared with safe doses (also referred to as health guidelines), which indicate levels below which non-cancer health effects are unlikely to occur due to exposure. The health guidelines are health-protective values that have incorporated various safety factors to account for varying human susceptibility and the use of animal data to evaluate human exposure. In the event that the calculated, site-specific exposure dose for a chemical is greater than the established health guideline, it is then compared to the exposure doses from individual studies documented in the scientific literature that have reported health effects. Toxicological profiles, prepared by ATSDR for various chemicals, are the primary source of the scientific literature that is utilized in this part of the evaluation process. Toxicological information derived by USEPA is also considered. If a COC has been determined to be cancer-causing (carcinogenic), a theoretical cancer risk is also estimated.

A complete discussion of ATSDR's evaluation process is presented in Appendix C.

Pathways of Human Exposure

ATSDR evaluates exposure pathways that might result in human exposure to contaminants at a site. ATSDR considers a human exposure pathway to consist of five principal elements: (1) a source of contamination, (2) transport through an environmental medium, (3) a point of exposure, (4) a route of human exposure, and (5) a receptor population. If all five elements of an exposure pathway exist currently or did exist in the past, the pathway is considered complete. If one element or more is missing or if exposure is possible but not likely to occur, the pathway is considered a potential pathway of exposure.

ATSDR evaluated possible exposure pathways for the W.R. Grace site. The completed and potential exposure pathways are discussed in the following paragraphs, and a summary of the information is provided in Appendix A, Tables 1 and 2.

A review of the environmental data and site conditions indicates one completed and three potential exposure pathways by which people could be or could have been exposed to contaminants from the site. Exposure to contaminants in public water supply wells is a completed pathway. Exposure to contaminants in private irrigation wells, surface water, and sediment are potential exposure pathways. The public health implications of exposure via these pathways is discussed in the following section. In addition, ATSDR considered exposure to contaminants through vapor intrusion and consumption of fish. ATSDR considers these pathways to be incomplete. Screening for vapor intrusion indicated that contaminants in indoor air should not accumulate at levels of health

concern. The fish consumption pathway is considered incomplete, because edible size fish could not be obtained after a considerable effort and no subsistence fish-consuming population was identified.

Public Water Supply, Private Well, and Groundwater Sampling

As part of a Remedial Investigation (RI) for Operational Unit 3[‡] (groundwater) and ongoing annual groundwater monitoring programs, W.R. Grace has collected groundwater samples from six private irrigation wells, five public water supply wells, and over 100 monitoring wells. Two private irrigation wells are located north of the W.R. Grace site, and five are located south of the site. The School Street wellfield, which includes the Christofferson, Lawsbrook, and Scribner public water supply wells, is located north of the site. The Assabet wellfield, which includes Assabet One and Assabet Two public water supply wells, is located south of the site (Figure 3). Groundwater monitoring wells are located throughout and around the perimeter of the site.

W.R. Grace identified six private irrigation wells located near the W.R. Grace site. On May 6, 2002, W.R. Grace collected samples from five of these wells and analyzed each sample for VOC's. Four of the private irrigation wells are located south of the site and beyond the horizontal extent of groundwater contamination. One is used as an irrigation well, two are used as non-contact cooling water sources for a manufacturing plant, and one serves as a drinking water, ice resurfacing, and bathing water source for a sports arena. No contaminants were found in these four wells during the May 6, 2002 sampling. Two of the private irrigation wells are located north of the W.R. Grace site and within the horizontal extent of the VOC groundwater contamination plume originating at the site. One of these wells contained vinyl chloride (0.35 parts per billion [ppb]) above its CV of 0.03. The remaining private well was not sampled, so W.R. Grace is seeking permission to collect a sample. Both of these wells were constructed after 1995 and are only used for irrigation.

W.R. Grace has conducted several rounds of public water supply and groundwater monitoring well sampling. As part of the RI, groundwater sampling at the W.R. Grace site was conducted between August 2000 and June 2002. During this period, W.R. Grace collected samples from the public water supply wells, 25 transect[§] locations beneath the Assabet River, 22 transect locations beneath Fort Pond Brook, and 10 transect locations adjacent to Fort Pond Brook. Samples were analyzed for VOCs, semi-volatile organic compounds (SVOCs), and metals [27]. Additional sampling under the site's annual monitoring program was conducted between October 14, 2002, and November 12, 2002. W.R. Grace collected samples, which were analyzed for VOCs, from public water supply wells and 101 monitoring wells [28]. A draft human health risk assessment also reports results from annual groundwater monitoring conducted in 1999, 2000, and 2001, as well as thallium sampling results from fall 2002 [35]. Arsenic and manganese were found above their CVs in public water supply wells. Groundwater monitoring wells contained 12 VOCs, 3 SVOCs, and 13 metals above CVs.

Surface Water and Sediment Sampling

W.R. Grace conducted surface water and sediment sampling to assess site contamination and to

‡ Term for each of a number of separate activities undertaken as part of a Superfund site cleanup. A typical operable unit would be removal of drums and tanks from the surface of a site.
§ A sample area, usually in the form of a long continuous strip.

support a human health and baseline ecological risk assessment. Sinking Pond and Gravel Pit Wetland samples are combined because these areas are proximate to each other. Fort Pond Brook and North Lagoon samples are combined because these two areas are hydraulically connected.

In 2002, W.R. Grace collected surface water samples from the Assabet River, Sinking Pond, Gravel Pit Wetland, Fort Pond Brook, and North Lagoon as part of a screening-level ecological risk assessment. The sampling results are summarized in a draft human health risk assessment [35]. W.R. Grace collected additional surface water samples from Sinking Pond in August, September, and December 2002. The eight samples collected from two sampling stations were analyzed for dissolved metals. Three dry weather samples were collected at Fort Pond Brook on August 12, 2002, and analyzed for VOCs, SVOCs, and total and dissolved metals. Three wet weather samples were collected on October 23, 2002, and analyzed for total and dissolved chromium and hexavalent chromium [33]. In April 2003, additional surface water samples were collected from Sinking Pond (two sample locations) and North Lagoon (two sample locations). Sinking Pond samples were analyzed for total and dissolved metals; North Lagoon samples were analyzed for VOCs, SVOCs, and total and dissolved metals [34]. Only arsenic was found above CVs in the Assabet River [Table 6]. Sinking Pond and the Gravel Pit Wetland contained antimony, arsenic, and manganese above CVs [Table 6]. Phenanthrene, a non-cancerous polycyclic aromatic hydrocarbon (PAH) which has no CV, was also detected at less than 1 ppb (below the CVs for other non-cancerous PAHs). Fort Pond Brook and North Lagoon contained bis(2-ethylhexyl) phthalate, arsenic, and manganese above CVs and phenanthrene below 1 ppb [Table 6].

In 2002, W.R. Grace collected sediment samples from the Assabet River, Sinking Pond, Gravel Pit Wetland, Fort Pond Brook, and North Lagoon as part of a screening-level ecological risk assessment. The sampling results were summarized in a draft human health risk assessment [35]. W.R. Grace conducted additional sediment sampling in Sinking Pond, Fort Pond Brook, and the North Lagoon Wetland from August 12 to 15, 2002. Twelve samples from Sinking Pond were collected and analyzed for SVOCs, pesticides, polychlorinated biphenyls (PCBs), and metals. Three samples were collected from Fort Pond Brook and analyzed for VOCs, SVOCs, and metals. Six samples from North Lagoon Wetland were collected and analyzed for metals [33]. On April 28 and 29, 2004, W.R. Grace collected sediment samples from Sinking Pond (six samples), Gravel Pit Wetland (six samples), Fort Pond Brook (two samples), and North Lagoon (nine samples). These samples were analyzed for VOCs, SVOCs, pesticides, PCBs, and/or inorganics [34]. Benzo(a)pyrene, arsenic, and thallium were found above CVs in the Assabet River [Table 7]. Acenaphthene, benzo(g,h,i)perylene, and phenanthrene (three PAHs without CVs) were also detected at concentrations below 1 part per million (ppm) (below the CVs for other non-cancerous PAHs). Sinking Pond and Gravel Pit Wetland contained benzo(a)pyrene, two PCBs, arsenic, iron, and manganese above CVs, as well as acenaphthene, benzo(g,h,i)perylene, and phenanthrene at concentrations below 1 ppm [Table 7]. Fort Pond Brook and North Lagoon contained three PAHs, arsenic, chromium, copper, iron, and manganese above CVs, as well as four PAHs, which have no CVs, at concentrations near or below 1 ppm [Table 7].

Fish Sampling

On September 24, 2002, W.R. Grace attempted to collect fish, using gill net and rod and reel methods, from Sinking Pond to support a baseline ecological risk assessment. Gill nets were set

and checked once and then again 24 hours later. Rods and reels were set with earthworms, lures, and corn. No fish were caught in Sinking Pond using either method. Therefore, another effort to collect fish using baited minnow traps was completed on October 23, 2002. Common shiner minnows were collected and analyzed for SVOCs, pesticides, PCBs, and metals [33]. Dieldrin, arsenic, and iron were detected above their CVs in minnow samples from Sinking Pond [Table 8]. Lead was also detected but has no CV.

Completed Exposure Pathways

Public Water Supply Wells

As previously discussed, arsenic and manganese were found above their CVs in public water supply wells; therefore, ATSDR calculated site specific exposure doses and compared them to health guidelines (Appendix C).

Arsenic

ATSDR calculated oral exposure doses using the maximum concentration of arsenic, 0.0052 milligrams per liter (mg/L), found in the public water supply wells. The estimated exposure doses for adults and children are 0.00015 milligrams per kilogram per day (mg/kg/day) and 0.00033 mg/kg/day, respectively. The estimated dose for adults is below the health guideline while the estimated dose for children is slightly above the established health guideline of 0.0003 mg/kg/day. The health guideline is the USEPA's Reference Dose (RfD), which is based on a human study that indicated hyperpigmentation[**], hyperkeratosis[††], and possible vascular complications in a chronically exposed population in Taiwan. While the estimated dose for a child slightly exceeds the ATSDR health guideline, it is well below the dose that would result from ingesting water with an arsenic level at the USEPA MCL of 0.010 mg/L. *Adverse effects are unlikely at these doses.*

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The World Health Organization (WHO), the Department of Health and Human Services (DHHS), and the USEPA have determined that inorganic arsenic is a human carcinogen [4]. The USEPA has calculated a cancer unity risk factor, which can be used to estimate the probability of excess theoretical cancer risk for a lifetime of exposure to arsenic. ATSDR calculated an excess lifetime theoretical cancer risk of 2.23E-05 based on the maximum concentration (0.0052 mg/L) of arsenic in the public supply wells, indicating a low increased theoretical cancer risk . In addition, ATSDR compared the calculated adult oral exposure dose to the Cancer Effect Level (CEL) in its toxicological profile for arsenic and found that it was more than 500 times lower than the CEL (0.038 mg/kg/day). *Therefore, it is unlikely that there is a significant increased risk of cancer due to ingestion of arsenic in public water supply wells.*

Manganese

ATSDR calculated oral exposure doses using the maximum concentration of manganese, 0.52 mg/L, found in the public water supply wells. The estimated exposure doses for adults and

** Hyperpigmentation is a common, usually harmless condition in which patches of skin become darker in color than the normal surrounding skin.

†† Hyperkeratosis is an excessive thickening of the outer layer of the skin.

children are 0.01486 mg/kg/day and 0.03250 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.05 mg/kg/day. The health guideline is the USEPA's RfD, which is based on a human study that indicated central nervous system effects based on chronic ingestion. *Because the calculated exposure doses are below health guidelines, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.*

Manganese is not classifiable as to human carcinogenicity. Existing studies are inadequate to determine its carcinogenicity.

Public Water Supply Wells, Historical Data, VOC's

In the 1992 Initial Release Public Health Assessment [44], prepared by the Massachusetts Department of Health, there is some historical data reported on Volatile Organic Compounds (VOC's) [Table 5]. This data is very limited, and there is no Quality Assurance or Quality Control (QA/QC) information on this data. ATSDR believes this data to be of limited value for determining public health conclusions for past exposure. In most cases there is only one data point per year for the VOC's listed. In the case of benzene, there is only one data point for the years 1978 through 1992. The exact duration of exposure to these contaminants in drinking water wells could not be determined. Routine monitoring did not occur in these wells after they were opened in 1970, so there is no way to determine when the wells were contaminated. The contamination was detected in 1978 as part of a monitoring conducted for an application for expansion of operations at the W.R. Grace site [44]. After the contamination was discovered in 1978 the wells were closed. For the purpose of this evaluation, we assumed that the maximum duration of exposure was nine years, the length of time the wells were opened (1970 to 1978). However, most likely the exposure duration is less than nine years. In addition, the water from these wells was mixed with water from other wells prior to distribution to homes, so it is highly unlikely that any home would have received the maximum reported contaminant concentration at the well from their tap.

Even though the data is limited, ATSDR has incorporated it in this assessment in an effort to answer community questions and concerns regarding past exposures. ATSDR has reviewed and calculated non-carcinogenic and carcinogenic risk for these VOC's, where possible. First, ATSDR compared the maximum concentrations for ethylbenzene, 1,1 dichloroethylene, benzene, 1,1,1 trichloroethane, trichloroethylene (TCE), methylene chloride, and 1,1 dichloroethane to its comparison values (CV's). The maximum concentrations of ethylbenzene, and 1,1,1 trichloroethane were below ATSDR's CV's and were therefore not evaluated further.

The following contaminants were reported at levels above their CVs in the historical public water supply well data, therefore; ATSDR calculated site specific exposure doses and compared them to health guidelines.

Benzene

ATSDR calculated oral exposure doses using the maximum concentration of benzene, 0.0021 milligrams per liter (mg/L), found in the Assabet wells. The estimated exposure doses for adults and children are 0.00009 milligrams per kilogram per day (mg/kg/day) and 0.00026 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.004 mg/kg/day. The health guideline is the USEPA's Reference Dose (RfD), which is based on a human study that indicated a decreased lymphocyte count in an inhalation occupational

study by Rothman, et al, 1996. The oral RfD is based on route to route extrapolation modeling of the results of benchmark dose modeling. *Because the calculated doses are well below the health guideline, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.*

Benzene can cause cancer of the blood-forming organs. The Department of Health and Human Services (DHHS) has determined that benzene is a known human carcinogen. Long-term exposure to relatively high levels of benzene in the air can cause cancer of the blood-forming organs. This condition is called leukemia. The health effects that might occur in humans following long-term exposure to too food and water contaminated with benzene are not as well known. In animals, exposure to food or water contaminated with benzene can damage the blood and immune system and can even cause cancer. Under the proposed revised Carcinogen Risk Assessment Guidelines (U.S. EPA, 1996), benzene is characterized as a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies [5].

ATSDR calculated an excess lifetime theoretical cancer risk of 4.75E-06 based on the maximum concentration (0.0021 mg/L) of benzene in the Assabet wells, indicating a very low increased theoretical cancer risk. In addition, ATSDR compared the calculated adult oral exposure dose to the Cancer Effect Level (CEL) in its toxicological profile for benzene and found that it was thousands of times lower than the CEL (50 mg/kg/day) [5]. *Therefore, it is unlikely that there is a significant increased risk of cancer due to ingestion of benzene from the Assabet wells in the past.*

Methylene Chloride

ATSDR calculated oral exposure doses using the maximum concentration of methylene chloride, 0.046 milligrams per liter (mg/L), found in the Assabet wells. The estimated exposure doses for adults and children are 0.00197 milligrams per kilogram per day (mg/kg/day) and 0.00575 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.06 mg/kg/day. The health guideline is the USEPA's Reference Dose (RfD), which is based on a study of male and female rats that caused liver toxicity. *Because the calculated doses are well below the health guideline, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.*

The Department of Health and Human Services (DHHS) has determined that methylene chloride may reasonably be anticipated to be a carcinogen. The International Agency for Research on Cancer (IARC) has classified methylene chloride in Group 2B, possibly carcinogenic to humans. The EPA has determined that methylene chloride is a probable human carcinogen [12].

ATSDR calculated an excess lifetime theoretical cancer risk of 1.42E-05 based on the maximum concentration (0.046 mg/L) of methylene chloride in the Assabet wells, indicating a low increased theoretical cancer risk. In addition, ATSDR compared the calculated adult oral exposure dose to the Cancer Effect Level (CEL) in its toxicological profile for methylene chloride and found that it was thousands of times lower than the CEL (320 mg/kg/day) [12]. *Therefore, it is unlikely that there is a significant increased risk of cancer due to ingestion of benzene from the Assabet wells in the past.*

Trichloroethylene (TCE)

ATSDR calculated oral exposure doses using the maximum concentration of TCE, 0.008 milligrams per liter (mg/L), found in the Assabet wells. The estimated exposure doses for adults and children are 0.00034 milligrams per kilogram per day (mg/kg/day) and 0.00100 mg/kg/day, respectively. The estimated dose for adults and children slightly exceeds the established health guideline of 0.0003 mg/kg/day. The health guideline is the USEPA's Reference Dose (RfD), from the Region 9, Preliminary Remediation Guide Table. ATSDR also compared the estimated exposure doses for adults and children with the LOAEL in the ATSDR toxicological profile for TCE and found that the estimated exposure doses were thousands of times lower than the LOAEL (250 mg/kg/day). The LOAEL is based on a study of rats exposed orally to TCE for 5 days per week for 52 weeks. *The available data indicates adverse health effects are not likely based on exposure to TCE in drinking water at this site.*

It is uncertain whether people who breathe or drink water containing TCE are at higher risk of cancer, or of having reproductive effects. More and more studies suggest that more birth defects may occur when mothers drink water containing TCE. In one study, people who used water for several years from two wells that had high levels of TCE, may have had a higher incidence of childhood leukemia than other people, but these findings are not conclusive [17]. In another study of TCE exposure from well water, increased numbers of children were reported to be born with heart defects, which is supported by data from some animal studies showing developmental effects of TCE on the heart. However, other chemicals were also in the water from this well and may have contributed to these effects. One study reported a higher number of children with a rare defect in the respiratory system and eye defects. Another study reported that the risk for neural tube defects and oral cleft palates were higher among mothers with TCE in their water during pregnancy. Children listed in the National Exposure Subregisty of persons exposed to TCE were reported to have higher rates of hearing and speech impairment. There are many questions regarding these reports. There were small numbers of children with defects and TCE levels at which the effects occurred were not defined well. Thus, it is not possible to make firm conclusions about the exact effects of TCE from these studies, and more studies need to be done [17].

We do not have any clear evidence that TCE alone in drinking water can cause leukemia or any other type of caner in humans. As part of the National Exposure Subregistry, ATSDR compiled data on 4,280 residents of three states (Michigan, Ilinnois, and Indiana) who had environmental exposure to TCE. It found no definitive evidence for an excess of cancers from TCE exposure. An increase of respiratory cancer was noted in older men, but this effect was thought to result from smoking rather than TCE exposure. A study in New Jersey found an association between leukemia in women and exposure to TCE in drinking water. A study in Massachusetts found that exposure was associated with leukemia in children. In studies with people, there are many factors that are not fully understood. More studies need to be done to establish the relationship between exposure to TCE and cancer [17].

In studies using high doses of TCE in rats and mice, tumors in the lungs, liver, and testes were found, providing some evidence that high doses of TCE can cause cancer in experimental animals. Based on the limited data in humans regarding TCE exposure and cancer, and evidence that high doses of TCE can cause cancer in animals, the International Agency for Research on Cancer (IARC) has determined that TCE is probably carcinogenic to humans. TCE has been

nominated for listing in the National Toxicology Program (NTP) 9th Report on Carcinogens. Evaluation of this substance by the NTP review committee is ongoing [17].

The EPA has withdrawn its Cancer Slope Factor (CSL) for TCE and a new one has not been adopted. ATSDR reviewed current literature on TCE and found that the California (CA) EPA has developed a CSF that is fairly commonly used in assessing excess lifetime theoretical cancer risk [25]. ATSDR calculated its excess lifetime theoretical cancer risk for TCE in this assessment using the withdrawn EPA CSF and the CA EPA CSF. ATSDR calculated an excess lifetime theoretical cancer risk of 5.26E-06 and 4.27E-06 based on the maximum concentration (0.008 mg/L) of TCE in the Assabet wells, indicating that there is a very low to insignificant increased theoretical cancer risk based on the assumptions and CSF's used. ATSDR also compared the calculated adult oral exposure dose to the Cancer Effect Level (CEL) in its toxicological profile for TCE and found that it was thousands of times lower than the CEL (1,000 mg/kg/day) [17]. *Based on the former EPA CSF and the CA EPA CSF, there is a very low to insignificant increased risk of cancer due to ingestion of TCE from the Assabet wells in the past.*

1,1, dichloroethane

ATSDR calculated oral exposure doses using the maximum concentration of 1,1 dichloroethane, 0.024 milligrams per liter (mg/L), found in the Assabet wells. The estimated exposure doses for adults and children are 0.00103 milligrams per kilogram per day (mg/kg/day) and 0.00300 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.10 mg/kg/day. The health guideline is the USEPA's Reference Dose (RfD), from the EPA Region III Risk-Based Concentration Table. *Because the calculated doses are well below the health guideline, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.*

The Department of Health and Human Services (DHHS), the IARC, and the EPA have not classified, 1,1 dichloroethane for carcinogenicity [8]. There is no cancer slope factor available for 1,1, dichloroethane, therefore an excess lifetime theoretical cancer risk could not be calculated.

1,1, dichlorethene (a.k.a. 1,1, dichloroethylene)

ATSDR calculated oral exposure doses using the maximum concentration of 1,1 dichloroethene, 0.055 milligrams per liter (mg/L), found in the Assabet wells. The estimated exposure doses for adults and children are 0.00236 milligrams per kilogram per day (mg/kg/day) and 0.00688 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.009 mg/kg/day. The health guideline is ATSDR's Chronic Minimal Risk Level [9]. *Because the calculated doses are well below the health guideline, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.*

The U.S. Department of Health and Human Services has not classified 1,1-dichloroethene with respect to carcinogenicity. The IARC has determined that 1,1-dichloroethene is not classifiable as to its carcinogenicity in humans, however the EPA has determined that 1,1-dichloroethene is a possible human carcinogen. NTP does not include it in its list of substances expected to be human carcinogens [9]. There is no cancer slope factor available for 1,1, dichloroethane; therefore, an excess lifetime theoretical cancer risk could not be calculated.

Potential Exposure Pathways

Private Wells

Six private irrigation wells near the W.R. Grace site were identified. Five of the six wells were sampled for VOC contamination. W.R. Grace sought permission to sample the remaining well, but permission could not be obtained. The property owner has requested that W.R. Grace properly abandon and close this well. No VOC contamination was found in four wells, but one well contained vinyl chloride above its CV. These wells are not used for drinking water purposes, so ATSDR developed a very health protective exposure scenario based on dermal contact and incidental ingestion. Inhalation of VOCs was considered; however, volatilization and dilution with outdoor air make this pathway insignificant.

Vinyl chloride

ATSDR calculated exposure doses for incidental ingestion and dermal contact using the maximum concentration of vinyl chloride, 0.00035 mg/L, found in the one private well. The estimated incidental ingestion doses for adults and children are 0.0000005 mg/kg/day and 0.000002 mg/kg/day, respectively. The estimated dose for adults and children are below the established health guideline of 0.00002 mg/kg/day. The health guideline is ATSDR's chronic Minimal Risk Level (MRL), which is based upon areas of cellular alteration in the livers of rats [18]. *Because the calculated doses are below both the MRL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed due to incidental ingestion of vinyl chloride in private well water.* ATSDR estimated dermal contact exposure doses for adults and children to vinyl chloride; however, no studies regarding vinyl chloride absorption in humans were found for comparison. Animal data suggest that dermal absorption of vinyl chloride vapor is not likely to be significant.

Based on evidence from human carcinogenicity studies, vinyl chloride is considered carcinogenic to humans by the DHHS, the International Agency for Research on Cancer (IARC), and the USEPA [18]. The USEPA has calculated a cancer unity risk factor, which can be used to estimate the probability of excess cancer risk for a lifetime of exposure to vinyl chloride. ATSDR calculated an excess lifetime theoretical cancer risk of 9.63E-08 from incidental ingestion and dermal contact based on the maximum concentration (0.00035 mg/L) of vinyl chloride. *Generally, an increased risk below 1.00E -06 is not considered significant; therefore, it is unlikely that there is a significant increased risk of cancer from vinyl chloride due to incidental ingestion and dermal contact with private well water.*

Surface Water

Surface water samples were collected and analyzed from the Assabet River, Sinking Pond, Gravel Pit Wetland, Fort Pond Brook, and North Lagoon. Arsenic, phenanthrene, antimony, manganese, and bis(2-ethylhexyl)phthalate were found above CVs, so ATSDR developed an exposure scenario based on dermal contact and incidental ingestion of contaminants in surface water.

Arsenic

ATSDR calculated incidental ingestion exposure doses using the maximum concentration of arsenic, 0.02705 mg/L, found in surface water. The estimated exposure doses for adults and children are 0.00000265 mg/kg/day and 0.00001159 mg/kg/day, respectively. The estimated

doses for adults and children are below the established health guideline of 0.0003 mg/kg/day. The health guideline is the USEPA's RfD, which is based on a human study that indicated hyperpigmentation, hyperkeratosis, and possible vascular complications in a chronically exposed population in Taiwan. *Because the calculated doses are below health guidelines, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from arsenic would be observed from incidental ingestion of surface water.* ATSDR calculated an exposure dose of 0.000481 mg/kg/day for an adult and 0.000834 mg/kg/day for a child for dermal contact to arsenic in surface water. The estimated dose for adults and children is thousands of times below the LOAEL (2.5 mg/kg/day) for dermal effects in ATSDR's arsenic toxicological profile [4]. This LOAEL is based on a mouse study that caused local hyperplasia‡‡ and irritation after 18 weeks of exposure. *Because the calculated doses are thousands of times below the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from arsenic would be observed from dermal contact with surface water.*

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The WHO, the DHHS, and the USEPA have determined that inorganic arsenic is a human carcinogen [4]. The USEPA has calculated a cancer unity risk factor, which can be used to estimate the probability of excess theoretical cancer risk for a lifetime of exposure to arsenic. ATSDR calculated an excess lifetime theoretical cancer risk for an adult of 7.21E-04 for incidental ingestion and dermal contact using the maximum concentration (0.02705 mg/L) of arsenic in surface water. *This indicates a low to moderate increased risk for an adult. Even though these calculations indicate a moderate increased theoretical cancer risk the assumptions are very conservative and most likely overestimate actual exposure.*

Phenanthrene (PAH)

ATSDR calculated an incidental ingestion exposure dose using the maximum concentration of phenanthrene, 0.000014 mg/L, found in surface water. The estimated exposure dose for adults and children are 0.000000001 mg/kg/day and 0.000000006 mg/kg/day, respectively. The estimated doses for adults and children are thousands of times lower than the intermediate LOAEL for animals in ATSDR's toxicological profile for polycyclic aromatic hydrocarbons (PAHs) (120 mg/kg/day) [14]. This LOAEL is based on a female mouse study that caused increased liver weight after 6 months of exposure. *Because the calculated doses are thousands of times below the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from phenanthrene would be observed from incidental ingestion of surface water.* ATSDR calculated an exposure dose of 0.00000007 mg/kg/day for an adult and 0.00000012 mg/g/day for a child for dermal contact to phenanthrene in surface water. The estimated dose is thousands of times below the NOAEL (0.09 mg/kg/day) for dermal effects in ATSDR's PAH toxicological profile [14]. This NOAEL is based on a mouse study that caused skin tumors. *Because the calculated doses are thousands of times below the NOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from phenanthrene would be observed from dermal contact with surface water.*

‡‡ The term *hyperplasia* refers to overgrowth of tissue due to an abnormal increase in the number of cells in the tissue.

Presently, a quantitative risk estimate has only been developed for benzo(a)pyrene. It is a known animal carcinogen and considered a probable human carcinogen by the IARC and the USEPA [14]. The USEPA has calculated a cancer unity risk factor for benzo(a)pyrene which ATSDR used to estimate the probability of excess cancer risk for a lifetime of exposure. Because there is no cancer unity risk factor for the other PAH's, USEPA and others developed toxicity equivalent factors (TEFs), based on relative potency to benzo(a)pyrene, to estimate carcinogenic risk [14]. ATSDR calculated an excess lifetime theoretical cancer risk for phenanthrene based on incidental ingestion and dermal contact with phenanthrene in surface water. This estimate indicates a 5.02E -10 increased theoretical cancer risk. ATSDR also compared the total estimated incidental ingestion and dermal contact dose to the CEL in its toxicological profile for PAH's and found that it was thousands of times lower than the CEL (0.57 mg/kg/day) [14]. *Therefore, it is unlikely that there is a significant increased risk of cancer from phenanthrene exposure to surface water.*

Antimony

ATSDR calculated incidental ingestion exposure doses using the maximum concentration of antimony, 0.02705 mg/L, found in surface water. The estimated exposure doses for adults and children are 0.00000265 mg/kg/day and 0.0000116 mg/kg/day respectively. The estimated dose for adolescents and children is below the established health guideline of 0.0004 mg/kg/day. The health guideline is the USEPA's RfD, which is based on a rat study that affected longevity, blood glucose, and cholesterol. *Because the calculated doses are more than thirty times lower than the RfD, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed.*

Antimony is not classifiable as to human carcinogenicity. Existing studies are inadequate to determine its carcinogenicity [3].

Manganese

ATSDR calculated an incidental ingestion dose using the maximum concentration of manganese, 4.8 mg/L, found in surface water. The estimated exposure doses for adults and children are 0.000470 mg/kg/day and 0.00206 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.14 mg/kg/day (USEPA IRIS). The health guideline is the USEPA's RfD, which is based on a human study that indicated central nervous system effects based on chronic ingestion [11]. *Because the calculated exposure doses are below the RfD, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed at these doses.* ATSDR reviewed available literature, but there were no studies located regarding tissue distribution of manganese.

Manganese is not classifiable as to human carcinogenicity. Existing studies are inadequate to determine its carcinogenicity [11].

Bis(2-ethylhexyl)phthalate (a.k.a. Di(2-ethylhexyl)phthalate)

ATSDR calculated an incidental ingestion dose using the maximum concentration of bis(2-ethylhexyl)phthalate, 0.017 mg/L, found in surface water. The estimated exposure doses for adults and children are 0.00000166 mg/kg/day and 0.00000729 mg/kg/day, respectively. The estimated dose for adults and children is below the established health guideline of 0.02 mg/kg/day [46]. *Because the calculated exposure doses are thousands of times below the RfD,*

and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed.

The USEPA considers bis(2-ethylhexyl)phthalate to be carcinogenic; however, it is not classified by DHHS or IARC [10]. The USEPA has calculated a cancer unity risk factor, which can be used to estimate the probability of excess cancer risk for a lifetime of exposure to bis(2-ethylhexyl)phthalate. ATSDR calculated an excess lifetime theoretical cancer risk for incidental ingestion and dermal contact with surface water of 4.25E-06, using the maximum concentration (0.017 mg/L) of bis(2-ethylhexyl)phthalate in surface water, indicating a very low increased theoretical cancer risk . *Therefore, it is unlikely that there is a significant increased risk of cancer from bis(2-ethylhexyl)phthalate.*

Sediment

Arsenic

ATSDR calculated exposure doses for incidental ingestion and dermal contact using the maximum concentration of arsenic from the North Lagoon Wetland, 3900 mg/kg, found in sediment. ATSDR considered adolescents to be the most likely age group to access/ trespass on the site in these areas and based its exposure assumptions on this population. The estimated doses for incidental ingestion and dermal contact for adolescents are 0.0009 mg/kg/day and 0.00006 mg/kg/day, respectively. The estimated incidental ingestion dose for an adolescent slightly exceeds the established health guideline of 0.0003 mg/kg/day. The health guideline is the USEPA's RfD, which is based on a human study that indicated hyperpigmentation, hyperkeratosis, and possible vascular complications in a chronically exposed population in Taiwan. *The estimated incidental ingestion dose for an adolescent does slightly exceed the RfD; however, very conservative assumptions (i.e., exposure frequency and duration) were made in calculating doses, and it is not likely that adolescents or others would ingest sediment with the highest concentrations as often as ATSDR has estimated.*

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The WHO, the DHHS, and the USEPA have determined that inorganic arsenic is a human carcinogen [4]. The USEPA has calculated a cancer unity risk factor, which can be used to estimate the probability of excess cancer risk for a lifetime of exposure to arsenic. ATSDR calculated an excess lifetime theoretical cancer risk for an adult of 5.73E -04 for incidental ingestion and dermal exposure using the maximum concentration of arsenic in sediment (3900 mg/kg). *This indicates a low to moderate increased risk for an adult. Even though these calculations indicate a moderate increased theoretical cancer risk the assumptions are very conservative (see appendix C) and most likely overestimate actual exposure, because adults are unlikely to trespass on the site and contact sediment as frequently as ATSDR estimated.*

Chromium

ATSDR calculated an incidental ingestion exposure dose using the maximum concentration of chromium, 223 mg/kg, found in sediment. The estimated exposure dose for an adolescent is 0.00005 mg/kg/day. The estimated dose is below the established health guideline of 0.003 mg/kg/day. The health guideline is the USEPA's RfD, which is based on a one year study of rats that ingested drinking water containing between 0.45 and 11.2 ppm hexavalent chromium.

ATSDR also compared the estimated exposure dose to the chronic oral LOAEL of 0.57 mg/kg/day in it toxicological profile for chromium [6]. The estimated dose is greater than 2,000 times below the LOAEL. *Because the calculated exposure dose is below the RfD and the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed.* ATSDR calculated an exposure dose of 0.00011 mg/kg/day for an adolescent for dermal contact to chromium in sediment. The estimated dose for an adolescent is approximately 40 times below the LOAEL (0.004 mg/kg/day) for dermal effects in ATSDR's chromium toxicological profile [6]. This LOAEL is based on a human study that caused nasal septum ulceration, possible gastritis, and ulcers after an average exposure of seven and a half years. *Because the calculated doses are below the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from chromium would be observed from dermal contact with sediment.*

Several studies have shown that chromium(VI) compounds can increase the risk of lung cancer and some animal studies have also shown an increased risk of cancer. The USEPA has determined that chromium(VI) in air is a human carcinogen, however carcinogenicity by the oral route of exposure cannot be determined and is classified as Group D [6]. There is no cancer slope factor chromium, therefore an excess lifetime cancer risk could not be calculated.

Copper

ATSDR calculated an incidental ingestion exposure dose for copper using the maximum concentration, 6,000 mg/kg, found in sediment. ATSDR calculated an adolescent exposure dose of 0.00137 mg/kg/day. This exposure dose is well below the chronic LOAEL of 0.056 mg/kg/day listed in ATSDR's toxicological profile for copper [7]. *ATSDR also compared the calculated exposure doses to the recommended dietary allowances and found that they were below levels that might be expected to cause adverse health effects* [41]. According to the National Academy of Sciences (NAS), an occasional intake of up to 10 mg/day is probably safe for human adults. ATSDR calculated an exposure dose for adolescents for dermal contact with sediment; however, no studies were located regarding the rate and extent of absorption following dermal exposure of humans and animals to copper.

Copper is not classifiable as to human carcinogenicity [7].

Iron

ATSDR calculated an incidental ingestion exposure dose for iron using the maximum concentration, 390,000 mg/kg, found in sediment. ATSDR calculated an adolescent exposure dose of 0.089 mg/kg/day. ATSDR has no toxicological profile or CV for iron, so ATSDR compared the estimated exposure doses to dietary intake levels published by the NAS. Adverse health effects are unlikely in a healthy person at daily intake levels between 25 and 75 mg/day according to the NAS [41]. *Therefore, it is unlikely that adverse health effects would be observed at these doses.* No human or animal studies were found regarding dermal exposure to iron.

Iron is not considered to be carcinogenic to humans or animals.

Manganese

ATSDR calculated an incidental ingestion dose for adolescents using the maximum concentration of manganese found in sediment from the North Lagoon Wetland (48,400 mg/kg). The estimated exposure dose for adolescents is 0.011 mg/kg/day. The estimated dose for

adolescents is below the established health guideline of 0.05 mg/kg/day (USEPA IRIS). The health guideline is the USEPA's RfD, which is based on a human study that indicated central nervous system effects based on chronic ingestion. ATSDR also compared the total estimated exposure doses for adolescents to the chronic oral LOAEL in its toxicological profile for manganese [11]. This LOAEL (0.059 mg/kg/day) is based on a human study that indicated mild neurological signs. *The incidental ingestion exposure dose for adolescents is below the RfD and the LOAEL; therefore, it is unlikely that adverse health effects will occur, because conservative assumptions were made in calculating doses.* No studies were located regarding tissue distribution of manganese to use for comparison.

As previously cited, manganese is not classifiable as to human carcinogenicity. Existing studies are inadequate to determine its carcinogenicity [11].

Methyl Isobutyl Ketone (MIEBK) a.k.a. 4-methyl-2-pentanone

ATSDR calculated an incidental ingestion dose for adolescents using the maximum concentration of MIEBK found in sediment (0.0056 mg/kg). The estimated exposure dose for an adolescent is 0.000000001 mg/kg/day. The estimated dose for adolescents is below the health guideline of 0.08 mg/kg/day. The health guideline is a former USEPA Reference Dose (RfD). This oral RfD for MIBK was withdrawn in 1991. The health effects data for MIBK were reviewed by USEPA at that time and determined to be inadequate for derivation of an oral RfD. *Because the calculated doses are below the former RfD, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from MIEBK would be observed from dermal contact with sediment.* No human or animal studies were found regarding dermal exposure to MIEBK.

Data are inadequate to determine the carcinogenicity of MIEBK. No data were located regarding the existence of an association between cancer and MIBK exposure in humans, but studies of the in vivo[§§] and in vitro[***] genotoxicity of MIBK overwhelmingly provided negative responses [46].

Polycyclic Aromatic Hydrocarbons (PAH's)
[Acenaphthylene, Benzo(a)pyrene, Benzo(b)flouranthene, Benzo(g,h,i)perylene, Dibenz(a,h)anthracene, Phenanthrene]

ATSDR calculated an incidental ingestion dose using the maximum concentration of acenaphthylene, benzo(a)pyrene, benzo(b)flouranthene, benzo(g,h,i)perylene, dibenz(a,h)anthracene, and phenanthrene found in sediment. The estimated total exposure dose for adolescents is 0.000001 mg/kg/day . There are no acute or chronic MRLs derived for PAH's because there are no adequate human or animal dose-response data available that identify threshold levels for appropriate noncancer health effects. Because there are no CV's, ATSDR compared the total estimated dose for adolescents and children to the LOAEL of 120 mg/kg/day in its toxicological profile for PAH's [14]. This LOAEL is based on hematopoetic[†††] effects in female mice that were exposed to benzo(a)pyrene for six months. *Because the calculated exposure doses are thousands of times below the LOAEL, and because conservative assumptions*

§§ in vivo is defined as *within a living organism; "in vivo techniques"*
*** in vitro is defined as *in an artificial environment outside the living organism; "in vitro fertilization*
††† Hematopoiesis is the formation of blood or blood cells in the living body.

were made in calculating doses, it is unlikely that adverse effects would be observed. No studies were located regarding the distribution of PAH's in humans following dermal exposure.

Presently, a quantitative risk estimate has only been developed for benzo(a)pyrene. It is a known animal carcinogen and considered a probable human carcinogen by the IARC and the USEPA [14]. The USEPA has calculated a cancer unity risk factor for benzo(a)pyrene which ATSDR used to estimate the probability of excess cancer risk for a lifetime of exposure. Because there is no cancer unity risk factor for the other PAH's, USEPA and others developed TEF's, based on relative potency to benzo(a)pyrene to estimate carcinogenic risk [14]. ATSDR calculated an excess lifetime theoretical cancer risk for acenaphthylene, benzo(a)pyrene, benzo(b)flouranthene, benzo(g,h,i)perylene, dibenz(a,h)anthracene, and phenanthrene. The combined estimated theoretical cancer risk of 1.63E -06 is based on the maximum concentration of each of the PAH's found in sediment. This estimate indicates a low increased theoretical cancer risk from PAH's. ATSDR also compared the total estimated adult oral exposure dose to the CEL in its toxicological profile for PAH's and found that it was thousands of times lower than the CEL (2.6 mg/kg/day) [14]. *Therefore, it is unlikely that there is a significant increased risk of cancer from PAH exposure to sediment at this site.*

Polychlorinated Biphenyls (PCB's)
[Aroclor 1260, Aroclor 1248]

ATSDR calculated incidental ingestion exposure doses for an adolescent using the maximum concentrations of Aroclor 1260 and Aroclor 1248 found in sediment, 0.340 mg/kg and 0.43 mg/kg respectively. The combined estimated exposure dose for an adolescent is 0.0000002 mg/kg/day. The estimated exposure dose is below the health guideline of 0.00002 mg/kg/day. The health guideline is USEPA's RfD and ATSDR's MRL. ATSDR also compared the estimated doses to the LOAEL for PCB's in its toxicological profile [13]. The estimated dose was thousands of times lower than the LOAEL (0.005 mg/kg/day), which is based on inflammation of the tarsal glands, nails, and nail beds in female infant rhesus monkeys after 72 months of exposure to Aroclor 1254 [13]. *Because the calculated exposure doses are well below the RfD and LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed.* ATSDR calculated an exposure dose of 0.00000005 mg/kg/day for an adolescent for dermal contact to PCB's in sediment. The estimated dose is thousands of times lower than the LOAEL (42.1 mg/kg/day) for dermal effects in ATSDR's PCB toxicological profile [13]. This LOAEL is based on a rabbit study that caused hyperkeratosis, acne, and decreased body weight after approximately 38 days of exposure. *Because the calculated doses is well below the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects from PCB's would be observed from dermal contact with sediment.*

A few studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer [13]. The DHHS has concluded that PCBs may reasonably be anticipated to be carcinogens. The USEPA and the IARC have determined that PCBs are probably carcinogenic to humans. ATSDR calculated an excess lifetime theoretical cancer risk for an adult of 1.45E-07 for sediment exposure to PCB's using the maximum concentrations in sediment (0.340 mg/kg and 0.43 mg/kg). *Because the estimated risk is below 1.00E -06 and because these assumptions are very conservative, it is unlikely that there is a*

significant increased risk of cancer from PCB's due to sediment exposure.

Thallium

ATSDR calculated an incidental ingestion dose using the maximum concentration of thallium, 0.11 mg/kg, found in sediment. The estimated exposure dose for an adolescent is 0.00000003 mg/kg/day. The estimated dose is well below the USEPA RfD of 0.00007 mg/kg/day. In addition, ATSDR compared the estimated doses to the intermediate LOAEL in its toxicological profile for thallium [16]. The estimated exposure dose is thousands of times below the LOAEL (0.7 mg/kg/day), which is based on a study that produced reproductive effects (histological alteration of testis) in rats that were exposed to thallium for thirty to sixty days by gavage[‡‡‡]. *Because the calculated exposure dose is thousands of times below the RfD and the LOAEL, and because conservative assumptions were made in calculating doses, it is unlikely that adverse effects would be observed.* No reliable quantitative studies were located regarding absorption in humans or animals after dermal exposure to thallium.

Thallium is not classifiable as to human carcinogenicity. Existing studies are inadequate to determine its carcinogenicity [16].

Pathways Considered and Eliminated

Fish

Although substantial effort was expended to collect fish of edible size from Sinking Pond, only minnows (Common Shiners) were obtained. Dieldrin, Arsenic, Lead, and Iron were detected at levels slightly above comparison values in fish tissue from the minnows; however, these types of minnows are not commonly eaten by people [Table 8]. One small Brown Bullhead was observed in the pond, but was not collected [37]. While people have access to this pond, it appears unlikely that fish of edible size are likely to be caught. Also, no subsistence fish-consuming population has been identified for this area. *This exposure pathway is currently considered to be incomplete and poses no public health hazard.* If future conditions at this pond change, due to fish stocking programs, or people report collecting fish of edible size, then we recommend that suitable samples be collected, analyzed and evaluated.

In 1994, MDPH issued a statewide advisory recommending that women should refrain from consumption of freshwater fish while they are pregnant. In 2001, MDPH expanded this advisory to include women of childbearing age who may become pregnant, nursing mothers, and children less than 12 years of age. This advisory does not apply to fish stocked in freshwater lakes or ponds. More information can be obtained by calling (617). 624.5757 or by visiting the MDPH website http://www.state.ma.us/dph/beha/beha.htm. None of the waterbodies associated with this site are specifically listed on the freshwater fish consumption advisory list posted at http://www.mass.gov/dph/beha/fishlist.htm.

Volatilization of VOC's from groundwater into Buildings

In its review of the environmental data for the W. R. Grace site ATSDR considered the possibility of the volatilization of VOCs from groundwater into homes overlying the W.R. Grace groundwater plume. Some of the contaminants in the W.R. Grace site groundwater plume are VOCs that can volatilize into vapor. This vapor can, in turn, move from the groundwater through

‡‡‡ Gavage is the introduction of material into the stomach by a tube.

soil, and eventually seep into basements and affect the indoor air. Indoor air sampling data were not available for homes in the vicinity of the W.R. Grace site groundwater plume. Therefore, ATSDR applied the USEPA's Johnson and Ettinger (1991) model to estimate possible indoor air concentrations for homes or buildings that may overly the groundwater plume.

ATSDR estimated the concentrations of benzene, 1,1-dichloroethene, 1,2-dichloroethene, 1,2-dichloropropane, and vinyl chloride in indoor air from vapor intrusion to determine whether indoor air contaminants in buildings over-lying the W.R. Grace site groundwater plume could be associated with any unhealthy effects. The maximum concentration of these VOCs in groundwater was used for these calculations. ATSDR then compared the estimated concentrations to health guidance levels, such as ATSDR's inhalation MRL's§§§, LOAEL's, or NOAEL's and to information in the toxicological literature on these VOCs. An estimated indoor air concentration below the health guidance level is not expected to cause adverse health effects. Table D-1 lists the estimated indoor air concentrations for benzene, 1,1-dichloroethene, 1,2-dichloroethene, 1,2-dichloropropane, and vinyl chloride and the available health-guidance values.

This comparison suggests that the maximum level of VOCs estimated in buildings is much lower than the level at which we would expect to see health adverse effects. ATSDR used a very health protective model and the highest values found in the groundwater to create a worst-case evaluation. As a result, *ATSDR concludes that homes or buildings above the W.R. Grace site groundwater plume should not accumulate indoor air concentrations of VOCs to levels that could pose harm to residents in the vicinity of the plume.*

Discussion

Public Health Implications

Based on ATSDR's evaluation and analysis of data for the W.R. Grace site, the primary risk occurs from exposure to arsenic and manganese. People may be exposed to arsenic and manganese through several exposure pathways, but the majority of elevated risk comes from exposure to these contaminants via soil, sediment, and surface water through incidental ingestion and dermal contact.

Comparison to Background Concentrations

To assist with determining background concentrations, USEPA and the Potentially Responsible Party chose a reference area for Sinking Pond to compare measurements of chemical concentrations. The reference area, called White Pond, is similar to Sinking Pond in that it is a kettle pond with no inlets or outlets, and it stratifies during the summer. A southwest cove in White Pond has organic sediments that are visually similar to those found in Sinking Pond. The average concentrations for arsenic and manganese in Sinking Pond were 471.4 mg/kg and 11,110 mg/kg, respectively. The average concentrations for arsenic and manganese in White Pond were 16.6 mg/kg and 125 mg/kg, respectively [37]. These samples indicate that the average concentrations of arsenic and manganese found on-site in Sinking Pond exceed the average concentrations in the reference pond.

§§§ The MRL is an amount of a contaminant in the air that is not expected to cause adverse health effects.

Arsenic

Arsenic is an element that occurs naturally in rock and soil in many areas. It has been used commercially in products such as wood preservatives and pesticides. Studies conducted in other countries found harmful effects in persons who regularly drank water containing arsenic at 100 parts per billion (ppb) to 300 ppb. Compared with other groups, more of these people developed several kinds of cancer (lung, liver, kidney, and prostate) and had darkening skin, thickening of the skin on the palms of their hands and soles of their feet, skin cancer, and many small warts or corns [4].

A few studies found no harmful effects in persons in the United States who throughout their lifetimes drank water containing arsenic at levels of 50 ppb to 100 ppb [4]. Even though harmful effects were not found in persons who drank water containing arsenic levels of 50 ppb to 100 ppb, reducing exposure to arsenic can reduce harmful health effects.

As discussed in the previous exposure pathways section, the estimated incidental ingestion dose for an adolescent slightly exceeds the USEPA RfD, but because very conservative assumptions were used and it is unlikely that an adolescent would actually come into contact with or ingest sediment from these areas as frequently as ATSDR estimated, adverse health effects are unlikely.

ATSDR also calculated a cumulative excess lifetime theoretical cancer risk for adults from arsenic exposure through (1) ingestion of municipal and private well water, (2) incidental ingestion of sediment, (3) dermal contact with sediment, (4) incidental ingestion of surface water, and (5) dermal contact with surface water of 1.37E -03 (Table 9). Because the combined estimated risk level exceeds the generally acceptable range of 1.00E -04 to 1.00E -06 there is an elevated excess lifetime theoretical cancer risk for trespassers if they frequently contact or incidentally ingest sediment.

Manganese

Manganese is a naturally occurring metal found in rocks. It has also been used in pesticides (maneb and mancozeb), as an additive in gasoline, and can be produced from the burning of fossil fuels. While manganese is an essential nutrient required for normal body function, too much manganese may result in illness. Conflicting human studies are available regarding the potential health effects of manganese exposure from ingestion. Most information regarding exposure via ingestion to manganese has been derived from animal studies. Studies in animals have shown that very high concentrations of manganese in food and water may result in neurological effects [11].

As previously discussed, the main pathway of exposure to manganese at this site is through incidental ingestion of sediment. *Based on the ATSDR estimated dose for an adolescent exposure scenario, adverse health effects are unlikely.*

Trichloroethylene (TCE)

TCE is a nonflammable, colorless liquid with a somewhat sweet odor and a sweet, burning taste. It is used mainly as a solvent to remove grease from metal parts, but it is also an ingredient in adhesives, paint removers, typewriter correction fluids, and spot removers. Trichloroethylene dissolves a little in water, but it can remain in groundwater for a long time [17].

TCE is considered to be a breakdown product of perchoroethylene (PCE), and the two are found in conjunction at many hazardous waste sites. Therefore, many of the studies pertaining to potential health effects discuss both PCE and TCE [17]. Based on ATSDR's evaluation of historical data from past exposure to TCE in the Assabet wells, people may have been exposed to TCE at levels that slightly exceed the health guideline. However, because conservative assumptions were used in calculating the exposure doses and because the possible duration of exposure was limited (9 years), it is unlikely that health effects would be observed.

To address community concerns regarding past exposure to TCE, ATSDR has reviewed and summarized the results of several human health studies related to PCE and TCE in the following paragraphs. In general, the levels of TCE found in the Assabet wells were *much lower* than those reported in these studies, and the estimated length of exposure to TCE in the Assabet wells was generally shorter than the duration of exposure in these studies.

The results of several studies of human exposure to PCE and TCE in the drinking water supply have been reviewed. The studies provide some information on the cancer and non-cancer health effects associated with exposure to humans. These studies are discussed in the following paragraphs.

A study was conducted at the U.S. Marine Corps Base Camp LeJeune in North Carolina that examined adverse pregnancy outcomes associated with exposure to VOCs, including PCE and TCE, in the drinking water supply. Although the actual concentrations that women were exposed to and the duration of their exposure are unknown, concentrations of PCE and TCE in the water supply may have been as high as 215 µg/L and 1,400 µg/L, respectively. Estimates indicate that exposure may have occurred for over 25 years. The study concluded that women over 35 years of age that were exposed to PCE and TCE were 4 times more likely to have infants that were small for gestation age. Only a slightly increased incidence was observed among the entire study group of women [19]. ATSDR is currently conducting a more extensive study of this community [20].

Residents of Woburn, Massachusetts were exposed to drinking water contaminated with VOCs, including PCE at concentrations of 21 µg/L and TCE at concentrations of 250 µg/L or greater. The findings of one of the studies of this community indicated that developmental anomalies related to the central nervous system, chromosomes, and oral cleft were associated with exposure [31]. However, the scientific community has noted limitations with this study regarding the biological relevance of grouping these anomalies for the purpose of statistical analysis. As a result, the findings of this study are difficult to interpret and apply to other exposed individuals [15]. Studies of the exposed individuals in Woburn have also reported an association between exposure to contaminated drinking water and an increased risk of childhood leukemia [30,31,39]. Numerous studies have evaluated the findings of the Woburn study and have identified several shortcomings (for example, lack of information on exposure doses, duration of exposure, and the fact that several of the leukemia cases reported in the study did not have access to the contaminated drinking water) [15,32,40,42,43,49].

In response to an increased incidence of childhood cancer in Dover Township, New Jersey, a study evaluated potential community exposures to toxic chemicals in the environment. Chemicals, which include PCE, TCE, and various other contaminants, were detected in the area's drinking water supply. In addition, contaminants were also likely to be present in the air as a result of emissions from the nearby hazardous waste sites. The results of the study indicate an

increased risk of leukemia among young females via prenatal exposure to contaminants in the drinking water supply and exposure to contaminants present in the air. Several uncertainties exist regarding this study, such as the potential exposure to various contaminants, limited available groundwater and air data, and lack of information on exposure doses and duration [38].

ATSDR reviewed another study of birth outcomes in 75 towns in New Jersey, where individuals ingested drinking water contaminated with VOCs, including PCE and TCE [23]. The study concluded that oral cleft defects were elevated. Several limitations have been identified in the study, such as confounders (smoking, additional occupational exposures, medical history), which do not allow for the results of this study alone to conclude definitively on the association between developmental effects and exposure [15]. A review of the available scientific data indicates that further study of the likelihood of the contaminants to result in developmental effects is necessary. Another study of these New Jersey communities reported an increased incidence of childhood leukemia and non-Hodgkin's lymphoma in females exposed to greater than 5.0 ug/L of TCE in drinking water [26]. Limitations of the studies include lack of information regarding contaminant levels, exposure duration, and water consumption variations among individuals.

A review of the available studies of PCE exposure via inhalation indicates that PCE and TCE has been associated with liver, kidney, neurological, developmental effects, and leukemia among subjects exposed to very high concentrations [15,17]. Studies have reported skin effects, such as chemical burns and blistering from prolonged exposure to concentrated PCE and TCE used in dry cleaning operations [29,17]. Exposure to PCE and TCE via inhalation and dermal contact is expected to be significantly lower than among study subjects. Although ingestion is the major route of exposure, individuals may also have been exposed to PCE and TCE via inhalation and dermal contact during showering.

ATSDR maintains the TCE exposure subregistry, which compiles health-related information from the nearly 5,000 participants that have been exposed to TCE from contaminated drinking water. The participants were exposed to TCE concentrations ranging from 2.0 µg/L to 24,000 µg/L (or 0.002 mg/L to 24 mg/L) for up to 18 years. Studies of this population indicate that individuals exposed to TCE may have an increased incidence of health effects, particularly stroke. Other health effects reported among participants of the TCE subregistry include anemia, urinary tract disorders, liver and kidney effects, diabetes, and skin conditions. Based on the available information, a cause-effect relationship has not been demonstrated between TCE exposure and the reported health effects. Studies of the participants of the subregistry and the potential for health effects related to TCE exposure are ongoing [1].

Child Health Considerations

ATSDR recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their water, soil, air, or food. Infants and children are usually more susceptible to toxic substances than adults due to their immature and developing organs. Children are smaller, which results in higher doses when compared with adults. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care. ATSDR's evaluation in this document considered children as a susceptible subpopulation. The possible risks for children

exposed to contaminants at the W.R. Grace site are discussed in the previous section under Public Health Implications.

Health Outcome Data

Health outcome data (HOD) record certain health conditions that occur in populations. These data can provide information on the general health of communities living near a hazardous waste site. They can also provide information on patterns of specified health conditions. Some examples of health outcome databases are tumor registries, birth defects registries, and vital statistics. Information from local hospitals and other health care providers can also be used to investigate patterns of disease in a specific population. ATSDR looks at appropriate and available health outcome data when there is a completed exposure pathway or community concern.

ATSDR requested that the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Center for Environmental Health conduct an evaluation of cancer incidence in Acton, Massachusetts. The evaluation was initiated based on community concerns about cancer and possible environmental exposures in relation to the W.R. Grace Site. The cancer incidence assessment was completed under cooperative agreement with ATSDR.

The purpose of the assessment of cancer incidence was to provide a review of the pattern of cancer in the town of Acton and census tracts within the town through comparison of the incidence of six cancer types with the incidence of those cancers in the state of Massachusetts as a whole. Additionally, available information about risk factors related to the development of cancer was evaluated. A qualitative evaluation of the geographic pattern of cancer diagnoses was also conducted for the town and its census tracts.

Cancer incidence data for the years 1982–2000 were obtained for the town of Acton from the Massachusetts Cancer Registry (MCR). Six cancer types were evaluated, including cancers of the bladder, brain and central nervous system (CNS), kidney, liver, and lung and bronchus, as well as leukemia. These cancer types were selected for evaluation based on knowledge of community cancer and environmental concerns and/or potential associations with contaminants of concern at the W.R. Grace site. To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–2000.

With one exception, the six cancer types evaluated occurred approximately at the expected rates in the town of Acton as a whole during the 19-year time period 1982–2000, as well as during the three smaller time periods. Brain and CNS cancer occurred more often than expected during 1982–2000 and two smaller time periods, 1982–1987 and 1988–1993. The observed elevation was statistically significant during the first time period, 1982–1987 (14 diagnoses observed vs. 6.9 expected). Lung and bronchus cancer occurred statistically significantly less often than expected among males and among both sexes combined during 1982–2000. Bladder cancer and kidney cancer occurred less often than expected, and leukemia and liver cancer occurred about as expected during 1982–2000. In general, most Acton census tracts experienced cancer approximately near the rates expected during 1982–2000 and during the smaller time periods evaluated in the report.

Available risk factor information for individuals diagnosed with cancer in Acton was compared to known or established trends to assess whether any unexpected patterns existed in the town. In general, trends observed in Acton were similar to those seen in the general population. Review of this data suggests that smoking likely played some role in the diagnosis of some cancer types (e.g., cancers of the bladder, kidney, and lung and bronchus) among some individuals in Acton. Also, occupational exposures may have been important in the development of cancer among some individuals.

For the majority of cancer types evaluated, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer in Acton revealed no apparent spatial patterns at the neighborhood level that are not likely to be attributed to factors such as areas of higher population density. While diagnoses of brain and CNS cancer were fairly evenly distributed throughout the town and seemed to coincide closely with the pattern of population, there were a few locations in Acton where two or three individuals with this cancer type were located in relatively close proximity to each other, with some located in the southern area of town near the W.R. Grace site. However, an analysis of the dates of diagnosis did not suggest any temporal trends among these individuals, and a variety of cell types of brain and CNS cancer were represented among children and adults, indicating that a common factor among individuals in these locations was unlikely.

In summary, based on the information reviewed in this evaluation, it does not appear that environmental exposures played a major role in the overall incidence of cancer in the Town of Acton as a whole or in relation to the W. R. Grace site during the 19-year time period 1982–2000.

MDPH's complete report on the Assessment of Cancer Incidence in Acton is attached as Appendix G.

Community Health Concerns

The ATSDR site team held a public availability session on Tuesday October 28, 2003 from 7:00pm to 9:00pm at the Acton Memorial Library, 486 Main Street, Acton, Massachusetts. Some of the community health concerns below were gathered at the meeting, and some were submitted to ATSDR in writing afterward.

Is the municipal drinking water supply safe? Are the municipal drinking water supply wells monitored?

ATSDR response: Municipal drinking water supplied by the Acton Water District (AWD) currently meets or exceeds all the requirements of the Safe Drinking Water Act. The AWD operates and maintains air strippers that remove volatile organic compounds that may be present in groundwater pumped from the town wells. The AWD routinely samples and treats the water provided to users to ensure a safe water supply is provided.

Was the well water actually sampled for vinyl chloride?

Table C-12 of the Initial Release ATSDR Public Health Assessment for the site lists "NA" (not analyzed?) under the category of vinyl chloride for Assabet 2 for 1978 and 1979. (The only other data provided for Assabet 2 in this report are from 1992. Data are provided for Assabet 1 for 1992 only—no data from 1978 or 1979.)

ATSDR response: ATSDR reviewed the November 4, 1992, initial release Public Health Assessment for the W.R. Grace Site, Acton, MA, completed by the Massachusetts Department of Public Health. It appears that data are "not available" or were not "not analyzed" in 1978 and 1979. Without access to the original sampling data it is not possible for ATSDR to determine why there is no data from 1978 or 1979. Samples may not have been collected for vinyl chloride at that time or they may have been collected, but not analyzed due to poor quality samples, contamination in the samples or a multitude of other reasons.

Even if the well had been sampled, couldn't the lack of vinyl chloride detection in the well water in the late 1970s to early 1980s be due to technological (sampling) limitations, rather than the actual absence of vinyl chloride in the drinking water?

*It is ACES**** understanding that routine VOC testing of drinking water supplies by certified labs was not required until after 1985. It is also our understanding that vinyl chloride was regulated differently than other VOCs due in part to the difficulty in obtaining accurate sampling results. Drinking water sampling for vinyl chloride, a "known human carcinogen of high potency" was not required for all systems, and sampling errors for vinyl chloride in drinking water, before 1987 could be plus or minus 40 percent. (See Federal Register, Vol. 50. No. 219 Nov. 13, 1985/ Proposed Rules and Federal Register Vol. 52, No. 130 July 8, 1987/ Rules and Regulations.)*

ATSDR response: Yes, it is possible that vinyl chloride was not detected due to technological sampling limitations in the late 1970's and early 1980's.

Given these considerations it seems appropriate that ATSDR consider that it was possible that individuals drinking Acton public water from the contaminated wells, between 1970 and 1978, did ingest vinyl chloride, and that vinyl chloride induced health effects could have occurred. (Exposure routes could have included ingestion, inhalation, and dermal exposure.)

ATSDR response: ATSDR has evaluated exposure to vinyl chloride in this Public Health Assessment based on the maximum concentration of vinyl chloride found in a private well. ATSDR considered incidental ingestion and dermal contact with private well water in its exposure assessment. ATSDR also estimated an excess lifetime theoretical cancer risk for an adult based on a 70 year exposure to the maximum concentration found in the private well. Because there is no historical vinyl chloride data available for the Assabet wells, ATSDR is unable to model past exposure. It is possible that persons receiving drinking water from the Assabet wells between 1970 and 1978 could have been exposed to vinyl chloride; however, ATSDR has no data that indicates there was vinyl chloride contamination in the wells during that period.

Was the well water actually sampled for acrylonitrile? What other data are available for acrylonitrile at the W.R. Grace Site? Many of the Tables in the 1992 ATSDR report show data

**** Acton Citizens for Environmental Safety (ACES)

gaps for acrylonitrile. According to these Tables, while 1700 ppb of acrylonitrile was detected in the groundwater under the Secondary Lagoon, acrylonitrile was NA (not analyzed), in the groundwater beneath the Primary Lagoon, North Lagoon, Blowdown Pit, and Battery Separator Area.

ATSDR response: In the 1992 Initial Release Public Health Assessment [44] acrylonitrile was sampled for in groundwater down-gradient from the landfill, groundwater and soil below the waste sites, groundwater and soil below the lagoons, sludge in the landfill, soil below the landfill and in surface water. However it does not appear that acrylonitrile was sample for in the Assabet wells. Of the locations sampled for acrylonitrile it was found only in groundwater below the secondary lagoon. Acrylonitrile was not found in more recent sampling of groundwater and surface water at the W.R. Grace Site most likely because it rapidly breaks down and disappears from water. ATSDR contacted the Massachusetts Department of Public Health to see if the data from the 1992 Initial Release PHA could be located for review to better interpret the data gaps, however these data could not be located.

How sensitive/accurate were sampling techniques for acrylonitrile in the late 1970's to mid 1980's? Could mid to low levels of acrylonitrile have gone undetected due to technological limitations at that time?
Given:

- that VOC testing of drinking water supplies by certified labs was not required until after 1985,

- acrylonitrile is not routinely included in VOC sampling rounds,

- the detection of 1700 ppb of acrylonitrile in groundwater onsite, (between 1979 and 1982),

- USEPA's limit of 0.058 ppb for surface water, because of public health concerns about ingestion of acrylonitrile, and

- that ATSDR's report states that "Acrylonitrile exposure occurred via ingestion, inhalation and dermal contact for residents who received water from Assabet One and Two"

It seems appropriate that ATSDR consider that individuals drinking Acton public water from the contaminated wells, between 1970 and 1978, may have been exposed to acrylonitrile and that the dose may have been at a high enough level to have caused health effects. (Exposure routes could have included ingestion, inhalation, and dermal exposure.)

ATSDR response: The limits of detection for most chemicals, including VOC's, have improved since the 1970's and 1980's. It is possible that low levels of acrylonitrile could have gone undetected due to technological limitations during this time period, and it is possible that persons receiving drinking water from the Assabet wells between 1970 and 1978 could have been exposed to acrylonitrile; however, ATSDR has no data that indicates there was acrylonitrile contamination in the wells during that period. Because there is no acrylonitrile data for time period, it is not possible to calculate an estimated exposure dose.

Could exposure to contaminants at this site contribute to squamous cell carcinoma of the throat?

ATSDR response: ATSDR worked with the Massachusetts Department of Public Health to select and evaluate cancer types based on their potential association with contaminants of concern identified at the W.R. Grace site. The six cancer types evaluated include: cancers of the bladder, brain and central nervous system, kidney, liver, lung and bronchus, and leukemia.

Cancers of the throat area, specifically cancers of the oral cavity, oropharynx, larynx, or esophagus are not thought to be carcinogenic health outcomes potentially associated with the contaminants of concern at this site. In general, the primary causes for cancers that occur in the throat area are tobacco and alcohol use [21,22]. ATSDR did conclude in this PHA that there is potential elevated carcinogenic risk from arsenic, primarily through incidental ingestion and dermal contact with surface water and sediment; however, it would be very difficult to determine if site related exposure may or may not have contributed to squamous cell carcinoma of the throat.

Are there birth defects related to contaminant exposures at this site?

ATSDR response: ATSDR found no specific birth defects related to contaminant exposures at this site. However, ATSDR has concluded that past exposure to trichloroethylene (TCE) may have slightly exceeded the health guideline between 1970 and 1978, but adverse health effects are not likely based on past exposure to TCE in drinking water at this site, because very conservative assumptions were used in the assessment and the duration of exposure was limited. ATSDR refers to some studies related to TCE exposure in the PHA, because these studies suggest that birth defects may occur when mothers drink water containing TCE during pregnancy. In general the levels of TCE found in the Assabet wells were much lower than those reported in these studies, and the estimated length of exposure to TCE in the Assabet wells was generally shorter than the duration of exposure in these studies. Therefore, birth defects related to TCE exposure at this site are unlikely.

Can benzene exposure lead to cancer?

ATSDR response: The Department of Health and Human Services has determined that benzene is a known human carcinogen. Long-term exposure to high levels of benzene in the air can cause leukemia, cancer of the blood-forming organs. Benzene was found above ATSDR comparison values in some groundwater samples from monitoring wells and in the historical data from Assabet wells One and Two. ATSDR estimated a lifetime excess cancer risk based on the highest concentration of benzene found in the Assabet wells (0.0021 mg/L) and a maximum exposure duration of nine years. Based on this information there is a very low increased theoretical cancer risk (4.75E-06). Therefore, ATSDR has concluded that it is unlikely there is a significant increased risk of cancer due to ingestion of benzene from the Assabet wells in the past.

Is there a cancer cluster in the Acton area?

ATSDR response: The Community Assessment Program of the Massachusetts Department of Health, Center for Environmental Health conducted an evaluation of cancer incidence in Acton, Massachusetts. Based on the information reviewed in the assessment there does not appear to be any unusual geographic pattern of individuals diagnosed with the six cancer types selected for evaluation based on contaminants of concern identified at the W.R. Grace site. The MDPH made the following conclusions in its assessment:

- In general, the six cancer types (cancers of the bladder, brain and central nervous system, kidney, liver, lung and bronchus and leukemia) evaluated occurred approximately at or near the expected rates for Acton and its individual census tracts during the 19-year time period 1982-2000.

- Review of the geographic distribution of individuals diagnosed with cancer in Acton revealed no apparent spatial patterns at the neighborhood level that would suggest a common factor (environmental or non-environmental) played a primary role in the incidence of cancer.
- Review of available risk factor information for individuals diagnosed with cancer (i.e. age, gender, smoking history, and occupation) suggests that trends observed in Acton are similar to those seen in the general population. This information suggests that smoking and, to a lesser extent, occupation, likely played some role in the incidence of some cancer types in Acton.

Conclusions

Based on the data evaluated, ATSDR has assigned a public health hazard category for each of the pathways evaluated in this PHA. Appendix D presents a description of each of the public health hazard categories that were considered during the classification process.

- Individuals supplied water by the AWD's Assabet One and Assabet Two wells between 1970 and 1978 may have previously drank, bathed, and showered with groundwater contaminated with VOC's. The historical data for this time period is very limited and there is no QA/ QC information available for the data, however, to address community concerns regarding past exposure the data was evaluated. *ATSDR concludes that past levels of VOC's in the Assabet wells posed no apparent public health hazard; however, levels of TCE may have exceeded current health guidelines, but because of the short duration of exposure (maximum 9 years) and the relatively low levels compared to potential effects in health studies, it is unlikely that adverse health effects have occurred.* There is no historical data from this period for manganese or arsenic in the Assabet wells. Therefore, *ATSDR considers past exposure to arsenic and manganese in the municipal drinking water supply to be an indeterminate public health hazard.* Since the early 1980's, the AWD has been treating the groundwater through an air stripping process to remove VOC contamination from the active water supply wells. Therefore, there is no current or expected future exposure to VOC's in the municipal drinking water supply. While recent sampling has indicated no VOC contamination (due the treatment process), it has indicated the presence of arsenic and manganese. Based upon the concentrations reported for the wells and toxicological evaluations, adverse health effects are not expected to occur. Therefore, *ATSDR considers current and future exposure to VOC's, arsenic, and manganese in the municipal drinking supply to be a no apparent public health hazard.*
- Six private irrigation wells have been identified in the vicinity of the W.R. Grace site and are used for non-drinking water purposes. One well contained vinyl chloride above the CV, four wells indicate no present VOC contamination, and one well has not been sampled. The un-sampled well was properly sealed and permanently closed per the request of the property owner. Potential exposures to the contaminated well are limited to possible dermal contact during swimming in pools that were filled with water and irrigation activities. Based on the concentrations reported for the well and toxicological evaluations, adverse health effects are not expected to occur. Therefore, *ATSDR*

concludes that exposure to groundwater from existing private irrigation wells for non-drinking water uses poses no apparent public health hazard.

- Based on the data reviewed, trespassers accessing the W.R. Grace site may have been exposed in the past, may be presently exposed, and may be exposed in the future to contaminants in soil, sediment and surface water. Of the contaminants evaluated in soil, sediment, and surface water, only arsenic indicates elevated risk. The evaluation of potential health effects from exposure to contaminants in sediment and surface water, conducted in this PHA, incorporated conservative assumptions for health effects, which may have resulted in an overestimation of potential exposure to individuals. However, *ATSDR concludes that trespassers exposed to the highest levels of arsenic in sediment and surface water from Sinking Pond and the North Lagoon Wetland may have a slightly elevated risk for adverse health effects. There is a moderate elevated excess lifetime theoretical cancer risk for trespassers who frequent the site and come into contact with or incidentally ingest the highest levels of contaminated sediment over a lifetime.*
- Due to elevated concentrations of VOC's present in shallow groundwater, it is appropriate to consider whether there may be health effects related to the possible migration of contaminants from groundwater to the soil and into indoor air in residences and other buildings overlying the plume near the W.R. Grace site. Based on modeled concentrations of contaminants, no adverse health effects are expected to occur. Therefore, *ATSDR concludes that this pathway is classified as posing no apparent public health hazard.*
- Based on the information provided to ATSDR, edible size fish from Sinking Pond could not be caught after several attempts. Because edible size fish could not be caught for evaluation, it is unlikely that persons accessing the W.R. Grace site are catching and consuming fish from Sinking Pond on a regular basis. If an occasional edible size fish were caught and consumed it is not likely that this infrequent exposure would be sufficient to cause adverse health effects. Therefore, *ATSDR considers fish consumption from Sinking Pond to be a no apparent public health hazard.*

Recommendations

- ATSDR recommends continued monitoring of the Assabet, Scribner, Lawsbrook, and Christofferson wells, by the Acton Water District, to ensure that the air strippers are adequately removing VOC contamination and that the municipal drinking water supply meets all the requirements of the Safe Drinking Water Act.
- Consistent with USEPA's clean up plan in the 2005 Record of Decision for the site, ATSDR recommends addressing the areas of the site with elevated levels of arsenic and manganese (i.e. Sinking Pond, North Lagoon Wetland) in sediment in a manner that would be protective of public health.
- The five identified active private irrigation wells in the area of the W.R. Grace site that are used for non-drinking water purposes should be monitored periodically by W.R. Grace to determine whether the levels of contaminants are of public health significance.
- It is recommended that no new private wells be installed in the vicinity of the groundwater plume near the W.R. Grace site. This should be accomplished through

institutional controls, local town ordinance and/ or deed restrictions. These controls are currently under consideration and will be put in place in the near future.

Public Health Action Plan

A public health action plan describes the actions designed to mitigate or prevent adverse human health effects that might result from exposure to hazardous substances associated with site contamination. The following paragraphs summarize the public health actions that have been taken at the W.R. Grace Site and the actions that are to be taken.

Actions Taken

- ATSDR conducted an initial site visit to the W.R. Grace site in July 2003.
- ATSDR conducted a public availability session to gather community concerns and discuss ATSDR's role in the Superfund process in October 2003.
- ATSDR accepted comments from ACES during the Initial release of the PHA and has incorporated responses to those comments in this version of the PHA.

Actions to Be Completed

- ATSDR will receive written comments on this draft W.R. Grace site PHA from the public for 30 days following the release of this document.
- ATSDR plans to follow-up with the community following the release of the draft and conduct an availability session in the summer of 2008.
- ATSDR expects to release the final PHA in fall 2008.

Responses to Comments Received

The Acton Citizens for Environmental Safety (ACES) community group has had a long history of active involvement with the W.R. Grace site in Acton, MA. The ACES group has been actively involved in working with the state, federal and other public health agencies. ATSDR provided its initial release PHA for ACES review. ACES prepared and submitted comments to ATSDR, which are addressed in this section.

Comment #1:
Evaluation of past exposures?
Summary, page iv
ACES and other members of the Acton community are looking to ATSDR to help answer some of the questions that will not be addressed under EPA's Superfund process as EPA looks at current and future risk at the WR Grace Superfund Site in Acton and Concord. These unanswered questions have to do with past exposures at the WR Grace Superfund Site, and they include:

a. What are the cumulative health risks (over the past 27 plus years), due to **past**, as well as present exposures to organic and inorganic contaminants from the WR Grace Site?

b. Have there been/could there be any adverse health effects, including increased cancer incidence, cancer mortality, or other health effects as a result of **past** plus present exposure to site contaminants?

EPA's Risk Assessment process does not take into account past exposures to site contaminants.

* Would ATSDR please do a comprehensive public health assessment that accounts for the past exposures that individuals may have had to site contaminants over many years, in addition to current potential exposure? *

For example, please consider the cumulative risk to an individual who has used tap water from the Assabet wells from 1978, (or 1970), until the present, was exposed to onsite contaminants in soils, sludge, groundwater, and surface water, both before and after soils and sludge in source areas were remediated, and has been exposed to airborne VOCs including those released into the air by the onsite stripping tower used to remove contaminants from groundwater.

Data are available for both the Assabet public wells and for onsite source areas, and groundwater from 1978 onward, and some of the older historical data have already been summarized in ATSDR's 1992 Initial Release PHA. (See comment 2. below.) Due to active cleanup on a portion of the site there have been changing conditions over the years. Some exposure pathways that are currently incomplete, were complete in the past (ie past exposure to contaminated soils, sludge, surface water from lagoon areas, higher level of contaminants in air due to air stripper, etc.).

Simply assessing current conditions does not answer the outstanding question of cumulative health effects due to past and present exposures to multiple chemicals over many years.

ATSDR Response:
ATSDR's estimated dose calculations for non-carcinogenic risk for all pathways (ingestion, inhalation, dermal contact), are based on a 30 year exposure duration, except for past exposure to VOC's which are based on a nine year exposure period (1970-1978). Thirty years is the national upper-bound time persons may spend at one residence. The nine year exposure period used for VOC's is from the time that Assabet wells One and Two were opened until they were taken out of service in 1978. ATSDR's exposure dose calculations are conservative but realistic (see Appendix C in the PHA for additional exposure parameters used in the exposure dose calculations).

ATSDR's calculations for theoretical excess cancer risk are based on a 70 year exposure duration, except for past exposure to VOC's which are based on a nine year exposure period (1970-1978). Seventy years is generally accepted as a lifetime exposure. ATSDR's theoretical excess cancer risk is conservative and would encompass the potential exposure period (see Appendix C in the PHA for additional exposure parameters used in the calculations for theoretical excess cancer risk).

ATSDR indicates in the Public Health Assessment that there is very limited historical data for the Town of Acton municipal wells for the time period of 1970 to 1978 (the year the wells were opened until the year the VOC contamination was first identified). Even though the data is limited, ATSDR did evaluate past exposure in the health assessment to try and address this community concern.

Comment #2:
Historical data available; Important to include 1992 ATSDR reports in 2005 PHA.
Summary, page iv
There is a lack of groundwater data and data from the public wells pre-1978, but data since then are available. (Contaminant levels in the wells between 1970 and 1978 may have been similar to those detected in the public wells before the ARS went online in 1985.)

ACES has hard copies of two different versions of ATSDR's 1992 PHA. One is the Initial Release PHA, dated September 30, 2005; with a notation that the "Comments Period Ends. November 4, 1992". The first paragraph of the Summary Section of the Aug 2005 PHA refers to this report.

The other version appears to be an earlier draft of the 1992 Initial Release PHA. It is labeled "Draft: Public Comment Release PHA". (See Attachment B to these comments.)

Both reports include data tables (C-1 through C-10), of maximum concentrations of various contaminants found in groundwater and soils below or near waste sites, (including the various lagoons, and the landfill), as well as in sludge and surface water in these areas. Table C-11 of these reports includes concentrations of contaminants in ambient air during on-site pilot

excavation activities. Table C-12 lists maximum concentrations of contaminants found in drinking water wells. The "Draft: Public Comment" version summarizes data for the two Assabet public wells for each of the years from 1978 to 1987. The "Initial Release" version lists data from 1978, 1979, and 1992. See summary below of contaminant detections as presented in one or both Tables C-12. (Also see Attachments B. and C.)

Summary of Maximum Concentration of Substance identified in Assabet Wells by Year

Contaminant Concentration (ppb) by Year

	1978	**1979**	**1980**	**1981**	**1982**	**1983**	**1984**	**1985**	**1986**	**1987**	**1992**
Ethylbenzene		23									
1,1, Dichloroethylene	1.2	56	32.5		7.0	9.0	31	32	33	5.2	8
Benzene		2.1									
TCA					134	151					6.7
TCE	8.0					0.11	0.12	0.8	2.9	1.0	
Methylene Chloride		46			3.0	0.5					
1,1-Dichloroethane						3.4	7.6	24	1.9		

Note: The 1992 Draft: Public Comment PHA cites the data sources for specific data, (AWD, GZD, GZA, MA DEP, or RMC). The report does not indicate whether these concentrations are in "raw" water or treated water.

[Are additional data for the public wells also available from the Acton Water District? What concentrations of contaminants have been detected in the past at the School Street wells in the Northeast part of the site? Are additional data available for the source areas and groundwater in Operational Unit -1 (OU-1) documents, (including the OU-1 Initial Site Characterization Report), and/or in the 1997 ISCR for OU-3?]

Requests:
Given the data included in the ATSDR 1992 PHA Reports, (and any other relevant historical data that may be available), and the outstanding questions about past exposures to contaminants:

a. Please include the 1992 ATSDR Initial Release Public Health Assessment [as an appendix??], in the 2005 Public Health Assessment.

b. At a minimum, please include Tables C-1 through C-12 in the current PHA, (including all the data from both versions of Table C-12), along with a discussion of these data, and the significance of past exposures.

c. Please include any additional relevant data since 1992, in the 2005 PHA.

d. Part of the WR Grace site is in Concord. Please consider that area residents, including those from Concord, may have accessed the site and been exposed to site contaminants before, during, and after the completion of soil remediation in 1997.

e. As already stated in comment 1, please include an analysis of the historical plus current risk posed by site contaminants, considering all possible past and present exposures.

ATSDR Response:
ATSDR believes that the limited data provided in the 1992 Initial Release Public Health Assessment, prepared by the Massachusetts Department of Health, is of limited value for determining public health conclusions for past exposure. In most cases there is only one data point per year for the VOC's listed in the table above. In the case of benzene there is only one data point for the years 1978 through 1992. However, in order to address ACES concern ATSDR has evaluated the available historical data in the Public Health Assessment.

Please refer to the section entitled: Public Water Supply Wells, Historical Data, VOC's

Comment #3:
Future exposure?
Summary, Fourth paragraph, last sentence:
Please omit the word "future" in this sentence. (See comparison of EPA Risk Assessment and ATSDR Public Health Assessment, re: time frame focus---on handout from Oct. 2003 public meeting in Acton---ATSDR looks at present and past exposures, while EPA looks at present and future.)
Also, the possibility of higher concentrations of contaminants, including arsenic and manganese reaching the School Street wells in the future, cannot be ruled out, especially given EPA's proposed plan to allow the plume of contamination in the Northeast Area to continue to be pulled into the public wells. There are much higher concentrations of arsenic and manganese within this plume, upgradient of the wells, than the current detections at the public wells. The arsenic detection at the public wells, from the Remedial Investigation, already results in a 1E-04 theoretical cancer risk due to arsenic, according to WR Grace's Public Health Risk Assessment.

ATSDR Response:
ATSDR removed the word "future" in the fourth paragraph, last sentence of the Summary.

ATSDR agrees that arsenic and manganese contaminant levels could increase in the School Street wells in the future; however, these wells are being monitored by the Acton Water District (AWD) in accordance with the Safe Drinking Water Act (SDWA). If levels of contaminants in these wells rise, the AWD will take action to ensure that water distributed through the public drinking water supply does not exceed the Maximum Contaminant Level or other standards set forth in the SDWA. Therefore, it is unlikely that persons will be exposed in the future to levels of arsenic and manganese through the municipal drinking water system at levels of public health concern.

Comment #4:
Irrigation wells, additional data available
Summary, Fifth paragraph
Additional data are available for at least two irrigation wells at the WR Grace Site, showing higher detections of VOCs than were referenced in the Aug. 2005 PHA. According to a "December 4, 2002 Letter Re: Lisa Lane and Bellantoni Drive, Public Irrigation Well Evaluation Results" detections included the following:

a. Lisa Lane Well
   - VDC (1,1-Dichloroethylene) at 14 to 17 ug/L
   - Vinyl chloride at approximately 0.3 ug/L
   - Benzene at approximately 0.5 to 0.6 ug/L
   - Acetone at approximately 3 to 36 ug/L

b. Bellantoni Drive Well
   - VDC (1,1-Dichloroethylene) at 4 to 5 ug/L
   - Vinyl chloride at approximately 0.2 to 0.3 ug/L
   - Acetone at approximately 2 to 5 ug/L

No testing was done for Arsenic or other inorganics in any of the irrigation wells at the site.

(The December 4, 2002 letter is in "Appendix A of Phase 2 Remedial Investigation Data Report, May 14, 2003"; See also Attachment A of these comments and http://doc.acton-ma.gov/dsweb/Get/Document-6723/WRGracePrivateWellEvaluation.PDF)

The Lisa Lane well was in use from 1995 to 2002. It is in one of the more concentrated contours of the VDC plume. Please consider that residents may have been exposed to this water via ingestion, inhalation, and dermal exposure over approximately eight years, and that past levels of contamination in this well may have been higher than those detected in 2002.

ATSDR Response:
ATSDR reviewed the GeoTrans, Inc. letter (dated December 4, 2002) regarding concentrations of VOC's found in the Lisa Lane and Bellantoni Drive irrigation wells. As ATSDR discussed in the response to comment 2, caution must be used when using very limited data sets for determining public health conclusions for past exposure.

However, in an effort to answer ACES question regarding past exposures, based on the limited data provided in the GeoTrans, Inc letter, ATSDR did review and calculate non-carcinogenic and carcinogenic risk for benzene, vinyl chloride, and 1,1 dichloroethane (VDC) for an eight year exposure to the maximum concentration of the contaminants found in either the Lisa Lane or Bellantoni Drive irrigation wells. ATSDR did not calculate risk for acetone, because the maximum concentration of acetone was below the ATSDR comparison value.

ATSDR calculated non-carcinogenic risk for adults and children for benzene, vinyl chloride, and VDC. In its exposure scenario for these calculations, ATSDR assumed an incidental ingestion rate of 0.1 liters per day for adults and children. ATSDR assumed that incidental ingestion

occurred 350 days per year and that an adult was exposed for 8 years and that a child was exposed for 6 years. ATSDR assumed the default weights of 70 kilograms for an adult and 16 kilograms for a child. Based on the maximum concentration and the exposure parameters above the estimated exposure doses for benzene, vinyl chloride, and VDC were below established EPA or ATSDR health guidelines. ATSDR also calculated a theoretical excess carcinogenic risk for an adult exposed to benzene and vinyl chloride, based on the assumptions above, for an eight year exposure. The theoretical excess cancer risks for an adult exposed to benzene (4.92E-08) and vinyl chloride (6.26E-08) are well below the generally acceptable excess lifetime cancer risk of 1 in 1 million (1.00E-06). The theoretical excess cancer risk for VDC could not be calculated, because there is no cancer slope factor available.

In addition, ATSDR calculated non-carcinogenic risk for adults and children for benzene, vinyl chloride and VDC for dermal contact. Exposure doses were calculated using the maximum detected concentration and a surface area of 6,074 square centimeters (cm^2) for adults and 2,178 cm^2 for children. A skin adherence factor (AF) of 0.07 was used for adults and an AF of 0.2 was used for children. If a chemical absorption factor (ABS) was available it was used, if not an ABS of 1.0 was used for conservatism. An exposure frequency of 24 days per year was assumed (2 days per week during 3 summer months). It was assumed that the exposure duration was 8 years for adults and 6 years for children. A body weight of 70 kilograms (kg) for adults and 16 kg for children was also assumed. *Based on the exposure parameters listed above, the estimated exposure doses for benzene, vinyl chloride, and VDC were below established EPA or ATSDR health guidelines.* ATSDR also calculated a theoretical excess cancer risk for an adult exposed to benzene and vinyl chloride, based on the assumptions above, for an eight year exposure. The theoretical excess cancer risks for an adult exposed to benzene (1.85E-08) and vinyl chloride (2.35E-08) are well below the generally acceptable excess lifetime cancer risk of 1 in 1 million (1.00E-06). The theoretical excess cancer risk for VDC dermal exposure could not be calculated, because there is no cancer slope factor available.

Comment #5:
Irrigation wells, additional data available
Summary, Fifth paragraph
In addition to the VOCs at both the Lisa Lane and Bellantoni Drive irrigation wells, two VOC contaminants: TCE (1.2 ug/L) and MTBE (0.31 ug/L) were detected in the Powder Mill Plaza irrigation well. (See: July 2005 Public Review Draft Remedial Investigation Report; p. 2-7.) This well is still being used.

ATSDR Response:
The levels of trichloroethylene (TCE) and methyl-tertiary butyl ether (MTBE) detected in the Powder Mill Plaza irrigation well are very low. The detected level of TCE (1.2 ug/L) is below the USEPA Maximum Contaminant Level (MCL) of 5ug/L or ppb. MCL's are set at levels to ensure that drinking water does not pose either a short-term or long-term health risk. Because the detected level is well below the MCL, no adverse health effects would be expected to occur. The detected level of MTBE (0.31 ug/L) is thousands of times lower than ATSDR's comparison value of 3,000 ug/L and is therefore unlikely to cause adverse health effects.

Comment #6:
Private Irrigation wells, may be used as potable water
Summary, Fifth paragraph, first sentence
Please consider that the private irrigation wells onsite may have been used for drinking water purposes.

Once a private well is in place there is no practical way to control, or even be aware of how this domestic water source is actually used. An outdoor spigot or hose could be used to supply drinking water to people or pets. A private well could be used for water in an outdoor shower, or in a greenhouse. An uninformed future property owner could convert it to a drinking water source.

In its handbook, the Massachusetts Association of Health Boards states:

"Boards of Health may be very correct in assuming that there is no such thing as a "non-potable" well serving a residence. Even if the intention is to provide water for car washing and lawns, there is always a risk that children will drink from a hose, that it will be used to fill a swimming pool, or at a future date, it may be hooked up to the house plumbing."††††

ATSDR Response:
Homes connected to a municipal drinking water supply generally do not use private irrigation wells as a drinking water source. Irrigation wells are used for watering yards and gardens, filling swimming pools, washing cars, etc. Where applicable, ATSDR considered these types of VOC exposures in its risk calculations. ATSDR considered incidental ingestion and dermal contact in its evaluation of exposure to vinyl chloride from private irrigation wells in the public health assessment and in its evaluation of vinyl chloride, benzene and VDC (from the Lisa Lane and Bellantoni Wells) in comment 4 above. ATSDR's exposure scenario considered that a person might be exposed through incidental ingestion and dermal contact by wading or swimming in pools filled with water from these irrigation wells 1.5 hours per day for 90 days per year. For vinyl chloride ATSDR estimated 30 year and 8 year maximum exposures. For VDC and benzene, ATSDR estimated an 8 year exposure (maximum length of time people may have been exposed to contaminants in the Lisa Lane and Bellantoni Wells). Based on these conservative assumptions, all of ATSDR's calculated VOC exposure doses are well below established health guidelines for non-carcinogenic risk and the calculations for theoretical excess cancer risk are well below the generally acceptable excess lifetime cancer risk of 1 in 1 million (1.00E-06).

In summary, based on ATSDR's evaluation, it is unlikely that intermittent exposure to VOC's found in private irrigation wells near the W.R. Grace would cause adverse health effects in people.

†††† Private Well Protection Handbook for Local Boards of Health, 1998 Revision. Massachusetts Association of Health Boards, pages 23-24

Comment #7:
Surface water, consider past concentrations/exposures
Summary, Sixth Paragraph
In addition to current concentrations of contaminants found in surface water, please consider past conditions. Were contaminant concentrations higher in surface water in the past? Were there additional contaminants to those found currently? Are surface water data available in pre-1997 OU-1 reports? In the 1997 Initial Site Characterization Report for OU-3? Please consider possible exposures to surface water contaminants over the past 27 years, in addition to current exposures.

ATSDR Response:
ATSDR cannot calculate doses for past exposure to surface water, because there is not sufficient data available.

Comment #8:
Fish: indeterminate risk
Summary, Eighth paragraph
This paragraph concludes that fish do not pose an apparent health hazard because edible sized fish were not caught. Since these conditions could change, please end the last sentence in this paragraph with the phrase, "at this time.", and add "Future reassessment will be needed if conditions change and edible sized fish are present in the pond."

(As noted later on in the report, (in the section: "Pathways Considered and Eliminated *Fish*"): "If future conditions at this pond change, due to stocking programs, or people report collecting fish of edible size, then we recommend that suitable samples be collected, analyzed and evaluated.")

ATSDR Response:
ATSDR edited the last two sentences in Summary paragraph eight to read as follows: Therefore, ATSDR presently considers fish consumption from Sinking Pond to be a no apparent public health hazard. However, if future conditions change at Sinking Pond, and fish of edible size can be caught and consumed by anglers then a reassessment of this pathway may be warranted.

Comment #9:
Purpose: evaluate past and present exposures
Purpose and Health Issues, First paragraph
As already stated in previous comments, please cumulatively assess past exposures as well as present ones.

Please change the last sentence in this paragraph to say: "The purpose of this public health assessment (PHA) is to evaluate and present information pertaining to whether exposure to site-related contaminants **could have occurred in the past or could be occurring presently**, and whether health effects **could have resulted/could result** from these exposures. (Suggested changes are highlighted in bold.)

ATSDR Response:
ATSDR edited the first paragraph under the Purpose and Health Issues section to read as follows:
The purpose of this public health assessment (PHA) is to evaluate and present information pertaining to whether exposures to site-related contaminants could have occurred in the past, are presently occurring, or may occur in the future and whether health effects could result from these exposures, based on available data.

Comment #10:
Soil remediation
Purpose and Health Issues, Background, Site Description and History.
Operable Unit-1 for the site, which included soil remediation, was completed in 1997.

ATSDR Response:
This sentence was added at the end of the section on Site Description and History.

Comment #11:
Recreational uses of Fort Pond Brook
Purpose and Health Issues, Background, Land Use and Natural Resources, Second paragraph
Recreational uses of Fort Pond Brook include fishing, canoeing, and kayaking. The MA Department of Fish and Game (aka MA Dept. of Fisheries and Wildlife), stocks Fort Pond Brook with trout every spring. A farm located across the stream from the WR Grace Site uses Fort Pond Brook water for irrigation purposes.

ATSDR Response:
The following was added to the Purpose and Health Issues, Background, Land Use and Natural Resources section:
It has been reported by the community, that recreational uses of Fort Pond Brook include fishing, canoeing, and kayaking. The Massachusetts Department of Fish and Game stocks Fort Pond Brook with trout every spring.

The following sentence was deleted: The extent of recreational use of Fort Pond Brook could not be determined.

Comment #12:
Private wells at site
Purpose and Health Issues, Background, Land Use and Natural Resources, Third paragraph
Please change the last sentence in this paragraph to say:
"There are no private drinking water wells onsite; however, six private irrigation wells have been identified as being on the site, or within 500 feet of the mapped plume."

ATSDR Response:
The last sentence in the third paragraph under the Purpose and Health Issues, Background, Land Use and Natural Resources section was modified as follows:

There are no private wells onsite, however six private irrigation wells have been identified in the vicinity of the plume.

Comment #13:
Wells/Wellfields
Discussion of Environmental Contamination, *Public Water Supply, Private Well, and Groundwater Sampling*, First paragraph
Scribner is actually a wellfield, rather than a single well. Several of the relevant public wells have been reconfigured over the years---so, the easiest/most accurate way to refer to them, individually or collectively, is to use the term: "well/wellfield".

ATSDR Response:
Thank you for this clarification.

Comment #14:
Irrigation wells
Discussion of Environmental Contamination, Public Water Supply, Private Well, and Groundwater Sampling, Second paragraph
As discussed in comments 4 and 5, above, additional data are available for the Lisa Lane, Bellantoni Drive and Powder Mill Plaza irrigation wells. Also see Attachment A, and July 2005 RI; p. 2-7.

ATSDR Response:
Please see ATSDR responses to comments 4 and 5, above.

Comment #15:
Details are available for groundwater data
Discussion of Environmental Contamination, Public Water Supply, Private Well, and Groundwater Sampling, Third paragraph

Re: Groundwater Monitoring
Details regarding well locations, number of samples collected, and target analytes are available in the July 2005 Public Review Draft Remedial Investigation Report, (See Table 3-3, Figures 4-1, 4-2, etc., Appendix B, Tables B-1 and B-2), as well as in several previous reports on the WR Grace Site, including:

- Phase I RI Data Report, Appendix E, April 30, 2001,
- Phase I. RI Data Report Addendum, Attachment F, August 16, 2002, and
- Draft RI, August 30, 2002

ATSDR Response:
The following sentence was deleted: Details regarding the sampling event and target analytes are unavailable.

Comment #16:
Details are available for surface water and sediment data
Discussion of Environmental Contamination, Surface Water and Sediment Sampling, Second and Third paragraphs

Re: Surface water samples and samples for Ecological Risk Assessment (and human health risk assessment)
Details regarding the sampling events and target analytes are available. See July 2005 Draft PHRA and BERA, as well as:

- Data Report for Ecological Risk Assessment Vol. 1 and 2, April 25, 2003
- Baseline Ecological Risk Assessment, July 30, 2004

ATSDR Response:
See response to previous comment.

Comment #17:
Arsenic and cancer
Discussion of Environmental Contamination, Completed Exposure Pathways, Public Water Supply Wells, Arsenic, Second paragraph

The first sentence of this paragraph states:
Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, and prostate cancer."
Since arsenic is a contaminant of concern at the site, please include all of these cancers in the 2005 PHA assessment of cancer incidence in the area. Prostate cancer and skin cancer are not included in this assessment in the current draft PHA. (Please also see comment 35 below.)

ATSDR/ MDPH Response:
To evaluate possible health effects associated with potential exposures to contaminants of concern such as arsenic and vinyl chloride at the W.R. Grace site, ATSDR and MDPH selected six cancer types for evaluation: cancers of the bladder, brain and central nervous system (CNS), kidney, liver, and lung and bronchus as well as leukemia. These cancer types are evaluated in the draft Health Consultation (Appendix G) and results of the analysis are summarized in the draft PHA.

The primary risk factor associated with development of skin cancer is sun exposure (ACS 2005a, 2006). While a number of studies in the scientific literature indicate that ingestion of arsenic can also increase the risk of developing some types of nonmelanoma skin cancer, it was not possible to include this cancer type. Specifically, the primary type of skin cancer observed to occur as a result of chronic arsenic ingestion is squamous cell carcinoma, and multiple basal cell carcinomas have been observed as well (ATSDR 2006). However, diagnoses of nonmelanoma skin cancer are not required to be reported to the Massachusetts Cancer Registry (MCR). While the MCR collects information on diagnoses of melanoma, this type of skin cancer has not been found to be associated with arsenic exposure.

While exposure to arsenic has been shown in the scientific literature to be associated with development of a number of internal tumors including prostate cancer, arsenic exposure is not a primary risk factor for the disease; therefore, this cancer type was not selected to evaluate possible health effects due to arsenic exposure at the W.R. Grace site. Known or suspected risk factors for prostate cancer include advanced age, family history of the disease, race, diet, and possibly a history of vasectomy (ACS 2005b). However, to address community concerns about this cancer type, rates for prostate cancer are provided below for males in Acton CT 3631.01 where the W.R. Grace site is located. Fewer males were diagnosed with prostate cancer than expected during the overall time period 1982-2000 and for two of the three smaller time periods. During the middle time period 1988-1993 prostate cancer occurred as expected.

Prostate Cancer Incidence

Census Tract 3631.01, Acton, MA

	Observed	Expected	SIR	95% CI
1982-1987	6	8.9	67	25 - 126
1988-1993	18	18.6	97	57 - 153
1994-2000	33	40.2	82	57 - 115
1982-2000	**57**	**65.7**	**87**	**66 - 112**

Source: Massachusetts Cancer Registry, Massachusetts Department of Public Health

Comment #18:
EPA and ATSDR calculated different risks due to arsenic
Discussion of Environmental Contamination, Completed Exposure Pathways, Public Water Supply Wells, Arsenic, Second paragraph

What accounts for the difference in cancer risk due to arsenic, when calculated by ATSDR versus EPA? The ATSDR estimate is an excess lifetime theoretical cancer risk of 2.23E-05; while the figure in the Public Health Risk Assessment, overseen by EPA, is 1E-04.

This is a reminder that risk assessments, by their nature, rely on assumptions (re: exposure frequency, etc.), They also rely on the availability of sampling data, and of relevant toxicological studies, etc. "Indeterminate public health hazard" (an ATSDR term), and "no unacceptable risk" (an EPA term), are not the same as "no risk".

ATSDR Response:
ATSDR and EPA use similar methods for calculating theoretical excess lifetime cancer risk, however, our exposure assumptions for the calculations sometimes differ slightly. The differences in the assumptions result in differences in the estimated risk. However, both ATSDR's and EPA's calculated theoretical excess cancer risk are in the range of 1E- 04 to 1E-06. There are varying suggestions among the scientific community regarding acceptable excess lifetime cancer risk, due to uncertainties regarding the mechanism of cancer. The recommendations of many scientists and EPA have been in the risk range of 1 in 1 million to 1 in

10,000 (as referred to as 1E -04 to 1E -06) excess cancer cases. An increased lifetime cancer risk of one in a million or less is generally considered an insignificant increase in cancer risk.

Comment #19:
VOCs in Private wells
Discussion of Environmental Contamination, Potential Exposure Pathways, Private wells

a. VOCs were found in at least three private irrigation wells. VOCs detected in one or more of these wells included: Acetone, benzene, MTBE, TCE, VDC (1,1-Dichloroethene), and vinyl chloride. See previous comments 4, 5, and 14 for details.

b. Please develop an exposure scenario for each of the VOCs detected in private irrigation wells.

c. Please evaluate inhalation exposures, as well as ingestion, and dermal contact.

d. Please consider past exposures, as well as current ones. The Lisa Lane well was installed in 1995. The Powder Mill Plaza well was installed in?

ATSDR Response:
Please refer to ATSDR responses to comments 4 and 5, above.

Comment #20:
Arsenic in Surface Water
Discussion of Environmental Contamination, Potential Exposure Pathways, Surface Water, Arsenic

ATSDR calculated an excess lifetime risk for an adult of 7.21E-04 for incidental ingestion and dermal contact using the maximum concentration of arsenic in surface water. The PHRA, done under EPA guidance, stated that there were not any risks above 1E-04 in surface water.

Again, this is a heads up to the public that risk assessments involve assumptions and judgement calls, in their calculations. The public is best protected when conservative assumptions are made and there is a policy to act on the side of safety. In this instance, ATSDR's assessment is more conservative than that overseen by EPA. (See also comment 18, above).

ATSDR Response:
It is true that risk assessment requires that assumptions be made regarding the parameters used in the risk assessment calculations and that results may differ slightly due to these assumptions. It is important to note that while ATSDR's and EPA's theoretical excess cancer risk estimates differ slightly they are both in the 1E -04 risk range. It is reassuring from a public health perspective that even though slightly different assumptions or methods may have been used by ATSDR and EPA that the theoretical excess cancer risk estimates were in the same range.

Comment #21:
Arsenic
Discussion Section, Public Health Implications, Arsenic, second paragraph

The text states that "A few studies found no harmful effects in persons in the United States who throughout their lifetimes drank water containing arsenic at levels of 50 ppb to 100 ppb [3]." Is this statement misleading, or at least not a complete characterization of the health hazards, including cancer risk posed by arsenic at these concentrations? Is there a shortage of studies in the U.S. of arsenic consumption at these concentrations? (Hopefully, no one is consuming water with arsenic at these concentrations!) Are there other studies that did find harmful effects from exposures at these levels?

The toxicity value used in the Public Health Risk Assessment is 0.045 ppb. The PHRA calculated a theoretical cancer risk of 1E-04, given the 5.2 ppb of arsenic detected in the School Street wells.

ATSDR Response:
The levels of arsenic detected in the Assabet and School Street public water supply wells are well below those discussed in the study cited from the ATSDR Toxicological Profile. Even at the higher levels cited in the study harmful effects were not found, therefore it is unlikely harmful effects would occur from the much lower concentrations found in the Assabet/ School Street wells. ATSDR used the maximum concentration of 5.2 ppb found in the School Street Wells for its estimated exposure dose calculations related to drinking water ingestion. The difference between EPA's estimated carcinogenic risk and ATSDR's is likely due to slightly different exposure parameters in the calculations.

Comment #22:
Child exposure to arsenic
Discussion Section, Public Health Implications, Arsenic, third paragraph
The last sentence in this paragraph ends: "…it is not likely that a one to six year old child would ingest or play in sediment with the highest concentrations as often as ATSDR has estimated."

Comment: Human behavior is unpredictable, and cannot be relied upon as a safety measure. Remediation is needed, including the removal of contaminated sediments, so that a child can 'safely' contact the sediment as often and as long as desired.

ATSDR Response:
It is true that human behavior is not easily predictable; however, ATSDR believes that its exposure assumptions used to calculate risk in this public health assessment are conservative, but realistic. It is unlikely that a one to six year old child would trespass on the site and have contact with soil or sediment as frequently as ATSDR estimated.

Comment #23:
Cumulative risk from Arsenic
Discussion Section, Public Health Implications, Arsenic, fourth paragraph

What concentrations were used for arsenic when calculating cancer risk due to ingestion of private well water? Please use the site wide groundwater data for this calculation, rather than the site specific data from the six known irrigation wells. The six irrigation wells were only sampled for VOCs, not for inorganics, so the arsenic levels in these wells are unknown.

ATSDR Response:
ATSDR did not calculate estimated exposure doses for arsenic in private well water, because people in the vicinity of the W.R. Grace site are connected to the municipal drinking water supply and because there was no data for arsenic available from these wells. Because people in the vicinity of the W.R. Grace site are connected to the municipal water supply it is unlikely they would ingest arsenic from these private wells.

There is no completed exposure pathway for persons near the W.R. Grace to groundwater concentrations of arsenic; therefore, using the site wide groundwater data to calculate an estimated exposure dose would not be prudent.

Comment #24:
Cumulative risk from site contaminants
Discussion Section, Public Health Implications, Arsenic, fourth paragraph

As was done with arsenic, please also calculate cumulative risk from multiple pathways for other site contaminants. Please consider past and present exposures, (including to soils, sludge, etc.) Please also consider synergistic effects when individuals are exposed to multiple chemicals.

ATSDR Response:
For contaminants exceeding comparison values and with available information ATSDR did calculate carcinogenic risk from multiple pathways (see Table 9, which includes arsenic, Bis(2-ethylhexyl)phthalate, Polycyclic Aromatic Hydrocarbons, Polychlorinated Biphenyl's and vinyl chloride).

ATSDR reviewed the limited data available for VOC's that was provided in the 1992 Initial Release Public Health Assessment, prepared by the Massachusetts Department of Health. As previously discussed, ATSDR believes that this data of limited value for determining public health conclusions for past exposure, because in most cases there is only one data point per year for the years 1978 through 1992 and in the case of benzene there is only data point for the years 1978 through 1992. However, in an effort to answer ACES question regarding past exposures to VOC's, based on this very limited data, ATSDR did evaluate the historical data in the Public Health Assessment. Please refer to the section entitled: Public Water Supply Wells, Historical Data, VOC's. ATSDR concludes that past levels of VOC's in the Assabet wells posed no apparent public health hazard; however, levels of TCE may have exceeded current health guidelines, but because of the short duration of exposure (maximum 9 years) and the relatively low levels compared to potential effects in health studies, it is unlikely that adverse health effects

have occurred. In addition, because the estimated exposure doses were below a CV or established health guideline for all of the VOC's, except TCE, it is unlikely that that there would have been synergistic effects to individuals exposed to these VOC's between 1978 and 1992.

As discussed previously, since the early 1980's, the AWD has been treating the groundwater through an air stripping process to remove VOC contamination from its active water supply wells. Therefore, there is no current or expected future exposure to VOC's in the municipal drinking water supply.

Comment #25:
Concord census tracts
Health Outcome Data, Second paragraph
Part of the WR Grace Superfund Site, is located in Concord, and Concord residents could have been exposed to onsite contamination over the past 27 plus years. These exposures could include exposures to contaminated soils, sludge, surface water, sediments, and airborne contaminants including those released by the ARS. Please evaluate cancer incidence in Concord as well as in Acton, especially for any Concord census tracts that include the WR Grace Site. The 1992 PHA looked at census data from Census Tract 3612.

ATSDR/ MDPH Response:
The MDPH will evaluate the six cancer types previously mentioned for Concord CT 3612 and incorporate the results into the ATSDR PHA and the Health Consultation report. It is also important to note that the MDPH will include an evaluation of cancer incidence for some of these cancer types (e.g. lung cancer, brain cancer, leukemia) in the town of Concord as part of a PHA currently being conducted for the Starmet site located in Concord.

Comment #26:
Comprehensive cancer review requested
Health Outcome Data, Second paragraph
Please provide information on all cancer data for the census tracts, rather than for just six preselected cancers. (The 1992 PHA took this more comprehensive approach.) In particular please include data on: colorectal cancer, Hodgkins disease, and non-Hodgkins lymphoma, prostate cancer, skin cancer, pancreatic cancer, breast cancer, and ovarian cancer or cancer of the uterus.

ATSDR/ MDPH Response:
The MDPH selected the six cancer types for evaluation based on available environmental data at the W.R. Grace site and information in the scientific literature on known or suspected associations with contaminants of concern (e.g. arsenic, vinyl chloride). Much of this information was not available at the time of the 1992 assessment. Community specific cancer data for most cancer types are available for review on the MCR's website at http://www.mass.gov/dph/bhsre/mcr/canreg.htm.

Comment #27:
Bladder Cancer
Health Outcome Data, Fifth paragraph; also Appendix G.
The text states that bladder cancer occurred less often than expected, but Tables 1a, 1b, and 1c in the current report show more bladder cancer cases than expected in Acton females in 1982-2000; and in 1982-1987 and 1988-1993. There was also increased incidence of bladder cancer in certain census tracks for specific time intervals.

ATSDR/ MDPH Response:
As described in the Draft Health Consultation (Appendix G), some elevations in bladder cancer incidence were observed for females in the town of Acton as a whole and for males and females combined in CT 3631.02 during certain time periods. However, none of the elevations were statistically significant and there were no consistent trends in these elevations over time. In addition, when the geographic distribution of individuals diagnosed with bladder cancer was examined in relation to the W.R. Grace site, no unusual patterns were observed that would suggest the existence of a common factor.

Specifically, as stated in the results section of the Draft Health Consultation report (Appendix G):

> "Bladder cancer occurred less often than expected among males and females combined in the town of Acton as a whole during 1982–2000, primarily due to a lower-than-expected rate among males in the town (24 diagnoses observed vs. 30.2 expected, SIR = 79). Females were diagnosed more often than expected during 1982–2000 (15 diagnoses observed vs. 11.3 expected, SIR = 133). This elevation was not statistically significant. Males in Acton experienced fewer diagnoses of bladder cancer than expected during each of the smaller time periods evaluated, while females were diagnosed slightly more often than expected during 1982–1987 and 1988–1993 and less than expected during 1994–2000 (see Tables 1a–1d). Each of the elevations among females represented an excess of about three cases and neither was statistically significant.
>
> While bladder cancer occurred less often than expected in three census tracts in Acton during 1982–2000 (i.e., CTs 3631.01, 3632.01, and 3632.02), this cancer type was diagnosed more often than expected among males and females combined in CT 3631.02 (13 diagnoses observed vs. 8.2 expected, SIR = 159). This elevation was not statistically significant (see Tables 3a–3d)."

Comment #28:
ACES Comments/Questions, Oct. 28, 2003
Community Health Concerns
ACES submitted a list of comments/questions to ATSDR at the October 28, 2003 public meeting held at Acton Town Hall. These comments are also available online at www.actonaces.org; and are in hard copy as Attachment E to this letter. Please respond to these comments/questions in the 2005 PHA.

ATSDR Response:
There were 12 comments/ questions on this list and they are included as comments 38 through 50 of this section.

Comment #29:
Acrylonitrile questions
Community Health Concerns
In the second set of questions about acrylonitrile, the points beginning: "Given: that VOC testing…" would be clearer if left as bullet points, (or separated by semicolons), since they are hard to follow in the current format. Also, please include the concluding comment that came after these bullet points in the original ACES comments (See attachment F):

"It seems appropriate that ATSDR consider that individuals drinking Acton public water from the contaminated wells, between 1970 and 1978, may have been exposed to acrylonitrile and that the dose may have been at a high enough level to have caused health effects. (Exposure routes could have included ingestion, inhalation, and dermal exposure.)"

These comments were written in response to discussion about acrylonitrile on pages 35 to 36 of the 1992 ATSDR "Draft: Public Comment" PHA. (See Attachment B to these comments.)

ATSDR Response:
Suggested formatting changes and the additional text were added to the Community Health Concerns section, as requested. ATSDR reiterates that it concurs that persons who received water from the Assabet wells between 1970 and 1978 may have been exposed to acrylonitrile; however, ATSDR has no data that indicates there was acrylonitrile contamination in the wells during that period. Because there is no data from the drinking water wells, ATSDR cannot calculate an estimated exposure dose.

Comment #30:
Cancer cluster?
Community Health Concerns
In answer to a question about cancer clusters, the PHA quotes the MDPH assessment as saying that there were no apparent spatial patterns at the neighborhood level; but a few pages earlier the PHA discussed a spatial distribution of brain and CNS cancer diagnoses. "…there were a few locations in Acton where two or three individuals with this cancer type were located in relative close proximity to each other, with some located in the southern area of town near the W.R. Grace site." The text then discusses a lack of temporal trends in diagnoses, and a variety of cell types of brain and CNS cancer among these individuals. Please include this information in the answer to the question about clusters, and/or refer the reader back to this part of the report.

ATSDR/ MDPH Response:
The Final PHA report language will be revised to include additional information about the geographic pattern of cancer in neighborhoods surrounding the W.R. Grace site, consistent with what is presented in the draft Health Consultation (Appendix G).

Comment #31:
AWD stripping towers at Assabet wells not in use until 1983 to 1984.
Conclusions, First bullet point.
The text states: "Since 1980, the AWD has been treating the groundwater through an air stripping process to remove VOC contamination from the active water supply wells."
Air strippers were not used on the Acton Water District wells until 1983-1984. Both Assabet 1 and Assabet 2 were taken offline in 1978. According to the AWD, Assabet 1 was brought back online with carbon filtration, for a few months in 1982. When testing showed that low levels of TCA were "breaking through" the carbon filters, they took this well offline again. In 1983 Assabet 1 was brought back online with both airstripping and carbon filtration technology in place. By March of 1984, Assabet 2 had also been brought back online with this same treatment. At some later date the AWD stopped using carbon filtration at these wells, but it continues to use an airstripper to remove VOCs from the water.

ATSDR Response:
Thank-you for this clarification. The text has been modified to reflect this information.

Comment #32:
Irrigation well data
Conclusions, Third bullet point.
Please revise this bullet point based on the additional VOC data that are available for the irrigation wells. See previous comments 4, 5, etc.

ATSDR Response:
Please see ATSDR responses to comments 4 and 5, above.

Comment #33:
Evaluation of soil?
Conclusions, Third bullet point.
The third bullet point refers to an evaluation of potential health effects from exposure to contaminants in soil. Was an evaluation of soil done as part of this PHA? If not please conduct such an evaluation, especially of past contamination of soil in site disposal areas.

Also see references to soil in the fourth bullet point under Conclusions and the second bullet point under Recommendations.

ATSDR Response:
A separate evaluation of soil data was not completed, because soil data was insufficient in the reference for making a health determination related to past exposures. If additional soil data becomes available a separate evaluation could be conducted.

Comment #34:
Demographic Map needs revision
Appendix A. Figure 1
The demographic map only shows the portion of the WR Grace site that is south of the MBTA line. A substantial portion of the site is also north of the MBTA line, and should be included on this figure. The one mile buffer should be redrawn accordingly.

ATSDR Response:
ATSDR has modified the demographic map to include the area north of the MBTA line and the one mile buffer has been redrawn accordingly.

Comment #35:
Comprehensive cancer review requested
Main ATSDR Report, Health Outcome Data, Second paragraph
As stated in comment 26 above, please provide information on all cancer data for the census tracts, rather than for just six preselected cancers. (The 1992 PHA took this more comprehensive approach.) In particular, please include data on: colorectal cancer, Hodgkins disease, and non-Hodgkins lymphoma, prostate cancer, skin cancer, pancreatic cancer, breast cancer, and ovarian cancer or cancer of the uterus.

Skin cancer and prostate cancer are associated with arsenic, one of the contaminants of concern at the site. Incidences of colorectal cancer in Acton males were found to be statistically significant in a 1991 review of available data. (See Attachment D.) There are community concerns about the other cancers listed above.

ATSDR/ MDPH Response:
Refer to responses to Comments # 17 and # 26 above.

Comment #36:
Concord census tracts
Health Outcome Data, Second paragraph
As already stated in comment 25: Part of the WR Grace Superfund Site, is located in Concord, and Concord residents could have been exposed to onsite contamination over the past 27 plus years. Please evaluate cancer incidence in Concord as well as in Acton, especially for any Concord census tracts that include the WR Grace Site. The 1992 PHA looked at census data from Census Tract 3612.

ATSDR/ MDPH Response:
Refer to response to Comment # 25 above.

Comment #37:
Brain and CNS cancer, and bladder cancer
Both Brain and CNS cancer, and female bladder cancer incidence were observed more than expected in several different time periods and/or census tracts. (See Tables 1a to 1c; 2a to 2d; and 3a to 3d.)

a. Please provide further discussion of brain and CNS cancer in the Discussion section (VII) of the MDPH assessment, including a discussion of the spatial distribution of this cancer type near the WR Grace Site.

b. Please provide some discussion of increased bladder cancer observations in females in the Discussion section (VII) of the assessment.

c. Please note these two cancers in the Conclusion section (VIII) of the cancer assessment.

ATSDR/ MDPH Response:
The MDPH will revise the Discussion and Conclusions sections of its Health Consultation to include additional information about statistically significant and non-statistically significant elevations observed among males and females diagnosed with brain/CNS cancer and among females diagnosed with bladder cancer for certain time periods and census tracts. It is important to note, however, that although these elevations were observed, based on all information reviewed (i.e. incidence rates, evaluation of geographic and temporal trends, and analysis of risk factor information), it does not appear that a common factor (environmental or nonenvironmental) played a primary role in the incidence of cancer in Acton as a whole or in relation to the W. R. Grace site.

Comment #38:
As part of the planned ATSDR Public Health Assessment of the WR Grace Site, please assess disease rates at both the census tract level in Acton and West Concord, as well as town-wide in Acton.

ATSDR Response:
Please see the attached Health Consultation: Assessment of Cancer Incidence in Acton and Concord Census Tract 3612: 1982-2000 prepared by the Massachusetts Department of Public Health under cooperative Agreement with ATSDR.

Comment #39:
Please include in the assessment all cancers tracked by the MA Cancer registry, paying special attention to the cancers addressed by the John Snow Institute (leukemias, bladder cancer, colorectal cancers, liver cancers, non Hodgkins lymphoma, Hodgkins disease, and all cancers combined.)

a. Please assess both cancer incidence and cancer death rates.
b. Please include all data from the earliest possible date through the present. (JSI looked at cancer mortality data in five groupings: 1969-1973, 1974-1978, 1979-1983, 1984-1986, and 1987-1988; and at cancer incidence rates from 1982-1988.)
c. Please present data in a form so that rates from different time periods can be directly compared. (The 1995-1999 data cautions about comparing different data sets from different years.)
d. Please indicate any data limitations when assessing cancer data or trends. For example the MA Department of Public Health annual report on “Cancer Incidence and Mortality in MA 1996-2000” warns that some cancers may be under-reported due to how and

where they are diagnosed –ie in a hospital or not, (leukemia, multiple myeloma, etc.) Also the actual incidence rates for some cancers with shorter survival times, (including liver and pancreatic cancer), may be 10% higher than initially reported due to delay and error. Please recheck all data, (including historical data), so that any corrections for under-reporting, error, or delayed reporting are made.

ATSDR/ MDPH Response:

a. MDPH/BEH typically evaluates cancer incidence because this is a more accurate and complete picture of cancer in a community than cancer mortality. Furthermore, many cancers are treatable, and while they may cause long term morbidity, they do not necessarily contribute to an individual's death. The cancer experience of the community may be underestimated if only cancer mortality is examined; the number of people affected by cancer will likely exceed the number who died from cancer. Also, cancer mortality rates are based on information recorded on death certificates, which depending on who completes the death certificate, who is present at the time of death, and what is know about the decedent's underlying health conditions, may contain information on an underlying cancer diagnosis.

   Because cancer incidence data provides a more complete picture of cancer in a community, the MDPH examined the pattern of cancer diagnoses in Acton and Concord using data collected by the MCR. An analysis of cancer incidence data can detect trends faster than an analysis of cancer mortality data as we are receiving reports of most cancer diagnoses within a shorter time period. The MCR collects information on all Massachusetts residents diagnosed with cancer, and state law requires that all new cancer diagnoses be reported to the MCR within 6 months of diagnosis, regardless of where the diagnoses occur. According to state regulations, all health care facilities and health care providers that diagnose, evaluate or provide cancer treatment to cancer patients shall report to the MCR (105 CMR 301). The MCR is considered by the North American Association of Central Cancer Registries to have achieved the gold standard for certification in recent years (MDPH 2006a).

   MDPH/BEH selects cancer types for evaluation based on knowledge of community concerns or potential associations with contaminants of concern at a particular site. In the case of the W.R. Grace site, six cancer types were evaluated in this investigation, including cancers of the bladder, brain and central nervous system (CNS), kidney, liver, and lung and bronchus as well as leukemia. Colorectal cancer, non-Hodgkin's lymphoma, and Hodgkin's disease were not evaluated in relation to the W.R. Grace site because these cancer types either have no known environmental risk factors (e.g. colon and rectum cancer) or are not thought to be associated with the primary contaminants of concern at the site (e.g., arsenic and vinyl chloride).

b. Cancer data for this report was provided by the Massachusetts Cancer Registry (MCR). The MCR is a division in the MDPH Bureau of Health Information, Statistics, Research, and Evaluation. It is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain in situ (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111. s 111b). The

MCR also gathers additional information (e.g., gender, age, address at diagnosis) on each individual reported. This information is kept in a confidential database. Data are collected daily and reviewed for accuracy and completeness on an annual basis. Due to the high volume of data collected and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will inherently be a minimum of 2 years prior to the current date. This data is the most accurate and complete data available to the CAP.

As previously stated, the MCR was created by the state legislature and began collecting cancer incidence data in 1982. Data available prior to 1982 are limited to cancer mortality data gained through a search of the cause of death as recorded on death certificates. As discussed above in the response to Section A of this comment, an analysis of cancer mortality data may underestimate actual incidence as the number of people diagnosed with cancer will likely exceed the number who died from cancer. A more accurate assessment of cancer in a community can be obtained by evaluating cancer incidence rather than cancer mortality.

c. In the Health Consultation, cancer incidence was examined over a 19-year period (1982-2000) as well as over three smaller time periods (1982-1987, 1988-1993, and 1994-2000). For each time period, the cancer incidence of Acton or its census tract was compared to that of the state of Massachusetts as a whole. The use of standardized incidence ratios (SIRs), as were calculated in the Health Consultation, allows for a comparison of a community's cancer experience to that of the state over time. Because the age distribution of a community changes over time, it is most appropriate to compare the community's cancer experience for a given time period to that of the state for that same time period. Time trends can be evaluated, therefore, by determining whether the community's cancer experience is, across time periods, above, below, or consistent with the statewide experience of cancer.

d. The health consultation conducted by the MDPH/BEH (Appendix G) is an investigation that analyzes readily-available descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers appears unusual. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further analyses or public health intervention are needed.

As mentioned earlier, the MDPH/BEH uses cancer incidence data collected by the MCR. The MCR is a population-based surveillance system that collects information on all Massachusetts residents diagnosed with cancer. According to state regulations , all health care facilities, including public and private hospitals and health care providers, health care facilities, oncology centers, nursing homes, hospices, laboratories, health maintenance organizations, and other outpatient facilities that diagnose, evaluate or provide cancer treatment to cancer patients must report diagnosis or treatment of cancer (105 CMR 301).

Estimates of MCR completeness in reporting have increased steadily since the registry was established in 1982. In its early years, the MCR was estimated to include 90-95% of all reportable cancer cases in Massachusetts. More recently, efforts to improve case ascertainment have increased completeness to more than 95%, and the MCR is considered by the North American Association of Central Cancer Registries to have achieved the gold standard for certification in recent years (MDPH 2006a).

Thus, MDPH/BEH is confident that the MCR data reflect all but a small percentage of all invasive cancers among Massachusetts residents. Our reports evaluate cancer incidence data and environmental contamination, and contain discussions on applicable limitations of the available data, the W.R. Grace Acton Health consultation is no exception.

Comment #40:
Please extend the assessments of infant mortality and low birth weights, up to the present, and provide and analyze all of the data from 1969 to present.

ATSDR/ MDPH Response:
The original scope of the MDPH's work did not include either of these two health outcomes in this report. To address the comment above, MDPH reviewed the contaminants of concern and completed exposure pathways identified by ATSDR in the public health assessment. As discussed in ATSDR's PHA, two contaminants, arsenic and manganese, were measured at levels above their comparison values in public water supply wells. According to ATSDR's toxicity profiles on these two contaminants, there is some scientific evidence that consumption of high levels of arsenic and manganese in drinking water may result in adverse reproductive effects in humans or animals.

Based on comparisons to health guidelines, drinking water standards, and calculations of site-specific exposure doses using maximum detected concentrations, ATSDR concluded in the PHA that no adverse non-cancerous health effects would be expected from exposure to the maximum detected concentrations of either of these compounds in the drinking water. Nonetheless, to address concerns about low birthweight births and infant mortality, MDPH conducted two evaluations: the first involved identifying in the ATSDR toxicological profiles for arsenic the lowest dose at which an adverse reproductive outcome has been reported, to compare to the estimated maximum dose from ingestion of the contaminants in the Acton public water supply, and the second involved a review of readily-available data on infant mortality and low birth weight for Acton, to compare to statewide data on these same health outcome measures. (MDPH focused on arsenic, instead of manganese, because of arsenic's greater relative toxicity compared to that of manganese; this allowed for a worst-case evaluation of the potential for adverse reproductive outcomes.)

MDPH calculated the estimated exposure dose for adults exposed to the maximum detected concentration of arsenic in drinking water in the Acton public water supply for 365 days per year for a lifetime to be 0.00015 milligrams per kilogram per day (mg/kg/day). This estimated exposure dose is 40 times lower than the lowest adverse effect level observed in scientific studies of humans consuming doses of arsenic (0.006 mg/kg/day) that increased the incidence of adverse reproductive health outcomes (ATSDR 2007)‡‡‡‡. This indicates that residents are unlikely to have an unusually increased risk of adverse reproductive health outcomes as a result of their opportunities for exposure.

‡‡‡‡ Margin of Safety (Adult) = $\frac{0.006 \text{ mg/kg/day}}{0.00015 \text{ mg/kg/day}} = 40$

MDPH also reviewed rates of infant mortality and low birthweight births in Acton and the state as a whole for the years 1989 through 2006.

Low birthweight is considered to be the live birth of a full-term infant weighing less than 2,500 grams (approximately 5.5 pounds). Babies born with a low birth weight are alternatively referred to as "small for gestational age," and the terms are used interchangeably. There are many risk factors for low birthweight, including insufficient placental growth; maternal risk factors (e.g., poor nutrition, smoking, drug or alcohol abuse); infections (e.g., rubella, cytomegalovirus, toxoplasmosis, syphilis); chronic renal failure; and other conditions (e.g., sickle cell anemia, phenylketonuria, thrombophilia, preeclampsia). Research is beginning to emerge on environmental risk factors for low birthweight. A recent British study found that increased maternal exposure to air pollution, particularly sulfur dioxide, was associated with low birthweight prevalence (Bobak 2000).

Low birthweight data are collected by the MDPH Bureau for Health Information, Statistics, Research, and Evaluation (BHISRE). The number of low birthweight births for Acton and the state as a whole from 1989–2006 were obtained through the Massachusetts Community Health Information Profile (MassCHIP). The year 1989 is the first year for which these data are available on MassCHIP. For this analysis, rates were calculated by comparing the number of low birthweight babies to the number of total births for the community within three time periods: 1989–1994, 1995–2000, and 2001–2006. A rate per 10,000 live births was then calculated to allow for easier comparison to the state.

To determine if the number of low birthweight births observed in Acton was statistically significantly different from the number of low birthweight births observed in the state or if the difference may be due solely to chance or natural variability, a 95% confidence interval (CI) was calculated for each rate. A 95% CI assesses the magnitude and stability of a rate. By definition, a 95% CI is the range of estimated values that has a 95% probability of including the true rate of low birthweight births for the population. A confidence interval also indicates the precision of a rate; the wider the interval, the less certain the rate. Statistically, the width of the interval reflects the size of the population and the number of events; smaller populations yield less precise estimates which have wider confidence intervals.

If the 95% confidence intervals for the community do not overlap with (i.e. fall within the range of) the state's confidence intervals, then the prevalence of low birthweight births in the community is considered to be statistically significantly different from the prevalence of low birthweight births in the state as a whole. For example, for the time period 2001-2006, a rate of 507 low birthweight births per 10,000 live births was observed in Acton, with confidence intervals of 391–622. In the state as a whole for this time period, a rate of 765 low birthweight births per 10,000 live births was observed, with confidence intervals of 757–773. Because the confidence intervals for Acton (391–622) do not fall within the range of the state's confidence intervals (757–773), and because the confidence intervals in Acton are lower than the state's confidence intervals, the rate of low birthweight births in Acton is considered to be statistically significantly lower than the rate of low birthweight births in Massachusetts. The results of the analysis of low birthweight data can be seen in Tables 1 (Acton) and 2 (State).

Table 1: Low Birthweight* Prevalence Estimates for Acton, Massachusetts, 1989-2006					
Time Period	Number of Births with Low Birthweight	Total Births	Rate per 10,000 Live Births	Confidence Intervals	
1989-1994	41	1456	282	197	367
1995-2000	54	1540	351	259	443
2001-2006	70	1382	507	391	622

*Low Birthweight is considered to be below 2500 grams (~ 5.5 lbs.) when carried to full term

Table 2: Low Birthweight* Prevalence Estimates for Massachusetts, 1989-2006					
Time Period	Number of Births with Low Birthweight	Total Births	Rate per 10,000 Live Births	Confidence Intervals	
1989-1994	31632	527545	600	593	606
1995-2000	32974	485900	679	672	686
2001-2006	36319	474759	765	757	773

*Low Birthweight is considered to be below 2500 grams (~ 5.5 lbs.) when carried to full term

As illustrated in the tables, Acton had prevalence estimates for low birthweight births that were statistically significantly lower than the state of Massachusetts as a whole for each of the three time periods. In the three time periods analyzed, between 41 and 70 infants were born in Acton that were considered to be of low birthweight. Because these numbers are relatively small, the calculated prevalence estimates produced wide confidence intervals, illustrating their statistical instability. However, for each time period, the 95% CIs for Acton are below those for the state as a whole. *Because the estimates are consistently lower than that of the state, it can reasonably be concluded that low birthweight prevalence has not been elevated in the town of Acton during the 16-year period evaluated, compared to the state as a whole.*

MDPH also reviewed rates of infant mortality in Acton and the state as a whole for the years 1989 through 2006, again using data from MassCHIP. Infant mortality is defined as the death of a child less than one year of age. For this analysis, rates were calculated by comparing the number of infant deaths to the number of total births for the community within the time period of 1989–2006. As with low birthweight data, a rate per 10,000 live births and 95% confidence intervals (CI) were then calculated. The results of the analysis of infant mortality can be seen in Tables 3 (Acton) and 4 (State).

Table 3: Infant Mortality Prevalence Estimates for Acton, Massachusetts, 1989-2006					
Time Period	Number of Infant Deaths	Total Births	Rate per 10,000 Live Births	Confidence Intervals	
1989-2006	11	4,378	25	10	40

Table 4: Infant Mortality Prevalence Estimates for Massachusetts, 1989-2006					
Time Period	Number of Infant Deaths	Total Births	Rate per 10,000 Live Births	Confidence Intervals	
1989-2006	8,292	1,488,204	56	55	57

As illustrated in the tables, Acton had an infant mortality rate that was statistically significantly lower than the state of Massachusetts as a whole. Further analysis of infant deaths in each year indicates that the number of infant deaths appears to be stable and low over time, with between 0 and 2 infant deaths reported each year. No temporal trends are evident from the data and the deaths were not clustered in any one year or group of years.

Based on a review of low birthweight and infant mortality data for the town of Acton, no apparent concentrations or trends of infant deaths or low birthweight births were observed in any one year or time period in Acton. The rates of low birthweight births and infant deaths for the town of Acton were statistically significantly lower than the rates observed for the state as a whole.

Comment #41:
One limitation that has been anecdotally related about previous assessments was that Acton's population was too small and too mobile to fully analyze the effects of the WR Grace contaminants on public health. Are there any new statistical or other analytical tools available that can now be used to overcome previous limitations? Could the remaining population of long-time residents be identified, surveyed, and assessed for health effects? If so, please help with the planning for these assessments.

ATSDR/ MDPH Response:
The issue of small numbers is common in any cluster evaluation. For that reason, MDPH/BEH developed a peer-review protocol for conducting such small-scale investigations and maximizes the use of readily-available health data (e.g., by reviewing trends; readily available risk factor information; histologies; residential history information). We believe that our approach best addresses community concerns and can be (and has been) the basis of recommending additional research when unusual patterns are observed. MDPH/BEH is one of the few health departments in the country that uses health outcome data to address community concerns.

It is also important to note that ATSDR evaluated all available environmental data for the site. This assessment included estimated potential exposure opportunities using worst-case assumptions (e.g. maximum concentration detected in drinking water). They concluded that exposure to groundwater from private irrigation wells and exposure to levels of VOCs in Assabet wells pose no apparent public health hazard; exposure to the highest levels of arsenic and manganese in sediment and surface water poses a public health hazard to trespassers; and past exposure to arsenic and manganese in the municipal water supply poses an indeterminate public health hazard.

While the lack of access to information about in- and out-migration (or mobility) can also be a data limitation, it is important to note that the MCR is a high quality cancer registry that captures more than 95% of all diagnosed cancers among Massachusetts residents and records their

address at the time of diagnosis. The MCR is rated by the North American Association of Central Cancer Registries as having achieved gold standard status, the highest rating for state cancer registries (MDPH 2006a). Although clearly some individuals who may have lived much of their lives in Acton or Concord moved away before their diagnoses, it is also true that some individuals lived elsewhere and then moved to Acton or Concord, where they were diagnosed with cancer. Thus, we generally assume that there is some off-setting of both in- and out-migration for any given community.

Comment #42:
People exposed to contaminated drinking water were exposed to several chemical contaminants together. Please assess the possible synergistic and cumulative effects of human exposure to multiple chemicals in the body.

ATSDR Response:
ATSDR reviewed the limited data available for VOC's that was provided in the 1992 Initial Release Public Health Assessment, prepared by the Massachusetts Department of Health. As previously discussed, ATSDR believes that this historical data is of limited value for determining public health conclusions for past exposure, because in most cases there is only one data point per year for the years 1978 through 1992 and in the case of benzene there is only data point for the years 1978 through 1992. However, in an effort to answer ACES question regarding past exposures to VOC's, based on this very limited data, ATSDR did review and calculate non-carcinogenic and carcinogenic risk, where possible (see Comment 2 response). Based on this assessment, the maximum concentrations of ethylbenzene, 1,1 dichloroethylene and methylene chloride were below ATSDR CV's and were therefore not evaluated further. Based on the maximum concentrations and reasonable exposure parameters (see Comment 2 response), estimated exposure doses for benzene, TCE, and 1,1, dichloroethane were below established EPA or ATSDR health guidelines. Because the estimated doses for these contaminants are either below a Comparison Value or below ATSDR or EPA health guidelines it is unlikely that a person would have experienced harmful effects. In addition, because the estimated exposure doses were below a CV or established health guideline it is unlikely that that there would have been synergistic effects to individuals exposed to these VOC's between 1978 and 1992.

As discussed previously, since the early 1980's, the AWD has been treating the groundwater through an air stripping process to remove VOC contamination from its active water supply wells. Therefore, there is no current or expected future exposure to VOC's in the municipal drinking water supply.

Comment #43:
The 1992 ATSDR report concentrates on the potential relationship between chemical exposure and cancer rates. Please also assess other health-related outcomes that may be related to chemical exposure. These could include: chromosomal disorders, other birth defects, multiple sclerosis, Lou Gehrig's disease, asthma, infertility, learning disabilities, autism, rates of ADD and ADHD, other Special Education needs, etc. Can Acton's rates of these or similar health issues be compared over time to state rates? Are there any clusters of these conditions within Acton? Could any of these health issues be related to the chemical contaminants at the WR Grace Site?

ATSDR/ MDPH Response:
Similar to other state health departments throughout the country, Massachusetts does not have a statewide registry to track diagnoses of most of the other diseases or conditions that are mentioned in this comment (e.g., multiple sclerosis, infertility). However, MDPH was recently mandated to establish a statewide ALS (Lou Gehrig's Disease) Registry, which is currently being implemented. Birth defects data are collected by the Massachusetts Center for Birth Defects Research and Prevention within the MDPH Bureau of Family and Community Health and Nutrition, BEH will request these data and will make them available as part of the final report.

Also, through our environmental public health tracking activities, MDPH/BEH has pediatric asthma data for grades K through 8. Statewide pediatric asthma surveillance has been conducted by the MDPH/BEH for school years 2003-2004, 2004-2005, 2005-2006, and 2006-2007 (MDPH 2005a, 2006b, 2007). As part of a cooperative agreement with the U.S. Centers for Disease Control (CDC), MDPH's BEH established a statewide tracking system for pediatric asthma. All public and private schools in Massachusetts with any grade kindergarten through 8 report the number of enrolled students with a diagnosis of asthma. This information is generally provided by school nurses through a standardized report form, however for private schools, administrative staff complete reporting forms. School enrollment data were collected from the Massachusetts Department of Education (MDOE) or from a school's administrative staff. Data available from the tracking program indicate that the statewide prevalence of asthma among children in grades K through 8 is approximately 10%.

Table 5 contains prevalence estimates for all of the Acton K-8 schools that have participated in the MDPH pediatric asthma surveillance program. The prevalence of asthma among Acton schools for all years reported was either similar to the state as a whole (meaning that any differences in prevalence may reflect natural or random variability) or statistically significantly below the state prevalence of asthma.

Table 5: Pediatric Asthma Prevalence by School, Acton, Massachusetts

	2003-2004	2004-2005	2005-2006	2006-2007
Acton Schools				
Douglas	10.7 (8.0-13.5)	8.5 (6.0-10.9)	9.4 (6.8-11.9)	10.0 (7.4-12.7)
Gates	11.3 (8.5-14.1)	9.8 (7.1-12.4)	10.8 (8.0-13.5)	10.4 (7.7-13.1)
Luther Conant	9.1 (6.6-11.7)	10.0 (7.3-12.7)	8.3 (5.9-10.8)	6.5 (4.3-8.7)*
McCarthy-Towne	6.7 (4.5-8.9)*	11.0 (8.3-13.8)	9.3 (6.7-11.8)	10.4 (7.7-13.1)
Merriam	8.1 (5.7-10.4)	8.4 (6.1-10.7)	7.3 (5.1-9.4)*	6.9 (4.8-9.0)*
Raymond J Grey JH	11.4 (9.4-13.4)	11.1 (9.1-13.2)	NR	8.1 (6.4-9.8)*
The Victor School	---^	---^	0.0 (0.0-0.0)*	0.0 (0.0-0.0)*
Statewide Prevalence	9.5 (9.4-9.6)	10.0 (9.9-10.1)	10.6 (10.5-10.7)	10.8 (10.7-10.8)

* Indicates a statistically significant difference from the state rate for that school year
^ Schools with K-8 enrollment < 15, data not shown to protect student confidentiality
NR - not reported

Finally, although no statewide registry for autism exists, MDPH/BEH completed a report examining prevalence estimates of autism and autism spectrum disorders (ASD) in Massachusetts (MDPH 2005b). The report evaluated three years of disability and school

enrollment data in 43 Massachusetts school districts for the school years 2002-2003, 2003-2004, and 2004-2005. School district prevalence was estimated as the number of children (ages 3-22) with autism or ASD. The range of prevalence estimates for autism in Massachusetts, based on three years of data, is 26 to 55 per 10,000 children. For ASD, the range of prevalence estimates is 71 to 81 per 10,000 children.

Table 6 contains prevalence estimates for the Acton school district and the state of Massachusetts. The prevalence estimates of autism among children enrolled in Acton schools for all years reported was either similar to the state as a whole or below the state prevalence estimates of autism.

Table 6: Autism Prevalence Estimates in Acton, Massachusetts

	Acton				Massachusetts			
	Autism Count	Total Enrollment	Rate*	95% CI	Autism Count	Total Enrollment	Rate*	95% CI
2002-2003	11	2,571	43	(18, 68)	4,080	991,641	41	(40, 42)
2003-2004	13	2,544	51	(23, 79)	4,876	991,478	49	(48, 51)
2004-2005	13	2,594	50	(23, 77)	5,467	986,662	55	(54, 57)

*Prevalence per 10,000 students based on IEP data and enrollment

Comment #44:
A 1984 ATSDR memo stated that an epidemiological study was being conducted in Battle Creek, Michigan, that could be relevant to conditions in Acton since three of the contaminants being assessed were the same as the ones from the Acton site. What were the results of that study? Please provide the Acton Board of Health, ACES and the Acton Memorial Library with hard copies of that and any other relevant studies from Battle Creek or elsewhere, including any ATSDR studies of the Superfund Site in Woburn, MA

ATSDR Response:
Hard copies of the Final Public Health Assessments for the Verona Well Field, Battle Creek, Calhoun County, Michigan (December 20, 1995) and the Final Public Health Assessment for the Wells G and H, Woburn, Middlesex County, Massachusetts (November 29, 2000) can be obtained by contacting the ATSDR Records Center. You may contact ATSDR toll free at 1-888-42ATSDR. These documents are also available for review on the ATSDR web site. The results are summarized below.

<u>Summary of the Verona Well Field PHA</u>
The Verona Well Field site was placed on the U.S. Environmental Protection Agency's National Priority List in September 1983. In 1981, some of the wells in the Verona Well Field, the primary source for the municipal water system of the City of Battle Creek, Michigan, and many private wells in the same area were found to be contaminated with volatile organic chemicals. The most contaminated municipal wells were taken out of service, and other appropriate steps were taken to reduce the contaminant levels in the municipal system to acceptable levels. Beginning the following year, residents with contaminated private wells were provided with bottled water and, by 1984, their homes and businesses were connected to the municipal water

supply. New municipal wells were drilled in uncontaminated areas. Several of the abandoned municipal wells were put in service as purge or blocking wells to intercept contamination before it would reach the wells currently in use. There has been no detectable contamination in the municipal water system since 1984. The contamination has been traced to three sources near the well field: the Thomas Solvent Raymond Road Facility, the Thomas Solvent Annex, and the Grand Trunk Western Railroad marshalling yard.

Remediation of the Thomas Solvent Raymond Road facility began in 1987. Construction of the remediation system at the Thomas Solvent Annex began in mid-1993. From 1984 to 1988, the Michigan Department of Health (MDPH) and the Centers for Disease Control (CDC) conducted a health study of individuals having used contaminated private wells in the Verona area and a representative sample of individuals using Battle Creek municipal water. The study attempted to correlate health effects with exposure to the contaminated drinking water. No health effects were found that could be associated with the contamination. The individuals studied have been included in the Agency for Toxic Substances and Disease Registry (ATSDR) National Exposure Registry, Trichloroethylene (TCE) Subregistry.

The site poses a public health hazard, due to past history of exposure via groundwater and the potential for future exposure. Future exposures could occur if the remedial measures that have been taken to capture the contaminated groundwater plume were interrupted or if residents used contaminated water from private wells for drinking, cooking, or other household uses contrary to public health advisories. The extent of surface soil contamination in the source areas should be evaluated. Health concerns at the site have been largely addressed by the MDPH/CDC health study and the ATSDR Exposure Registry. Participants in the Exposure Registry will continue to benefit from registry follow-up and updates as new information becomes available. Community health education is recommended to remind those who have private wells of the risks associated with using contaminated groundwater.

Summary of the Wells G and H, Woburn PHA

The Wells G and H site encompasses two municipal wells located in Woburn, Massachusetts. The water drawn from these wells was used as a municipal water supply for the city of Woburn from 1964 to 1979. A Health Assessment was completed in 1989. This public health assessment addendum focuses on the indoor air monitoring studies conducted at the Wells G and H site since the completion of the Health Assessment in 1989. Three residences and a day care center were sampled to determine the extent of indoor air contamination due to volatilization of contaminants from the groundwater plume into the basements of the residences and the day care center. The sampling was conducted in July 1989, and in April and October 1991. The primary contaminants detected in indoor air were carbon tetrachloride, benzene, 1,2-dichloroethane, trichloroethylene, 1,1-dichloroehtylene, methylene chloride, and chloroform.

Populations potentially exposed to indoor air contaminants from the study area include residents and workers in the area and workers and children at the day care center. The exposure pathway of potential concern is the inhalation of indoor air. Although individuals have been exposed to and are exposed to contaminants in the indoor air in the residences and day care center, adverse health effects are unlikely due to the low concentrations detected.

It is unlikely that the chemicals detected during these sampling investigations are site related. The concentrations measured were typical of concentrations seen in many studies of ambient or indoor air. The majority of the samples taken at three Woburn residences and the Puddle Duck Day Care center resulted in either non-detectable levels of contaminants or low levels of contaminants. The highest levels were detected consistently at the 6 Dewey Avenue residence, which stored a considerable amount of household products. Such products contain a variety of the compounds of concern associated with this investigation; therefore suggesting another potential source of contamination. The proximity of various industrial companies, including a printing press, may be a source of contamination for the indoor air of the Puddle Duck Day Care Center. Therefore, identifying the contaminated groundwater as the only source contributing to air concentrations in the basements, cannot be made with any certainty.

Based on the available information, the indoor air in the site vicinity represents no apparent public health hazard. However, the conclusions of this public health addendum do not change the original conclusions of the 1989 Public health Assessment, which said that the Wells G and H site represents a public health hazard because of the risk to human health resulting from current and past exposure to hazardous substances, in the soil and municipal drinking water respectively, at concentrations that may result in adverse health effects.

The contaminants of concern for both of these Public Health Assessments are VOCs. The Verona Well Field/ Battle Creek PHA focused mostly on the ingestion of contaminated well water and the Summary of the Wells G and H, Woburn PHA focused primarily on exposure to VOC contaminants due to vapor intrusion or the volatilization of contaminants into indoor air from a contaminated groundwater plume.

Both of these pathways of exposure have been addressed in this W.R. Grace PHA. Based on the very limited historical data available, ATSDR has concluded that past exposure to VOCs in the Assabet wells posed no apparent public health hazard; however, levels of TCE may have exceeded current health guidelines, but because of the short duration of exposures (maximum 9 years) and the relatively low levels compared to potential health effects in other studies, it is unlikely that adverse health effects have occurred. To address vapor intrusion, ATSDR applied the USEPAs Johnson and Ettinger (1991) model to estimate possible indoor air concentrations for homes or buildings that may overly the groundwater plume. ATSDR used a very health protective model and the highest values found in groundwater to create a worst-case evaluation. As a result, ATSDR concludes that homes or buildings above the W.R. Grace site groundwater plume should not accumulate indoor air concentrations of VOCs to levels that could pose harm to residents in the vicinity of the plume.

Comment #45:
Please avoid generalizations and be as precise as possible, especially when describing potential health risks. Please use statistical means to explain risk rather than undefined terms such as “significant health risk”, “significantly increased risk of cancer”, “appreciable health effect”, or “excess cancer risk”, etc.

ATSDR Response:
ATSDR and MADPH have used statistical means to explain potential health impacts, where possible, but the use of some general or descriptive language is necessary to help convey findings in this report.

Comment #46:
Please address the question of odors and possible health effects associated with airborne contaminants from the site.

ATSDR Response:
ATSDR reviewed the information in the September, 1992 DRAFT Public Health Assessment pertaining to airborne site related contaminants.

No ambient air data for VOCs was collected during plant operations, so it is not possible for ATSDR to evaluate ambient air exposure from that time period. Site workers and trespassers may have been exposed to some VOC contaminated air during plant operations, but the levels are unknown. In addition, it is possible for VOC emissions to have migrated off-site in ambient air, however it is likely that those levels would have been much lower than those on-site. Because there is no ambient air data, it is not possible for ATSDR to evaluate past exposure to residents near the site. *Past exposure for residents near the W.R. Grace site to VOCs from plant operations in ambient air is considered an indeterminate public health hazard.*

After the air stripping towers for the Aquifer Restoration System (ARS) became fully operational in March 1985 to remove VOCs from groundwater there were some complaints of odor and concern about potential releases of VOCs into the atmosphere from the towers. After these complaints two pilot studies were conducted. One study conducted by VFL Technologies evaluated the levels of contaminants in air which could be released during the excavation of sludge from the waste areas. The data collected indicated that VOCs did not appreciably migrate from the area of excavation and that exposure to airborne contaminants would occur only in the work area or directly downwind of the work area, but still within site boundaries. The other study determined that granular activated carbon was effective in removing benzene, ethylbenzene and toluene from the air stripping tower emissions, however it was not as effective in removing vinyl chloride and DCE. The study recommended replacing the granular carbon filtration filters with activated carbon filters. This recommendation was carried out in the form of an odor control system, which was installed in September 1992 and has been in continuous service since. The odor control system consists of a booster air blower and three carbon canisters to treat the air stream prior to being discharged to the atmosphere. Due to the installation of the filters and the odor control system it is unlikely that there would be significant off-site VOC emissions from the air stripping towers. However, there could be some limited on-site exposure to VOCs for workers or trespassers who are in close proximity to the air stripping towers. An odor study was conducted by Odor Science & Engineering, Inc. in June, 2000. The analytical results confirmed that the carbon treatment system was effectively keeping VOCs at concentrations less than their odor thresholds (with the possible exception of isobutene). This report noted that the probable source of odor complaints was the stripper column effluent. The odor of the stripper column effluent was not due to elevated concentrations of any single odorant, rather it appeared to be a mixture of several compounds near their odor threshold concentration levels. It is unlikely that

odors from the stripper column effluent would migrate off-site. ATSDR has no new data for further evaluation of the ambient air pathway at the site.

Comment #47:
Since no treatment technology is 100 percent effective 100 percent of the time, please assess the health impacts of the contaminants from the northeast plume to drinking water customers, assuming no treatment of the water. What are the worst-case potential health impacts due to exposure via ingestion, dermal exposure, and inhalation?

ATSDR Response:
ATSDR believes that this is an unlikely scenario and that a speculative worst-case analysis would not be meaningful. It is highly unlikely that public drinking water customers in the Acton Water District would be exposed to untreated drinking water. The Acton Water District routinely monitors its public drinking water supply wells and if levels of contaminants are detected, in excess of health guidelines set forth in the Safe Drinking Water Act, immediate action will be taken.

Comment #48:
Private irrigation wells in the plume to the northeast may have brought contamination to the surface in the past. Please assess any possible health effects this past exposure may lead to. Please also assess any potential synergistic effects that may have occurred if residents were exposed to WR Grace contaminants in conjunction with lawn chemicals.

ATSDR Response:
ATSDR has addressed the issue of contaminants in private irrigation wells in this Public Health Assessment. Please refer to the Section: *Potential Exposure Pathways, Private Wells.* This section evaluates the most likely potential exposures pathways from private wells; dermal contact and incidental ingestion.

ATSDR has no data regarding the types or quantities of lawn chemicals used by private well owners in the vicinity of the W.R. Grace site. Therefore, it is not possible to conduct this type of analysis.

Comment #49:
Please do an analogous health assessment to that done for the previous question, assuming that in the future an irrigation well were to bring the most concentrated level of contaminants to the surface, both with and without accompanying human exposure to lawn chemicals.

ATSDR Response:
ATSDR believes that this is an unlikely scenario and that a speculative worst-case analysis would not be meaningful. In addition, ATSDR has no data regarding the types or quantities of lawn chemicals used by private well owners in the vicinity of the W.R. Grace site. Therefore, it is not possible to conduct this type of analysis.

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Appendices

Appendix A

Tables

Table 1. Completed Exposure Pathways for the W.R. Grace site							
Pathway Name	Environmental Media and Transport Mechanisms	Point of Exposure	Route of Exposure	Exposure Population	Estimated Number Exposed	Time of Exposure	Chemical
Drinking water (public water supply wells)	Movement of contaminant from source to groundwater	Municipal drinking water supply	Ingestion, inhalation, direct skin contact	Residents	Unknown	Past	arsenic, manganese, ethylbenzene, 1,1, dichloroethylene, benzene, 1,1,1 trichloroethane, TCE, methylene chloride

Table 2. Potential Exposure Pathways for the W.R. Grace site

Pathway Name	Environmental Media and Transport Mechanisms	Point of Exposure	Route of Exposure	Exposure Population	Estimated Number Exposed	Time of Exposure	Chemical
Private well water	Movement of contaminant from source to groundwater	Private wells	Incidental ingestion, inhalation, direct skin contact	Residents	Unknown	Past, present, future	vinyl chloride
Surface water	Movement of contaminants into surface water	Sinking Pond, Fort Pond Brook and associated wetlands	Incidental ingestion, inhalation, direct skin contact	Recreational users of Sinking Pond and Fort Pond Brook	Unknown	Past, present, future	Antimony, arsenic, manganese, phenanthrene, bis(2-ethylhexyl) phthalate
Sediment	Movement of contaminants into sediment	Sinking Pond, Fort Pond Brook and associated wetlands	Incidental ingestion, inhalation, direct skin contact	Recreational users of Sinking Pond and Fort Pond Brook	Unknown	Past, present, future	Arsenic, aroclor 1248, aroclor 1260, benzo(a)pyrene, chromium, copper, iron, manganese, thallium

Table 3. Pathways considered incomplete for the W.R. Grace site							
Pathway Name	Environmental Media and Transport Mechanisms	Point of Exposure	Route of Exposure	Exposure Population	Estimated Number Exposed	Time of Exposure	Chemical
Volatilizing of VOCs from groundwater into buildings	Indoor air	Air within homes and buildings overlying the plume	Inhalation	Residents, workers, visitors	Unknown	Past, present, future	1,1,-DCE, vinyl chloride
Consumption of fish	Fish tissue	Sinking Pond	Ingestion	Recreational anglers in Sinking Pond	Unknown	Past, present, future	arsenic, dieldrin, iron, lead

Table 4. Contaminants Detected Above Comparison Values in Assabet Public Water Supply and School Street Wellfield Public Water Supply					
Contaminant	Minimum Detected (ppb)	Maximum Detected (ppb)	Frequency of Detection[1]	Comparison Value (ppb)	Source
Inorganics					
Arsenic	--	5.2 (J)	N/A	0.02	CREG
Manganese	0.99 (J)	520	N/A	500	Child RMEG

Source: GeoTrans 2002, 2003; Menzie-Cura 2003c.

Notes:
-- no minimum value available, only a single detection was reported
(J) - data qualifier; reported value is an estimate
N/A - not available
ppb - parts per billion
CREG - Cancer Risk Evaluation Guideline for drinking water
EMEG - Exposure Media Evaluation Guideline for drinking water
LTHA - Lifetime health advisory for drinking water
MCL - USEPA maximum contaminant level for drinking water
RBC-N - USEPA Region III Risk Based Concentration for tap water, non-carcinogenic effects
RMEG - Reference Media Evaluation Guideline for drinking water

Table 5. Contaminants Detected Above Comparison Values in Assabet Wells between 1970 and 1978, Historical Data

Contaminant	Minimum Detected (ppb)	Maximum Detected (ppb)	Frequency of Detection[1]	Comparison Value (ppb)	Source
Volatile Organic Compounds					
Benzene	--	2.1	N/A	0.6	CREG
Trichloroethylene (TCE)	--	8.0	N/A	0.2	MRL
1,1 dichloroethane	1.9	24	N/A	0.1	RfD
Methylene Chloride	1.0	46	N/A	0.06	RfD
1,1 dichloroethene/ 1,1 dichloroethylene	1.2	55	N/A	0.009	MRL

Source: 1992 Initial Release Public Health Assessment, MADPH, ACES comments submitted 9/16/2005

Notes:
-- no minimum value available, only a single detection was reported
N/A - not available
ppb - parts per billion
CREG - Cancer Risk Evaluation Guideline for drinking water
MRL – ATSDR Minimal Risk Level
RfD – EPA Reference Dose, EPA Region III RBC

Table 6. Contaminants Detected Above Comparison Values in Groundwater Monitoring Wells						
Contaminant	Minimum Detected (ppb)	Maximum Detected (ppb)	Frequency of Detection[1]		Comparison Value (ppb)	Source
Organics						
Acetone	1.4	1,500		N/A	1,000	Child RMEG
Benzene	0.22 (J)	6,000		N/A	0.6	CREG
Bis(2-chloroethyl)ether	1.6 (J)	30		N/A	0.03	CREG
Bis(2-ethylhexyl)phthalate	0.46 (J)	7.5 (J)		N/A	3	CREG
Bromodichloromethane	--	0.76 (J)		N/A	0.6	CREG
Chloroethane	0.4 (J)	85		N/A	3.6	RBC-C
Chloromethane	0.29 (J)	10.45		N/A	3	LTHA
1,2-Dichloroethane	0.23 (J)	120		N/A	0.4	CREG
1,1-Dichloroethene	0.21 (J)	660		N/A	90	Child chronic EMEG
Indeno(1,2,3-cd)pyrene	--	0.6		N/A	0.092	RBC-C
Methylene Chloride	0.21 (J)	140		N/A	5	CREG
Tetrachloroethene	0.21 (J)	11		N/A	10	LTHA
1,1,2-Trichloroethane	0.25 (J)	1.4		N/A	0.6	CREG
Trichloroethene	0.24 (J)	26		N/A	5	MCL
Vinyl Chloride	0.2 (J)	200		N/A	0.03	CREG
Inorganics						
Aluminum	13.7 (J)	35,400		N/A	20,000	Child intermediate EMEG
Antimony	3.7 (J)	75.7		N/A	4	Child RMEG
Arsenic	3.9 (J)	1,240		N/A	0.02	CREG
Cadmium	0.32 (J)	4.3		N/A	2	Child chronic EMEG
Chromium	0.71 (J)	5,150		N/A	30	Child RMEG (chromium VI)
Cobalt	2.1 (J)	1,230		N/A	100	Child intermediate EMEG
Copper	1.9 (J)	450		N/A	300	Child intermediate EMEG
Iron	26.7	107,000		N/A	11,000	RBC-N
Lead	1.6 (J)	144		N/A	15	MCL action level
Manganese	2.2	13,000		N/A	500	Child RMEG
Nickel	1.6 (J)	945		N/A	100	LTHA
Thallium	0.11 (J)	21.6		N/A	0.5	LTHA
Vanadium	2.4 (J)	39.8		N/A	30	Child intermediate EMEG

Source: GeoTrans 2002, 2003; Menzie-Cura 2003c

Notes:
-- no minimum value available, only a single detection was reported
(J) - data qualifier; reported value is an estimate
CREG - Cancer Risk Evaluation Guideline for drinking water
EMEG - Exposure Media Evaluation Guideline for drinking water
LTHA - Lifetime health advisory for drinking water
MCL - USEPA maximum contaminant level for drinking water
RBC-N - USEPA Region III Risk Based Concentration for tap water, non-carcinogenic effects
RBC-C - USEPA Region III Risk Based Concentration for tap water, carcinogenic effects
RMEG - Reference Media Evaluation Guideline for drinking water
N/A - not available
ppb - parts per billion
1 Frequency of detection could not be accurately determined; sources presented overlapping data sets.

Table 7. Contaminants Detected Above Comparison Values in Surface Water

Contaminant	Minimum Detected (ppb)	Maximum Detected (ppb)	Frequency of Detection[1]		Comparison Value (ppb)	Source
Assabet River						
Inorganics						
Arsenic	--	1.6	1	7	0.02	CREG
Sinking Pond and Gravel Pit Wetland						
Organics						
Phenanthrene	0.012	0.014	2	8	N/A	N/A
Inorganics						
Antimony	0.69 T	5.2 (J)	4	12	4	Child RMEG
Arsenic	2.6	27.05	12	12	0.02	CREG
Manganese	49.8 (J)	2,400 T	20	20	500	Child RMEG
Fort Pond Brook and North Lagoon						
Organics						
Bis(2-Ethylhexyl) Phthalate	0.61	17	6	11	3	CREG
Phenanthrene	--	0.013	1	11	N/A	N/A
Inorganics						
Arsenic	0.38 T	7 T	11	11	0.02	CREG
Manganese	160 T	4,800 T	11	11	500	Child RMEG

Source: Menzie-Cura 2003a, 2003b, 2003c

Notes:
-- no minimum value available, only a single detection was reported
(J) - data qualifier; reported value is an estimate
CREG - Cancer Risk Evaluation Guideline for drinking water
EMEG - Exposure Media Evaluation Guideline for drinking water
LTHA - Lifetime health advisory for drinking water
MCL - USEPA maximum contaminant level for drinking water
RBC-N - USEPA Region III Risk Based Concentration for tap water, non-carcinogenic effects
RMEG - Reference Media Evaluation Guideline for drinking water
1 - Frequency of detection = times detected/times sought
D - Dissolved
N/A - not available
ppb - parts per billion
T - Total

Table 8. Contaminants Detected Above Comparison Values in Sediment						
Contaminant	Minimum Detected (ppm)	Maximum Detected (ppm)	Frequency of Detection[1]		Comparison Value (ppm)	Source
Assabet River						
Organics						
Acenaphthylene	0.016 (EB)	0.14 (EB)	7	7	N/A	N/A
Benzo(a)pyrene	0.043 (EB)	0.68 (EB)	7	7	0.1	CREG
Benzo(g,h,i)perylene	0.023 (EB)	0.22 (EB)	7	7	N/A	N/A
Phenanthrene	0.055	0.8	7	7	N/A	N/A
Inorganics						
Arsenic	2.3	6.6	7	7	0.5	CREG
Thallium	0.061	0.11	7	7	5.5	RBC-N
Sinking Pond and Gravel Pit Wetland						
Organics						
Acenaphthylene	0.0018	0.09	31	31	N/A	N/A
Benzo(a)pyrene	0.0071 (J)	0.4 (J)	31	31	0.1	CREG
Benzo(g,h,i)perylene	0.0018 (JEB)	0.16	30	31	N/A	N/A
Phenanthrene	0.0085 (EB)	0.68 (EB)	31	31	N/A	N/A
Pesticides and Polychlorinated Biphenyls						
Aroclor 1248	--	0.430	1	16	0.32	RBC-C
Aroclor 1260	0.039 (J)	0.34	6	16	0.32	RBC-C
Inorganics						
Arsenic	3.3	1,500	31	31	0.5	CREG
Iron	10,000	260,000	28	28	23,000	RBC-N
Manganese	78	80,000 (J)	28	28	3,000	Child RMEG
Fort Pond Brook and North Lagoon Wetland						
Organics						
Acenaphthylene	0.0025	0.220	21	27	N/A	N/A
Benzo(a)pyrene	0.0023	1.1	23	27	0.1	CREG
Benzo(b)fluoranthene	0.0015	1.2	24	27	0.87	RBC-C
Benzo(g,h,i)perylene	0.0019	0.830 (J)	23	27	N/A	N/A
Dibenzo(a,h)anthracene	0.0022	0.220 (J)	21	27	0.087	RBC-C
4-Methyl-2-pentanone	--	0.0056 (J)	1	27	N/A	N/A
Phenanthrene	0.0024	1.2	24	27	N/A	N/A
Inorganics						
Arsenic	6.9 (J)	3,900	33	33	0.5	CREG
Chromium	1.4	223	33	33	200	Child RMEG (chromium VI)
Copper	3.9	6,000	32	32	2,000	Child intermediate EMEG
Iron	5,200	390,000	33	33	23,000	RBC-N
Manganese	60	48,400 (J)	33	33	3,000	Child RMEG

Source: Menzie-Cura 2003a, 2003b, 2003c

Notes:

-- no minimum value available, only a single detection was reported
(EB) - contaminant detected in the equipment blank
(TB) - contaminant detected in the trip blank
CREG - Cancer Risk Evaluation Guideline for drinking water
1 - Frequency of detection = times detected/times sought
(J) - data qualifier; reported value is an estimate
ppm parts per million
N/A - not available

EMEG - Exposure Media Evaluation Guideline for drinking water
LTHA - Lifetime health advisory for drinking water
MCL - USEPA maximum contaminant level for drinking water
RBC-N - USEPA Region III Risk Based Concentration for Residential, non-carcinogenic effects
RBC-C - USEPA Region III Risk Based Concentration for Residential, carcinogenic effects
RMEG - Reference Media Evaluation Guideline for drinking water

Table 9. Contaminants Detected Above Comparison Values in Fish

Contaminant	Minimum Detected (ppm)	Maximum Detected (ppm)	Frequency of Detection[1]		Comparison Value (ppm)	Source
Sinking Pond						
Pesticides and Polychlorinated Biphenyls						
Dieldrin	0.00039	0.00073	3	3	0.0002	RBC-C
Inorganics						
Arsenic	2.3	3.1	3	3	0.0021	RBC-C
Iron	410	650	3	3	410	RBC-N
Lead	0.032	0.057	3	3	N/A	N/A

Source: Menzie-Cura 2003a
Notes:
-- no minimum value available, only a single detection was reported
N/A - not available
(J) - data qualifier; reported value is an estimate
ppm - parts per million
1 - Frequency of detection = times detected/times sought
RBC-N USEPA Region III Risk Based Concentration for fish, non-carcinogenic effects
RBC-C USEPA Region III Risk Based Concentration for fish, carcinogenic effects

Table 10. Estimated Carcinogenic Risk

Chemical	Routes of Exposure	Pathway	Numeric Cancer Risk Estimate	Total Numeric Cancer Risk Estimate	Qualitative Cancer Risk Estimate
Arsenic	Ingestion, Direct Contact	Ingestion of municipal & private well water	2.23E -05	9.50E -04	Low
		Incidental ingestion of sediment	1.83E -04		
		Dermal contact with sediment	2.31E -05		
		Incidental ingestion of surface water	3.81E -07		
		Dermal contact with surface water	7.21E -04		
Benzene	Ingestion, Inhalation	Historical ingestion of municipal well water	4.75E -06	4.75 -06	Slight
Bis(2-ethylhexyl)phthalate	Ingestion, Direct Contact	Incidental ingestion of surface water	2.24E -08	4.25E -06	Slight
		Dermal contact with surface water	4.23E -06		
Methylene Chloride	Ingestion, Inhalation	Historical ingestion of municipal well water	1.42E -05	1.42 -05	Very low
Polycyclic Aromatic Hydrocarbons (PAHs)	Ingestion, Direct Contact	Incidental ingestion of sediment	1.60E -06	2.48E -06	Slight
		Dermal contact with sediment	8.83E -07		
		Incidental ingestion of surface water	9.60E -12		
		Dermal contact with surface water	4.92E -10		
Polychlorinated Biphenyls's (PCBs)	Ingestion, Direct Contact	Incidental ingestion of sediment	1.45E -07	2.34E -07	Insignificant
		Dermal contact with sediment	8.98E -08		
Trichloroethylene	Ingestion, Inhalation	Historical ingestion of municipal well water	5.26E -06 to 4.27 -06	5.26E -06 to 4.27 -06	Slight
Vinyl Chloride	Ingestion, Direct Contact	Incidental ingestion private wells	7.00E -08	9.36E -08	Insignificant
		Dermal contact from private well water	2.63E -08		

Notes: See Appendix C see for explanation of numeric cancer risk estimates

Theoretical Cancer Risk

Cancer risk estimates do not reach zero no matter how low the level of exposure to a carcinogen. Terms used to describe this risk are defined below as the number of excess cancers expected in a lifetime:

Term		# of Excess Cancers
moderate	is approximately equal to	1 in 1,000
low	is approximately equal to	1 in 10,000
very low	is approximately equal to	1 in 100,000
slight	is approximately equal to	1 in 1,000,000
insignificant	is less than	1 in 1,000,000

Appendix B

Figures

Figure 1 – Demographic Map

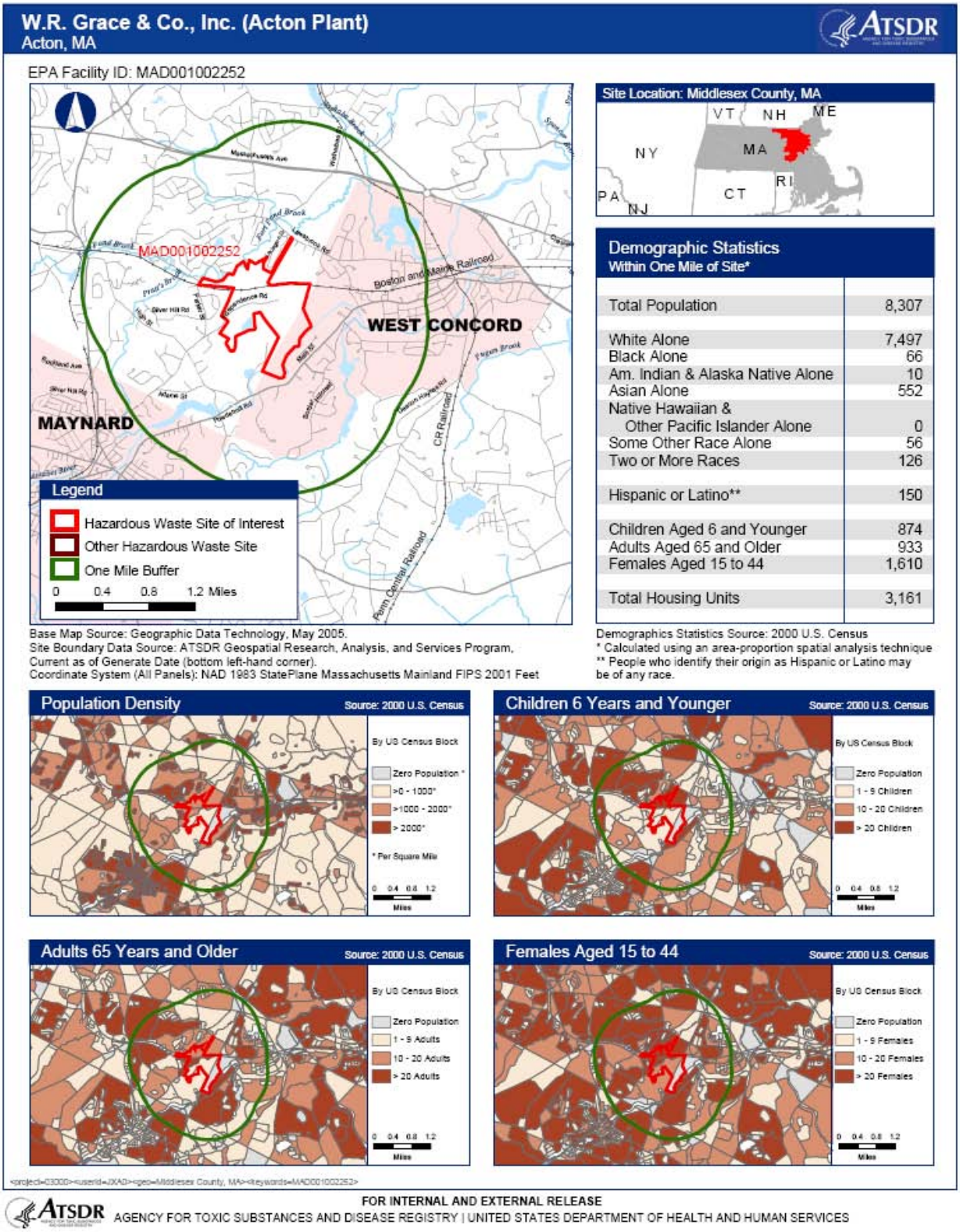

Figure 2 – Plume Map

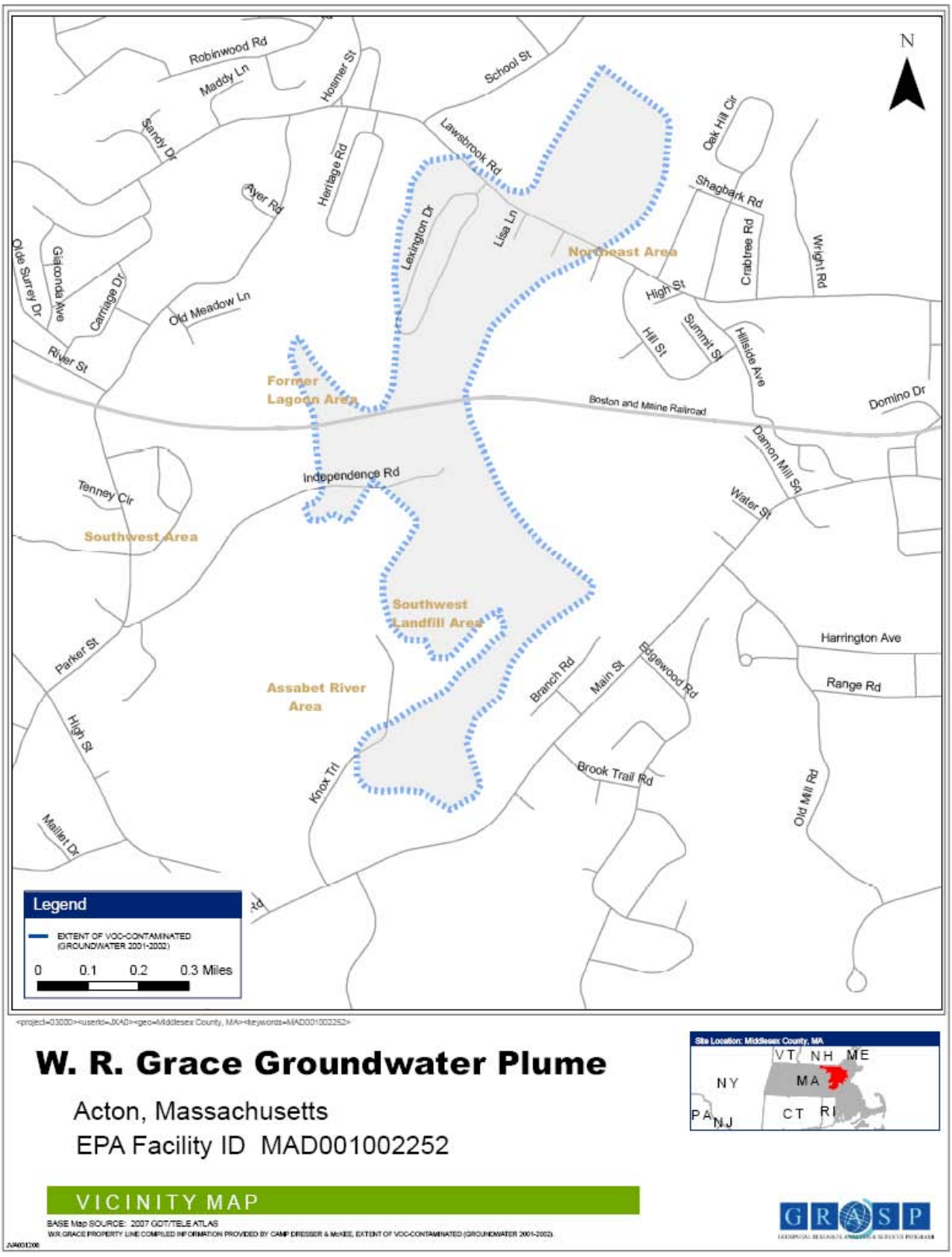

Figure 3 – Public Water Supply Wells

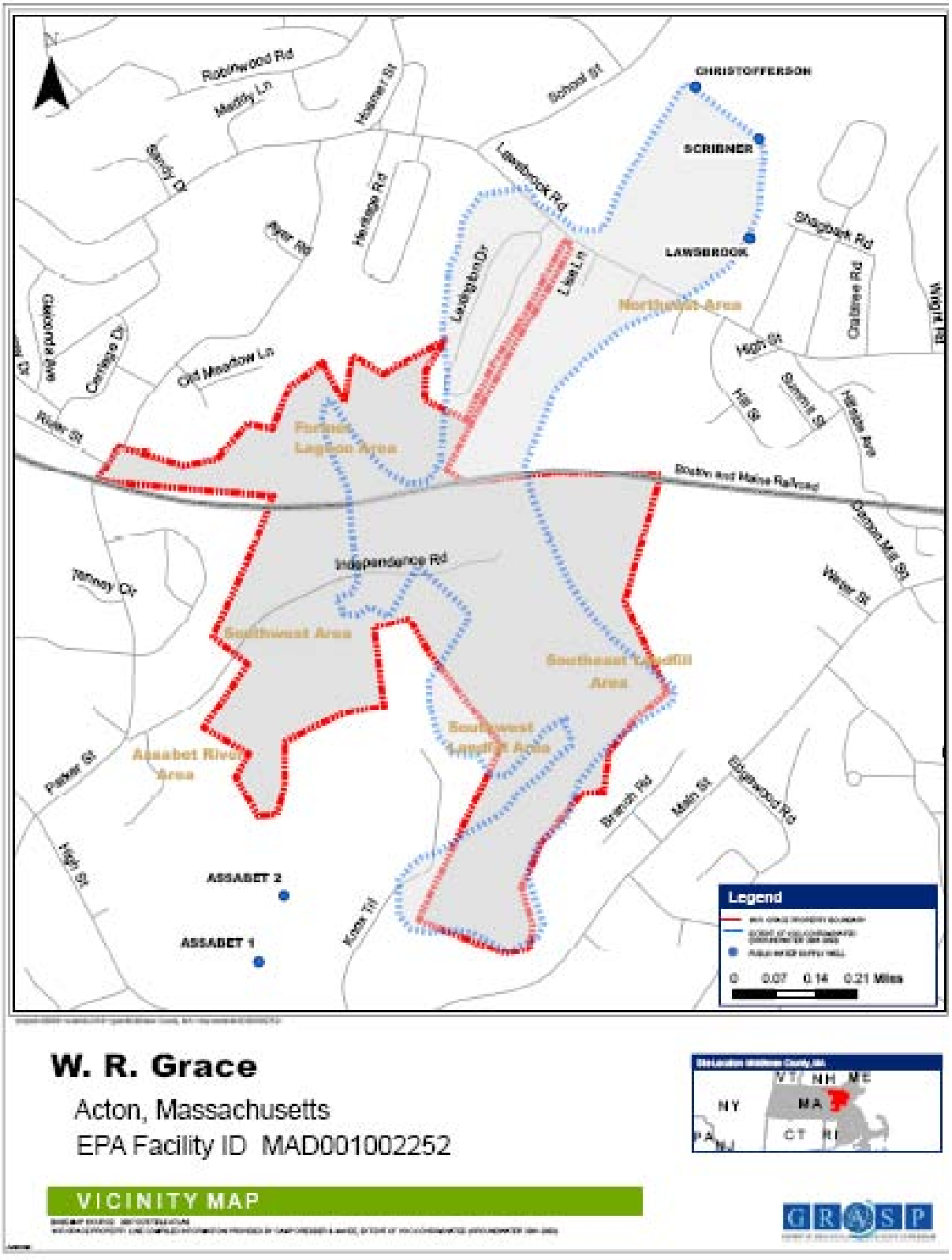

Figure 4 – Entrance Gate

Figure 5 – Sinking Pond

Figure 6 – Signs

Figure 7 - Dirt Bike Tracks

Figure 8 – Groundwater Treatment Area

Figure 9 – Fort Pond Brook

Appendix C

ATSDR's Evaluation Process

ATSDR's Evaluation Process

Step 1 – Comparison Values and the Screening Process

In order to evaluate the available data, ATSDR used comparison values (CVs) to determine which chemicals to examine more closely. CVs are the contaminant concentrations found in a specific media (for example: air, soil, or water) and are used to select contaminants for further evaluation. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of air, water, and soil that someone may inhale or ingest each day. CVs are generated to be conservative and non-site specific. These values are used only to screen out chemicals that do not need further evaluation. CVs are not intended to be used as environmental clean-up levels or to indicate that health effects occur at concentrations that exceed these values.

CVs can be based on either carcinogenic (cancer-causing) or non-carcinogenic effects. Cancer-based comparison values are calculated from the U.S. Environmental Protection Agency's (USEPA) oral cancer slope factor (CSF) or inhalation risk unit. CVs based on cancerous effects account for a lifetime exposure (70 years) with an unacceptable theoretical excess lifetime cancer risk of 1 new case per 1 million exposed people. Non-cancer values are calculated from ATSDR's Minimal Risk Levels (MRLs), USEPA's Reference Doses (RfDs), or USEPA's Reference Concentrations (RfCs). When a cancer and non-cancer CV exists for the same chemical, the lower of these values is used in the comparison for conservatism. The chemical and media-specific CVs utilized during the preparation of this PHA are listed below:

A **Reference Dose Media Evaluation Guide (RMEG)** is a comparison concentration that is based on USEPA's estimate of the daily exposure to a contaminant that is unlikely to cause adverse health effects.

A **Cancer Risk Evaluation Guide (CREG)** is a comparison concentration that is based on an excess cancer rate of one in a million persons and is calculated using USEPA's cancer slope factor (CSF).

A **Maximum Contaminant Level (MCL)** is a contaminant concentration that USEPA deems protective of public health, and may consider the availability and economics of water treatment technology.

A **Life Time Health Advisory (LTHA)** is developed by USEPA and is considered a lifetime exposure level for contaminants specifically in drinking water (assuming 20% of an individual's exposure comes from drinking water) at which adverse, non-carcinogenic health effects would not be expected to occur.

Preliminary Remediation Goal (PRG) is a screening tool, generated by EPA Region IX, which is used at the early stages of human exposure evaluation and clean-up considerations at contaminated sites. PRGs are risk-based concentrations derived from standardized equations, combining exposure assumptions and USEPA toxicity data. These values are generic and do not take into account available site-specific information.

Step 2 – Evaluation of Public Health Implications

The next step in the evaluation process is to take those contaminants that are above their respective CVs and further identify which chemicals and exposure situations are likely to be a health hazard. Separate child and adult exposure doses (or the amount of a contaminant that gets into a person's body) are calculated for site-specific exposure scenarios, using assumptions regarding an individual's likelihood of accessing the site and contacting contamination. A brief explanation of the calculation of estimated exposure doses for the site is presented below. Calculated doses are reported in units of milligrams per kilograms per day (mg/kg/day). Separate calculations have been performed to account for non-cancer and cancer health effects for each chemical based on the health impacts reported for each chemical. The same dose equations have been used for non-cancer and cancer calculations with the indicated modifications. Some chemicals are associated with non-cancer effects while the scientific literature many indicate that cancer-related health impacts are not expected from exposure.

Exposure Dose Estimation

When chemical concentrations at the site exceed the established CVs, it is necessary for a more thorough evaluation of the chemical to be conducted. In order to evaluate the potential for human exposure to contaminants present at the site and potential health effects from site-specific activities, ATSDR estimates human exposure to the site contaminant from different environmental media by calculating exposure doses. A brief discussion of the calculations and assumptions is presented below. The equations and the assumptions are based on the USEPA Risk Assessment Guidance for Superfund, Part A and the USEPA Exposure Factors Handbook, unless otherwise specified. A discussion of the cancer and non-cancer evaluation of exposure is presented following the equations for each pathway.

Ingestion of Contaminants in Municipal Wells

The exposure dose for ingestion of drinking water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

C = chemical concentration (mg/L)
IR = ingestion rate (L/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). An ingestion rate (IR) of 2 liters per day (L/day) for adults, 1.5 L/day for adolescents, and 1 L/day for children. An exposure

frequency (EF) of 350 days per year was assumed (year minus two weeks of vacation or other time spent away from the home).
It was assumed that the exposure duration (ED) was 30, 12, and 6 years for adults, adolescents, and children, respectively. A body weight (BW) of 70 kilograms (kg) for adults, 45 kg for adolescents and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults, 4,200 days for adolescents, and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Incidental Ingestion of Contaminants in Private Well

The exposure dose for incidental ingestion of private well water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

C = chemical concentration (mg/L)
IR = ingestion rate (L/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). An incidental ingestion rate (IR) of 0.1 liters per day (L/day) for adults and children. An exposure frequency (EF) of 350 days per year was assumed (year minus two weeks of vacation or other time spent away from the home).

It was assumed that the exposure duration (ED) was 30 years for adults and 6 years for children. A body weight (BW) of 70 kilograms (kg) for adults and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Direct Skin Contact to Contaminants in Private Well Water

The exposure dose for direct skin contact with drinking water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times SA \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

C = chemical concentration (mg/L)
SA = surface area (cm^2)
PC = permeability constant (cm/hour)
ET = exposure time (hours/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). A surface area (SA) of 18,150 square centimeters (cm^2) for adults and 7,195 cm^2 for children. The chemical specific permeability constants (cm/hour) are based on USEPA's 1992 Dermal Exposure Guidance, if available. If no chemical specific permeability constant (PC) was available, ATSDR assumed a PC value of one for conservatism. An exposure frequency (EF) of 90 days per year was assumed (3 summer months). An exposure time (ET) of 1.5 hours per day was assumed (estimate of time wading or swimming in a pool per day). It was assumed that the exposure duration (ED) was 30 and 6 years for adults and children, respectively. A body weight (BW) of 70 kilograms (kg) for adults and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Direct Skin Contact to Contaminants in Surface Water

The exposure dose for direct skin contact with surface water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times SA \times PC \times ET \times EF \times ED \times CF}{BW \times AT}$$

C = chemical concentration (mg/L)
SA = surface area (cm^2)
PC = permeability constant (cm/hour)
ET = exposure time (hours/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). A surface area (SA) of 18,150 square

centimeters (cm^2) for adults, 14,900 for adolescents, and 7,195 cm^2 for children. The chemical specific permeability constants (cm/hour) are based on USEPA's 1992 Dermal Exposure

Guidance, if available. If no chemical specific permeability constant (PC) was available, ATSDR assumed a PC value of one for conservatism. An exposure frequency (EF) of 24 days per year was assumed (2 days per week during 3 summer months). An exposure time (ET) of 1 hour per day was
assumed (estimate of time wading in river, pond or wetland per day). It was assumed that the exposure duration (ED) was 30, 12, and 6 years for adults, adolescents, and children, respectively. A body weight (BW) of 70 kilograms (kg) for adults, 45 kg for adolescents, and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults, 4,200 for adolescents and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Incidental Ingestion of Contaminants in Surface Water

The exposure dose for incidental ingestion of private well water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

C = chemical concentration (mg/L)
IR = ingestion rate (L/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). An incidental ingestion rate (IR) of 0.1 liters per day (L/day) for adults, adolescents and children. An exposure frequency (EF) of 24 days per year was assumed (2 days per week for 3 summer months).

It was assumed that the exposure duration (ED) was 30 years for adults, 12 years for adolescents and 6 years for children. A body weight (BW) of 70 kilograms (kg) for adults, 45 kg for adolescents, and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults, 4,200 for adolescents and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Incidental Ingestion of Contaminants in Sediment

The exposure dose for incidental ingestion of private well water is presented below.

$$\text{Dose (mg/kg/day)} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

C = chemical concentration (mg/L)
IR = ingestion rate (L/day)
EF = exposure frequency (days/years)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/L). An incidental ingestion rate (IR) of 100 milligrams per day (mg/day) for adults, 150 mg/day for adolescents and 200 mg/day for children. An exposure frequency (EF) of 24 days per year was assumed (2 days per week for 3 summer months). An exposure time (ET) of 1 hour per day was assumed.

It was assumed that the exposure duration (ED) was 30 years for adults, 12 years for adolescents and 6 years for children. A body weight (BW) of 70 kilograms (kg) for adults, 45 kg for adolescents, and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults, 4,200 for adolescents and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Direct Skin Contact with Contaminants in Sediment

The exposure dose for direct skin contact with sediment is presented below.

$$\text{Dose (mg/kg/day)} = \frac{DA_{event} \times EF \times ED \times EV \times SA}{BW \times AT}$$

DA_{event} = Absorbed dose per event (mg/cm^2-event)
EF = exposure frequency (days/years)
ED = exposure duration (years)
EV = Event frequency (events/day)
SA = skin surface area (cm^2)
BW = body weight (kg)
AT = averaging time (days)

$\underline{DA_{event}} = C \times CF \times AF \times ABS_d$

C = Chemical concentration in soil
CF = Conversion factor (10^{-6} kg/mg)
AF = Adherence factor of soil to skin
ABS_d = Dermal absorption fraction

Exposure doses were calculated using the maximum detected concentration of a contaminant (C) from the environmental data in milligrams per liter (mg/kg). A surface area (SA) of 6,074 square centimeters (cm^2) for adults, 4,471 for adolescents, and 2178 cm^2 for children. A skin adherence factor of 0.07 was used for adults and adolescents and AF of 0.2 was used for children [USEPA, RAGS, Part E, Chap 3]. If a chemical specific absorption factor (ABS) was available it was used, if not an ABS of 1.0 was used for conservatism. An exposure frequency (EF) of 24 days per year was assumed (2 days per week during 3 summer months). It was assumed that the exposure duration (ED) was 30, 12, and 6 years for adults, adolescents, and children, respectively. A body weight (BW) of 70 kilograms (kg) for adults, 45 kg for adolescents, and 16 kg for children was also assumed. It should be noted that different averaging times (AT) are used for evaluating non-cancer and cancerous health effects. The averaging time (AT) for non-cancer chemicals is equal to the ED multiplied by the EF, which is 10,500 days for adults, 4,200 for adolescents and 2,100 days for children. For chemicals associated with cancerous effects, AT of 25,550 days was used to account for lifetime exposure to a particular chemical (365 days per year multiplied by 70 years).

Non-Cancer Health Effects

The doses calculated for exposure to each individual chemical are then compared to an established health guideline, such as a Minimal Risk Level (MRL) or a Reference Dose (RfD), in order to assess whether adverse non-cancer health impacts from exposure are expected. These health guidelines, developed by ATSDR and USEPA, are chemical-specific values that are based on the available scientific literature and are considered protective of human health. Non-carcinogenic effects, unlike carcinogenic effects, are believed to have a threshold, that is, a dose below which adverse health effects will not occur. As a result, the current practice for deriving health guidelines is to identify, usually from animal toxicology experiments, a No Observed Adverse Effect Level (or NOAEL), which indicates that no effects are observed at a particular exposure level. This is the experimental exposure level in animals (and sometimes humans) at which no adverse toxic effect is observed. The NOAEL is then modified with an uncertainty (or safety) factor, which reflects the degree of uncertainty that exists when experimental animal data are extrapolated to the general human population. The magnitude of the uncertainty factor considers various factors such as sensitive subpopulations (for example; children, pregnant women, and the elderly), extrapolation from animals to humans, and the completeness of available data. Thus, exposure doses at or below the established health guideline are not expected to result in adverse health effects because these values are much lower (and more human health protective) than doses, which do not cause adverse health effects in laboratory animal studies. For non-cancer health effects, the following health guidelines are described

below in more detail. It is important to consider that the methodology used to develop these health guidelines does not provide any information on the presence, absence, or level of cancer risk. Therefore, a separate cancer evaluation is necessary for potentially cancer-causing chemicals detected in samples at this site. A more detailed discussion of the evaluation of cancer risk is presented in the Cancer Risks Section of this Appendix.

Non-Cancer Health Guidelines

Minimal Risk Levels (MRLs) – developed by ATSDR

ATSDR has developed MRLs for contaminants commonly found at hazardous waste sites. The MRL is an estimate of daily exposure to a contaminant below which non-

cancer, adverse health effects are unlikely to occur. MRLs are developed for different routes of exposure, such as inhalation and ingestion, and for lengths of exposure, such as acute (less than 14 days), intermediate (15-364 days), and chronic (365 days or greater). At this time, ATSDR has not developed MRLs for dermal exposure. A complete list of the available MRLs can be found at http://www.atsdr.cdc.gov/mrls.html.

References Doses (RfDs) – developed by USEPA

An estimate of the daily, lifetime exposure of human populations to a possible hazard that is not likely to cause non-cancerous health effects. RfDs consider exposures to sensitive sub-populations, such as the elderly, children, and the developing fetus. USEPA RfDs have been developed using information from the available scientific literature and have been
calculated for oral and inhalation exposures. A complete list of the available RfDs can be found at http://www.epa.gov/iris.

If the estimated exposure dose for a chemical is less than the health guideline value, the exposure is unlikely to result in non-cancer health effects.

If the calculated exposure dose is greater than the health guideline, the exposure dose is compared to known toxicological values for the particular chemical and is discussed in more detail in the text of the PHA. The known toxicological values are doses derived from human and animal studies that are presented in the ATSDR Toxicological Profiles and USEPA's Integrated Information System (IRIS). A direct comparison of site-specific exposure doses to study-derived exposures and doses found to cause adverse health effects is the basis for deciding whether health effects are likely to occur. This in-depth evaluation is performed by comparing calculated exposure doses with known toxicological values, such as the no-observed adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from studies used to derive the MRL or RfD for a chemical. As part of this comparison to toxicological values, a margin of exposure (MOE) is calculated by dividing the NOAEL and/or LOAEL by the site-specific exposure dose. Generally, when the MOE is greater than 1,000, harmful health effects are not expected. When the MOE ranges from approximately 100 to 1,000, further toxicological evaluation is necessary to determine whether harmful effects are likely. This may include a

closer look at the studies used to derive the NOAELs and LOAELs. Adverse health effects may occur when the MOE is less than 10.

Cancer Risk

Exposure to a cancer-causing compound, even at low concentrations, is assumed to be associated with some increased risk for evaluation purposes. The estimated excess risk of developing cancer from exposure to contaminants associated with the site was calculated by multiplying the maximum contaminant concentration by ATSDR's estimated site-specific adult exposure parameters by USEPA's chemical-specific Cancer Slope Factors (CSFs or cancer potency estimates), which are available at http://www.epa.gov/iris.

An increased excess lifetime theoretical cancer risk is not a specific estimate of expected cancers. Rather, it is an estimate of the increase in the probability that a person may develop cancer sometime during his or her lifetime following exposure to a particular contaminant. Therefore, the theoretical cancer risk calculation incorporates the equations and parameters (including the exposure duration and frequency) used to calculate the dose estimates, but the estimated value is divided by 25,550
days (or the averaging time), which is equal to a lifetime of exposure (70 years) for 365 days/year.

There are varying suggestions among the scientific community regarding an acceptable excess lifetime cancer risk, due to the uncertainties regarding the mechanism of cancer. The recommendations of many scientists and USEPA have been in the risk range of 1 in 1 million to 1 in 10,000 (as referred to as 1×10^{-6} to 1×10^{-4}) excess cancer cases. An excess lifetime cancer risk of one in one million or less is generally considered an insignificant increase in cancer risk.

Cancer risk less than 1 in 10,000 are not typically considered a health concern. An important consideration when determining cancer risk estimates is that the risk calculations incorporate several very conservative assumptions that are expected to overestimate actual exposure scenarios. For example, the method used to calculate USEPA's CSFs assumes that high-dose animal data can be used to estimate the risk for low dose exposures in humans. As previously stated, the method also assumes that there is no safe level for exposure. Lastly, the method computes the 95% upper bound for the risk, rather than the average risk, suggesting that the cancer risk is actually lower, perhaps by several orders of magnitude.

Because of the uncertainties involved with estimating carcinogenic risk, ATSDR employs a weight-of-evidence approach in evaluating all relevant data. Therefore, the carcinogenic risk is also described in words (qualitatively) rather than giving a numerical risk estimate only. The numerical risk estimate must be considered in the context of the variables and assumptions involved in their derivation and in the broader context of biomedical opinion, host factors, and actual exposure conditions. The actual parameters of environmental exposures have been given careful and thorough consideration in evaluating the assumptions and variables relating to both toxicity and exposure. A complete review of the toxicological data regarding the doses associated with the production of cancer and the site-specific doses for the site is an important

element in determining the likelihood of exposed individuals being at a greater risk for cancer. A table with the numeric and qualitative cancer risk estimates for each of the completed or potential exposure pathways is presented in Appendix A, Table 9.

Appendix D

Vapor Intrusion and Johnson and Ettinger Indoor Air Model

VOC Indoor Air Modeling

The U.S. Environmental Protection Agency (USEPA) developed the Johnson and Ettinger model to estimate indoor air concentrations and associated health hazards from subsurface vapor intrusion into buildings. This model is a *screening-level* model that estimates the transport of contaminated vapors from either subsurface soils or groundwater into the spaces directly above the source of contamination [48].

The Johnson and Ettinger model is a first-tier screening tool that uses data about properties of the soil, chemical properties of the contaminant, and structural properties of the building. All but the most sensitive parameters have been set to either an upper bound value or the median value. As a result, the model is very conservative when predicting indoor air concentrations. To predict indoor air concentrations in homes in the vicinity of the W.R. Grace site groundwater plume, ATSDR entered the maximum groundwater concentrations for benzene, 1,1-dichloroethene, 1,2-dichloroethene, and 1,2-dichloropropane and vinyl chloride into the Johnson and Ettinger model. The model generates an infinite source building indoor concentration, which is the estimated indoor air concentration of the VOC contaminant for a house located above the plume. Although the model is a useful tool that enables ATSDR and other scientists to conservatively predict indoor air concentration, it has limitations:

- It does not consider the effects of multiple contaminants.
- Its calculations do not account for preferential vapor pathways due to soil fractures, vegetation root pathways, or the effects of a gravel layer beneath the floor slab.
- The groundwater model does not account for the rise and fall of the water table due to aquifer discharge and recharge.
- The model also assumes that all vapors will enter the building, implying that a constant pressure field is generated between the interior spaces and the soil surface.
- It neglects periods of near zero pressure differential.
- Soil properties in the area of contamination are assumed to be identical to those in the area above the contamination.

VOC Indoor Air Model Approach

Table D-1 lists the indoor air concentrations that ATSDR estimated for VOCs considered in this analysis. We emphasize that these are *conservative estimates:* our initial modeling application assumed that the maximum concentration of VOCs detected in the plume entered the home. As the table indicates, the estimates of the indoor air concentrations of the VOCs were lower than the associated ATSDR chronic inhalation LOAEL or NOAEL and the levels at which effects have been observed in animal studies or exposed humans (also known as the lowest-observed-adverse-effect levels). These findings suggest that the estimated air concentrations of VOCs inside homes above the W.R. Grace site groundwater plume would not reach unhealthy levels.

Rather than simulating the many complex factors that affect how toxic chemicals disperse in air, ATSDR evaluated a simple and overestimated exposure situation: *What would be the estimated*

indoor air concentration of a VOC contaminant for a house located directly above a groundwater plume with a VOC concentration equal to the highest level measured in the specified area? Though obviously unrealistic, this scenario provides an extreme upper bound estimate of what the actual ambient air concentrations might have been. We used the Johnson and Ettinger indoor air model to estimate indoor air concentrations for residences near the Former lagoon area, Southeast landfill area, and the Southwest landfill area.

For predicting indoor air concentrations in homes at or near these areas, ATSDR entered the maximum groundwater concentrations for each identified VOC of concern into the Johnson and Ettinger model. Incremental risks obtained for each contaminant of concern for vapor intrusion into indoor air were then converted into predicted concentrations and compared to a reference value for that compound. Based on this strategy, ATSDR found that none of the predicted incremental risks or air concentrations exceeded reference values and thus were not at levels that are known to cause adverse health effects.

Table D-1. Model Incremental Risk and Indoor Air Concentrations

Exposure Point	Chemical of Potential Concern	Maximum Groundwater Concentration (ppb)	Incremental Risk (unitless)	Model Air Concentration (ppb)	Guidance Value (ppb)	Source
Former Lagoon Area	1,1-Dichloroethene	100	5.4E-03[1]	272	25,000	NOAEL
	Vinyl chloride	27	1.5E-06	156	10,000	LOAEL
Southeast Landfill Area	1,1-dichloroethene	59	2.1E-03[1]	106	25,000	NOAEL
	1,2-dichloroethene	110	2.6E-03[1]	23	None	N/A
	1,2-dichloropropane	15	7.1E-08	3	15,000	LOAEL
	Benzene	190	8.2E-07	77	780	LOAEL
	Vinyl chloride	17.5	6.5E-07	67	10,000	LOAEL
Southwest Landfill Area	Benzene	7.2	9.1E-08	9	780	LOAEL
	Vinyl chloride	3	5.3E-08	5	10,000	LOAEL

[1]Denotes the hazard quotient for a noncarcinogenic contaminant.

References:

Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings. Available at http://www.epa.gov/oswer/riskassessment/airmodel/johnson_ettinger.htm.
EPA. Integrated Risk Information System. Available at http://www.epa.gov/iris/.

Appendix E

Levels of Public Health Hazard

Levels of a Public Health Hazard

ATSDR categorizes exposure pathways at hazardous waste sites according to their level of public health hazard to indicate whether people could be harmed by exposure pathways and site conditions. The categories are:

Urgent Public Health Hazard:	This category applies to exposure pathways and sites that have certain physical features or evidence of short-term (less than 1 year), site-related chemical exposure that could result in adverse health effects and requires quick intervention to stop people from being exposed.
Public Health Hazard:	The category applies to exposure pathways and sites that have certain physical features or evidence of chronic (long-term), site-related chemical exposure that could result in adverse health effects.
Indeterminate Public Health Hazard:	The category applies to exposure pathways and sites where important information is lacking about chemical exposures, and a health determination cannot be made.
No Apparent Public Health Hazard:	The category applies to pathways and sites where exposure to site-related chemicals may have occurred in the past or is still occurring, however, the exposure is not at levels expected to cause adverse health effects.
No Public Health Hazard:	The category applies to pathways and sites where there is evidence of an absence of exposure to site-related chemicals.

Appendix F

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Environmental Health 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 (USEPA), 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).

Absorption
The process of taking in. For a person or 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 occurs 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 (more than 1 year) [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.

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 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 Aexposure dose@ is how much of a substance is encountered in the environment. An Aabsorbed 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**.

USEPA
United States Environmental Protection Agency.

Epidemiologic 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.

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

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

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

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

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

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

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

Feasibility study
A study by USEPA 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_2)
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 estimate 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/cm^2
Milligram per square centimeter (of a surface).

mg/m^3
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, condition, or 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)**
USEPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

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 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 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 health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to USEPA 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 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 USEPA 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 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 Amendments and Reauthorization Act (SARA)
In 1986, SARA amended 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 **epidemiologic 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/dictionaries.html

Appendix G

Health Consultation, Assessment of Cancer Incidence in Acton and Concord Census Tracts 3612: 1982-2000

Health Consultation

PUBLIC COMMENT RELEASE

Assessment of Cancer Incidence in
Acton and Concord Census Tract 3612: 1982 - 2000

W.R. GRACE SUPERFUND SITE
ACTON, MIDDLESEX COUNTY, MASSACHUSETTS

EPA FACILITY ID: MAD001002252

AUGUST 26, 2008

COMMENT PERIOD END DATE: SEPTEMBER 26, 2008

Approved for Release by ATSDR:

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

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You May Contact ATSDR Toll Free at
1-800-CDC-INFO
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HEALTH CONSULTATION

PUBLIC COMMENT RELEASE

Assessment of Cancer Incidence in Acton and Concord Census Tract 3612: 1982 - 2000

W.R. GRACE SUPERFUND SITE
ACTON, MIDDLESEX COUNTY, MASSACHUSETTS

EPA FACILITY ID: MAD001002252

Prepared By:

Massachusetts Department of Public Health
Bureau of Environmental Health
Under a cooperative agreement with the
The U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

This information is distributed by the Agency for Toxic Substances and Disease Registry for public comment under applicable information quality guidelines. It does not represent and should not be construed to represent final agency conclusions or recommendations.

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APPENDIX B: Risk Factor Information for Selected Cancer Types

I. BACKGROUND AND STATEMENT OF ISSUES

In response to a request from the U.S. Agency for Toxic Substances and Disease Registry (ATSDR), the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH) conducted an evaluation of cancer incidence in Acton, Massachusetts, and Concord Census Tract (CT) 3612 in southwestern Concord, Massachusetts. This evaluation was initiated based on community concerns about cancer and possible environmental exposures in relation to the W.R. Grace Acton Plant Site (W.R. Grace), located at 50 Independence Road in Acton. This project was conducted by MDPH under a cooperative agreement with ATSDR.

II. OBJECTIVES

This report provides a descriptive evaluation of the occurrence of cancer in the town of Acton as a whole and its individual census tracts for the years 1982–2000, the time period for which the most recent and complete cancer incidence data were available from the Massachusetts Cancer Registry (MCR) at the initiation of this analysis. Cancer incidence data were also reviewed for Concord CT 3612, which is located adjacent to Acton, in response to a supplemental request from the community because part of the W.R. Grace site is located in Concord (see Figure 1 for the geographic extent of analysis). This report provides a review of the pattern of cancer in the town of Acton and in Concord CT 3612 through comparison of the incidence of six cancer types with the incidence of these cancers in the state of Massachusetts as a whole. Additionally, available information about risk factors, including environmental factors, related to the development of cancer was evaluated.

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, to determine if a common cause or etiology is possible, and can 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, however, 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 of the patterns of cancer in the town of Acton and Concord CT 3612 in order to determine whether recommendations for further public health action are needed. The specific objectives of this investigation were as follows:

- To evaluate the incidence of six cancer types in the town of Acton as a whole, in the census tracts that divide the town, and in Concord CT 3612.

- To evaluate the geographic distribution of individual cancer cases in the town of Acton and in Concord CT 3612, to see if there are any patterns in any one particular area or in relation to the W.R. Grace site.

- To review descriptive information available in the Massachusetts Cancer Registry for individuals diagnosed with cancer in Acton and Concord CT 3612, to see if there are any particular characteristics related to established risk factors for developing these diseases.

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

III. METHODS

A. Case Identification/Definition

Cancer incidence data (i.e., reports of new cancer diagnoses) for the years 1982–2000 were obtained for the town of Acton and Concord CT 3612 from the MCR, a division of the Bureau of Health Information, Statistics, Research and Evaluation within the MDPH. Six cancer types were evaluated in this investigation, including cancers of the bladder, brain and central nervous system (CNS), kidney, liver, and lung and bronchus as well as leukemia. [Coding for cancer types in this report follows the International Classification of Diseases for Oncology (ICD-O) system. See Appendix A for the incidence coding definitions used in this report for these cancer types.] These cancer types were selected for evaluation based on knowledge of community cancer and environmental concerns and/or potential associations with contaminants of concern at

the W.R. Grace site (e.g. arsenic and vinyl chloride). [Note: while throat cancer was also identified as a community concern, this cancer type was not evaluated in relation to the W.R. Grace site since throat cancer has no known environmental risk factors and the primary cause for the disease is smoking]. Only cases reported to the MCR as a primary cancer for one of the six cancer types and diagnosed among a resident of the town of Acton or Concord CT 3612 were included in the analysis. Cases were identified and included 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 initiation of this analysis.[1]

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Epidemiologic studies have revealed that different types of cancer are individual diseases with separate causes, risk factors, characteristics and patterns of survival (Berg 1996). Cancers are classified by the location in the body where the disease originated (the primary site) and the tissue or cell type of the cancer (histology). Therefore, each of the cancer types reviewed in this report was evaluated separately. Cancers that occur as the result of the metastasis or the

[1] The data summarized in this report are drawn from data entered on MCR computer files before June 21, 2004 for the town of Acton and before June 2, 2006 for Concord CT 3612. 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.

spread of a primary site cancer to another location in the body are not considered as separate cancers and therefore were not included in this analysis.

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. The data in this report have been controlled for duplicate cases by excluding them from the analyses. Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting hospital/diagnostic facility and obtaining additional information regarding the histology and/or pathology of the case. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than 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 Acton, five duplicate reports were identified during the years 1982–2000 and excluded from the analyses. In Concord CT 3612, there was one duplicate report that was excluded.

B. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer cases occurred in the town of Acton or Concord CT 3612, cancer incidence data were tabulated by gender according to 18 age groups to compare the observed number of cancer cases to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for the period 1982–2000 for each of the six primary cancer types in Acton as a whole, in each Acton CT, and in Concord CT 3612. SIRs were also calculated for three smaller time periods (1982–1987, 1988–1993, and 1994–2000) in order to evaluate patterns or trends in cancer incidence over time.

In order to calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 U.S. census data for each census tract in Acton, for the town of Acton as a whole, and for Concord CT 3612 (U.S. DOC 1980, 1990, 2000). The United States Census reports a total population of

17,544 individuals, 17,872 individuals, and 20,331 individuals, respectively, for the 1980, 1990, and 2000 censuses in the Town of Acton. The United States Census reports a total population of 5,197 individuals, 4,511 individuals, and 5,491 individuals, respectively, for the 1980, 1990, and 2000 censuses in Concord CT 3612. Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990 and 1997). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the 10-year interval between each census.[2]

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

According to the U.S. Census, the town of Acton is subdivided into four census tracts (i.e., CTs 3631.01, 3631.02, 3632.01, 3632.02) (U.S. DOC 2000). The majority of the W.R. Grace site is located in Acton CT 3631.01. Part of the site is located in Concord CT 3612, which is adjacent to Acton CT 3631.01. The 2000 United States Census reports a total of 9,035 residents in Acton CT 3631.01. Town boundaries and census tract locations for this analysis are illustrated in Figure 1. Three cases for which census tract designation was not possible were included in the town totals for Acton.

C. 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.

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

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

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

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

D. Calculation of the 95% Confidence Interval

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

chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer cases.

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

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Again, due to the instability of incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

E. Evaluation of Cancer Risk Factor Information

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, smoking history and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to bladder, kidney, and lung and bronchus cancers. Other cancer risk factors may include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with

asbestos, have been shown to be carcinogenic (cancer-causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for residents of the town of Acton and Concord CT 3612 who were diagnosed with any of the six cancer types included 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, and therefore, it was not possible to evaluate them in this investigation.

F. Determination of Geographic Distribution of Cancer Cases

In addition to calculation of SIRs, address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 2004). This allowed assignment of census tract location as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cancer cases in each of the four communities. 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 telephone books issued within two years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. [Note: MDPH is bound by Massachusetts General Laws and the federal Health Insurance Portability and Accountability Act (HIPAA) not to reveal the name or identifying information of an individual diagnosed with cancer whose case is reported to the MCR.]

IV. RESULTS OF CANCER INCIDENCE ANALYSIS

The following sections present cancer incidence rates for Acton, the four Acton census tracts, and Concord CT 3612 during the 19-year time period, 1982–2000 (Tables 1a–6d). To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1987, 1988–1993, and 1994–2000. SIRs were not calculated for some cancer types in smaller time periods due to the small number of observed cases (less than five). However, the expected

number of cases was calculated during each time period, and the observed and expected numbers of cases were compared to determine whether excess numbers of cancer cases were occurring.

In general, with one exception noted below, the six cancer types evaluated in this report occurred approximately at the expected rates in the town of Acton as a whole during the 19-year time period, 1982–2000, as well as smaller time periods (i.e., 1982–1987, 1988–1993, and 1994–2000) (see Tables 1a–1d). Brain and CNS cancer occurred more often than expected during 1982–2000 and during the first two smaller time periods. The observed elevation was statistically significant during the first time period, 1982–1987. Bladder cancer and kidney cancer occurred less often than expected among males and females combined and leukemia and liver cancer occurred about as expected during 1982–2000. Lung and bronchus cancer occurred statistically significantly less often than expected among males and among both sexes combined during 1982–2000. In general, with some exceptions noted below, residents of most Acton census tracts experienced cancer approximately near the rates expected during 1982–2000 and during the smaller time periods evaluated in this report (see Tables 2a–5d).

In Concord CT 3612, most of the six cancer types evaluated generally occurred at or near the rates expected, and several occurred below expected rates (see Tables 6a–6d). One statistically significant elevation was observed in the census tract. Specifically, more leukemia than expected was diagnosed among males and females combined during the middle time period, 1988–1993. This elevation was based on approximately four more diagnoses than expected and resulted in a wide 95% confidence interval.

A. Bladder Cancer

Bladder cancer occurred less often than expected among males and females combined in the town of Acton as a whole during 1982–2000, primarily due to a lower-than-expected rate among males in the town (24 diagnoses observed vs. 30.2 expected, SIR = 79, 95% CI = 51–118) (Table 1a). Females were diagnosed more often than expected during 1982–2000 (15 diagnoses observed vs. 11.3 expected, SIR = 133, 95% CI = 75–220). This elevation was not statistically significant. Males in Acton experienced fewer diagnoses of bladder cancer than expected during each of the smaller time periods evaluated, while females were diagnosed slightly more often than expected during 1982–1987 and 1988–1993 and less than expected during 1994–2000 (see

Tables 1b–1d). Each of the elevations among females represented an excess of about three diagnoses and neither was statistically significant.

While bladder cancer occurred less often than expected in three census tracts in Acton during 1982–2000 (i.e., CTs 3631.01, 3632.01, and 3632.02), the cancer type was diagnosed more often than expected among males and females combined in CT 3631.02 (13 diagnoses observed vs. 8.2 expected, SIR = 159, 95% CI = 85–272). This elevation was not statistically significant (see Tables 3a–3d).

Bladder cancer occurred less often that expected in Concord CT 3612 among males and females combined during 1982–2000 (8 diagnoses observed vs. 14.4 expected, SIR = 55, 95% CI = 24–109). When evaluated separately, males in CT 3612 experienced fewer diagnoses of bladder cancer (5 diagnoses observed vs. 11.3 expected, SIR = 44, 95% CI = 14–104), whereas bladder cancer occurred at about the rate expected among females during 1982–2000 (3 diagnoses observed vs. 3.2 expected). The incidence of bladder cancer occurred near or below the rates expected among both males and females during each of the three smaller time periods (Tables 6a–6d).

B. Brain and Central Nervous System (CNS) Cancer

Brain and CNS cancer occurred more often than expected in the town of Acton as a whole during 1982–2000 (35 diagnoses observed vs. 24.4 expected, SIR = 143) (Table 1a). The observed elevation was of borderline statistical significance (95% CI = 100–199). Both males and females experienced increases in the incidence of brain and CNS cancer during this time period; however, neither elevation was statistically significant. This cancer type was also elevated above the expected during the first two smaller time periods evaluated. Brain and CNS cancer occurred statistically significantly more often than expected based on the state rate among males and females combined during 1982–1987 (14 diagnoses observed vs. 6.9 expected, SIR = 204, 95% CI = 112–343) (Table 1b). The elevation of brain and CNS cancer during 1988–1993 was not statistically significant (14 diagnoses observed vs. 8.3 expected, SIR = 169, 95% CI = 92–284) (Table 1c). During the most recent time period evaluated, 1994–2000, the incidence of brain and CNS cancer among males and females combined occurred less than expected (7 diagnoses observed vs. 9.1 expected) (Table 1d).

During the 19-year time period 1982–2000, brain and CNS cancer occurred approximately at expected rates in three of the census tracts in Acton (i.e., CTs 3631.02, 3632.01, and 3632.02). Residents of CT 3631.01, however, experienced an elevation in the incidence of this cancer type (18 diagnoses observed vs. 10.9 expected, SIR = 165). This observed increase was again of borderline statistical significance (95% CI = 98–261). Increased incidence within this census tract during each of the three smaller time periods was not statistically significant.

In Concord CT 3612, the incidence of brain and CNS cancer was near the rate expected among males and females combined (8 diagnoses observed vs. 7.2 expected) during 1982-2000 (Table 6a). When evaluated separately, the rates among males and females were at or near expected during this time. Specifically, four males were diagnosed with brain or CNS cancer and four were expected; four females were diagnosed with brain or CNS cancer whereas 3.2 were expected. When evaluated by smaller time periods, slightly more brain and CNS cancer was observed among males and females combined during the first time period, 1982–1987, whereas brain and CNS cancer occurred near or below expected during 1988–1993 and 1994–2000 (Tables 6b–6d). The elevation during 1982–1987 was attributed to a slight elevation in this cancer type among females (3 diagnoses observed vs. 1.0 expected).

C. Kidney Cancer

During the 19-year time period, 1982–2000, kidney cancer in the town of Acton as a whole occurred below expected rates (25 diagnoses observed vs. 29.3 expected, SIR = 85, 95% CI = 55–126) (Table 1a). Both males and females in the town were diagnosed less often than expected. Residents of each of the four census tracts experienced kidney cancer near or below the expected rate for this time period and the three smaller time periods. The exception was CT 3631.01 during 1988–1993, when seven diagnoses occurred and 4.2 were expected (SIR = 165, 95% CI = 66–340) (Table 2c). However, this elevation was not statistically significant. Incidence of kidney cancer in this census tract was less than expected during 1982–1987 and 1994–2000 (Tables 2b, 2d).

The incidence of kidney cancer was near the expected rate in Concord CT 3612 during 1982-2000 among males and females combined (10 diagnoses observed vs. 9.5 expected, SIR = 105, 95% CI = 50–194) and when evaluated separately by gender (Table 6a). Slightly more kidney

cancer was observed among males and females during the earliest time period, 1982-1987 (4 diagnoses observed vs. 2.0 expected) (Table 6b). Kidney cancer occurred at or near the rates expected among both males and females during 1988-1993 and 1994-2000 (Tables 6c, 6d).

D. Leukemia

Leukemia was diagnosed about as expected in Acton during 1982–2000 (24 diagnoses observed vs. 25.5 expected, SIR = 94, 95% CI = 60–140) (Table 1a). While females were diagnosed less often than expected (7 diagnoses observed vs. 11.1 expected, SIR = 63, 95% CI = 25–130), males were diagnosed slightly more often than expected (17 diagnoses observed vs. 14.4 expected, SIR = 118, 95% CI = 69–189). This elevation was due to approximately one additional diagnosis among males during each of the three smaller time periods and was not statistically significant (Tables 1b–1d). During 1982–1987 and 1988–1993, the overall rate of leukemia was slightly lower than expected, while leukemia incidence was greater than expected during 1994–2000 (15 diagnoses observed vs. 12.3 expected, SIR = 122, 95% CI = 68–201). The elevation was based on the increased incidence of leukemia in CT 3632.02 during this time period (5 diagnoses observed vs. 2.1 expected, SIR = 235, 95% CI = 76–549) (Table 5d). Neither of these elevations was statistically significant.

Overall rates were also lower than or about as expected in three of the four census tracts in Acton. The exception was CT 3632.02, where six diagnoses of leukemia were observed, while 4.8 were expected from 1982–2000 (SIR = 126, 95% CI = 46–273) (Table 5a). This elevation was due to approximately one additional diagnosis among females in the census tract and was not statistically significant. Similar trends were noted when these data were reviewed by smaller time periods. Except for CT 3632.02 during 1994–2000, as mentioned above, leukemia generally occurred at or near expected rates (i.e., within one or two cases of the expected number) for each of the census tracts during each smaller time period.

A slight elevation in the incidence of leukemia was noted in Concord CT 3612 among males and females combined during 1982–2000 (9 diagnoses observed vs. 7.8 expected, SIR = 116, 95% CI = 53–220) (Table 6a). This elevation was due to two additional diagnoses among females in the census tract and was not statistically significant. As noted previously, a statistically significant elevation in leukemia was observed among males and females combined during the middle time

period, 1988–1993 (6 diagnoses observed vs. 2.2 expected, SIR = 278, 95% CI = 102–606), which was largely due to an elevation among females during this time (4 observed vs. 0.8 expected) (Table 6c). The incidence of leukemia was less than expected during the other two time periods (Tables 6b, 6d).

E. Liver

Liver cancer occurred as expected in Acton during 1982–2000 (7 diagnoses observed vs. 7.0 expected) (Table 1a). When evaluated separately by gender, males were diagnosed with liver cancer less often than expected. Among females, there was about one additional diagnosis above the number expected (3 diagnoses observed vs. 1.8 expected), although this elevation was not statistically significant. When examined by the three smaller time periods, liver cancer occurred about as expected for both genders (Tables 1b–1d). Diagnoses among residents of each census tract also occurred near expected rates (Tables 2a–5d).

In Concord CT 3612, the incidence of liver cancer occurred near or below what would be expected based on the statewide incidence of this cancer type for all time periods evaluated (Tables 6a–6d).

F. Lung and Bronchus

The incidence rate of lung and bronchus cancer was statistically significantly lower than expected in the town of Acton as a whole (111 diagnoses observed vs. 169.8 expected, SIR = 65, 95% CI = 54–79) during the 19-year time period, 1982–2000 (Table 1a). Analysis of trends over time revealed that incidence rates for this cancer type were lower than expected during each of the smaller time periods evaluated and were statistically significantly lower than expected during 1982–1987 and 1994–2000 (Tables 1b–1d).

The incidence of lung and bronchus cancer was lower than expected in all four Acton census tracts during the 19-year time period, 1982–2000 (Tables 2a, 3a, 4a, 5a). Moreover, the rates were statistically significantly lower than expected among males and females combined in CTs 3631.01, 3631.02, and 3632.01 and among males when evaluated separately by gender in CTs 3631.01, 3632.01, and 3632.02. Similar trends were observed when these data were evaluated for smaller time periods, with the exception of CT 3631.02 during 1988–1993 (12 diagnoses

observed vs. 9.3 expected, SIR = 129, 95% CI = 67–226) (Table 3c). This elevation was not statistically significant.

With one exception, the incidence rate of lung and bronchus cancer was lower than expected among both males and females in Concord CT 3612 during all time periods evaluated (Tables 6a–6d). An elevated rate of lung and bronchus cancer was observed among females when evaluated separately during the first time period, 1982–1987 (8 diagnoses observed vs. 4.9 expected, SIR = 165, 95% CI = 71–325) (Table 6b). However, this elevation was not statistically significant, and fewer females than expected were diagnosed with this cancer type during 1988–1993 and 1994–2000 (Tables 6c, 6d).

V. REVIEW OF CANCER RISK FACTOR INFORMATION

As previously mentioned, cancer is not just one disease, but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (the time between exposure and development of disease), characteristics, and trends in survival. Available information from the MCR related to age and gender, as well as other factors related to the development of cancer such as smoking and occupation, was reviewed for individuals diagnosed with cancer in the town of Acton and Concord CT 3612. Information for each of the six cancer types evaluated in this report was compared to known or established incidence trends to assess whether any unexpected patterns exist among these cases. It is important to note, however, that personal risk factors such as family history, pre-existing medical conditions, hormonal events, diet, and other factors also influence the development of these cancer types. This information is not collected by the MCR or any other readily accessible source, and therefore, it was not possible to evaluate the role these types of risk factors may have played in the incidence of cancer in Acton or Concord CT 3612 in this investigation. For detailed information regarding risk factors associated with the cancer types evaluated in this report, please refer to Appendix B.

Age and gender are risk factors in many types of cancers, including bladder cancer, brain and CNS cancer, kidney cancer, liver cancer, lung and bronchus cancer, and leukemia. Therefore, a

review of age group-specific SIRs was conducted. Where numbers of cases in each age group were too small to calculate SIRs, the distribution of cases by age was reviewed.

Tobacco use is also a known or suggested causal risk factor in several types of cancer, including bladder cancer, kidney cancer, and lung and bronchus cancer. The smoking history of individuals diagnosed with these cancer types in Acton and Concord CT 3612 was reviewed to assess the role tobacco smoking may have played in the development of these cancers among residents. However, results of smoking history analysis should be interpreted with caution because of the number of individuals for which smoking status was unknown.

In some studies, an association has been found with exposures to specific occupations and an increase in the incidence of bladder cancer, brain and CNS cancer, kidney cancer, leukemia, liver cancer, and lung and bronchus cancer. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these cancer types to determine the role that occupational factors may have played in the development of these cancers in Acton and in Concord CT 3612. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. Further, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

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

A. Bladder Cancer

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

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

Studies have revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20–25% of all bladder cancers diagnosed among men in the 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, as well as 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, who are potentially exposed to motor exhaust for extended periods of time), 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).

1. Age and Gender

A review of individuals diagnosed with bladder cancer in Acton from 1982–2000 revealed that the majority of diagnoses in the town were male (62%, n = 24). In Concord CT 3612, five of the eight bladder cancer diagnoses were among males. Males comprised 72% of bladder cancer diagnoses statewide for this time period. The mean age at diagnosis in Acton was 68 years, and the mean age in Concord CT 3612 was 71 years. Both are consistent with statewide bladder cancer trends (mean age = 70 years).

2. *Smoking History*

Of the 39 individuals in Acton who were diagnosed with bladder cancer during the years 1982–2000, 35 reported a smoking status. Of those with a reported smoking status, 63% (n = 22) reported being current or former tobacco users and 37% (n = 13) reported no tobacco use. Tobacco history information was available for seven of the eight individuals diagnosed with bladder cancer in Concord CT 3612. Of these individuals, five reported being current or former tobacco users and two reported no tobacco use. During this same time period, 67% of bladder cancer cases in the state of Massachusetts with a known smoking status were current/former tobacco users and 33% reported no tobacco use.

3. *Occupation*

Review of occupation for individuals diagnosed with bladder cancer in Acton revealed that none of the individuals reported working at a job in which occupational exposures potentially related to the development of bladder cancer may have been possible. Occupations reported for the individuals diagnosed are not likely to be related to an increased risk of this cancer type. However, occupation was reported as retired or unknown for many of these individuals (33%, n = 13). Occupation was reported as retired or unknown for half (n = 4) of the eight individuals diagnosed with bladder cancer in Concord CT 3612, and one individual reported an occupation where exposures related to the development of bladder cancer could have been possible.

B. Brain and Central Nervous System (CNS) Cancer

According to epidemiological literature, brain tumor incidence (cancerous and noncancerous) declines after a peak in childhood (under 10 years of age), increases from age 25 to 75, and levels off after age 75 (Preston-Martin and Mack 1996). Certain types of brain tumors are more likely to develop in children and others are more typically seen in adults (Black 1991, NCI 1996). In the state of Massachusetts as a whole, diagnoses of brain and CNS cancer occurred roughly equally between genders (51% were among males and 49% were among females).

Various studies on worker exposure to vinyl chloride and chemicals in the petrochemical industry have had conflicting results as to the association between these chemicals and the development of brain tumors. Studies investigating the possible association between parental

occupational exposures (e.g., paper or pulp mill, aircraft, rubber, and electric workers) and the onset of brain tumors (cancerous and noncancerous) in their children have also provided inconsistent results (Preston-Martin and Mack 1996).

1. *Age and Gender*

From 1982 to 2000 in Acton, the age at diagnosis for individuals with brain and CNS cancer was consistent with the pattern expected based on the scientific literature for this cancer type. All individuals diagnosed with brain or CNS cancer were diagnosed prior to age 10 or after age 25. The townwide elevation in the incidence of brain and CNS cancer was due to increased incidence among both males and females in Acton during 1982–2000. Review of age group-specific SIRs suggests that this elevation was not the result of increased diagnoses among males in any one age group. Females generally experienced brain and CNS cancer approximately at or below the expected rates, with the exception of females aged 35–39 years who were diagnosed more often than expected (5 diagnoses observed vs. 0.7 expected).

In Concord CT 3612, the age of individuals at the time of diagnosis ranged from 23 years to 85 years which was also consistent with the scientific literature for brain and CNS cancer. Of the eight brain or CNS cancer diagnoses during 1982-2000, four were among males and four were among females.

2. *Histology*

Of the 35 individuals diagnosed with brain and CNS cancer in Acton during 1982–2000, 83% (n = 29) had brain cancer and 17% (n = 6) had CNS cancer. This distribution was very similar to the statewide distribution of brain and CNS cancer. Among individuals diagnosed with brain and CNS cancer in Massachusetts during this time period, 81% had brain cancer and 19% had CNS cancer. According to ACS, 90% of brain tumors in adults older than 45 years are gliomas, a general category that includes glioblastomas, astrocytomas, oligodendrogliomas, and ependymomas (ACS 2004). In Acton, 14 out of 18 (78%) adults older than 45 years with brain cancer were diagnosed with gliomas. Among malignant brain tumors, the most common type is glioblastoma, which is a type of glioma (ACS 2004). During 1982–2000, glioblastomas represented about 45% of the primary brain cancers diagnosed among adults and children in

Massachusetts. In Acton during this same time period, 28% (n = 8) of primary brain cancers diagnosed in adults and children were glioblastomas. The distribution of brain cancer in Acton was slightly different than the statewide distribution; however, considering the relatively small number of total diagnoses in the town of Acton compared to the state as a whole during 1982–2000, the overall pattern does not appear to be atypical. Of the two remaining types of brain cancer diagnosed in adults in Acton, one was meningioma in four adults (16%) in their late 50s and older. Of these four individuals, three were female and one was male. According to ACS, meningiomas account for 20–40% of primary brain tumors in adults and are most often diagnosed in women and in older individuals (ACS 2004). The other type of brain cancer was a nerve sheath tumor diagnosed in one adult. The four children diagnosed with brain or CNS cancer in Acton all had cancer of the brain, and three different histology types were diagnosed.

In all, the pattern of brain and CNS cancer in Acton adults was fairly consistent with the pattern seen in the general population, and a variety of types of brain cancer were represented among children and adults, indicating no atypical pattern of any one type from 1982–2000.

Of the eight individuals diagnosed with brain and CNS cancer in Concord CT 3612 during 1982–2000, seven had brain cancer and one had CNS cancer. Each of the seven individuals with brain cancer was diagnosed with a type of glioma, and of these, four were diagnosed with the glioblastoma subtype. These patterns are consistent with the statewide experience for brain cancer.

3. Occupation

Among adults in Acton diagnosed with brain or CNS cancer, an occupation was reported for 24 individuals (77% of adult cases). Two of the individuals might have worked at a job in which occupational exposures potentially related to the development of brain and CNS cancer may have been possible. However, occupation was reported as retired or unknown for 23% of adults diagnosed with brain and CNS cancer (n = 7). Two of the four diagnoses among children reported occupation information for one parent. Based on the available information, parental occupation was not likely associated with exposure to the chemicals listed above.

In Concord CT 3612, an occupation was reported for five of the eight individuals diagnosed with brain or CNS cancer. Two of the individuals reported an occupation in which occupational exposures potentially related to the development of brain or CNS cancer may have been possible. Occupation was reported as retired or unknown for three individuals.

C. Kidney Cancer

Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in the fifth and sixth decades of life (50–70 year age group) (ACS 2001a). The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 30% to 100% (ACS 2001a). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of those in women may be caused by cigarette smoking (ACS 2001a).

Although kidney cancer is not generally considered an occupationally associated cancer, some studies have suggested that environmental and occupational factors may be associated with its development. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. In addition, exposure to cadmium is associated with an increased incidence of kidney cancer, particularly among men who smoke. Workplace exposure to organic solvents, such as trichloroethylene (TCE), may also increase the risk of this cancer (ACS 2001a). More recently, renal cell carcinoma (RCC), the most common type of kidney cancer, has been suggested to be associated with occupational exposure to petroleum and 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).

1. *Age and Gender*

The average age of individuals diagnosed with kidney cancer in Acton during 1982–2000 was 61 years. Eighty-four percent (n = 21) were age 50 or older at the time of diagnosis. These age patterns are consistent with what would be expected in the general population. Overall, males experienced kidney cancer at about the rate expected while females were diagnosed less often than expected based on the state rate. Review of age group-specific SIRs revealed that males were diagnosed about as expected. Females in most age groups were diagnosed with kidney cancer approximately at or below rates expected.

The age and gender patterns of those diagnosed with kidney cancer in Concord CT 3612 were also consistent with what would be expected in the general population. Specifically, the average age at diagnosis was 65 years, and nine of the ten individuals were age of 50 or older at the time of diagnosis. Six of the ten individuals with kidney cancer were male and four were female.

2. *Smoking History*

Of the 25 individuals diagnosed with kidney cancer in Acton during 1982–2000, information on tobacco use was reported for 23 individuals. Of those individuals, 48% (n = 11) reported being current or former smokers at the time of diagnosis and 52% (n = 12) were nonsmokers. This pattern differed slightly from the statewide pattern of kidney cancer during this time period. In Massachusetts, 58% of individuals diagnosed with kidney cancer with a known smoking status were current or former smokers at the time of diagnosis and 42% of those with a known smoking status reported being nonsmokers.

In Concord CT 3612, seven of the ten individuals diagnosed with kidney cancer during 1982–2000 reported being current or former smokers at the time of diagnosis, and three reported never using tobacco.

3. *Occupation*

Most individuals diagnosed with kidney cancer in Acton during 1982–2000 did not report working at a job in which occupational exposures possibly associated with kidney cancer would

have been likely. However, one individual reported an occupation in which an increased risk is possible. Occupation was reported as retired or unknown for 24% of those diagnosed (n = 6).

Occupation was reported as retired or unknown for six of the ten individuals diagnosed with kidney cancer in Concord CT 3612. None of the four remaining individuals reported working in an occupation where exposures possibly associated with kidney cancer would have been likely.

D. Leukemia

In 2006, leukemia is expected to have affected approximately 35,070 individuals (20,000 males and 15,070 females) in the United States, resulting in 22,280 deaths (ACS 2006). In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2003, representing more than 2% of all cancer diagnoses (ACS 2006). There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also several rare types of leukemia (e.g., hairy cell leukemia, myelomonocytic leukemia). In adults, the most common types are AML and CLL. Leukemia is the most common type of childhood cancer, accounting for more than 30% of all cancers diagnosed in children. The majority of these cases are of the ALL type (ACS 2003).

The various subtypes of leukemia occur with different frequencies in the population. For the purpose of classification in this evaluation, if the histology (i.e., cell type) of the leukemia diagnosis was not otherwise specified or not classified as one of the four main subtypes, then the individual case was categorized as "other." Available information regarding the expected distribution of leukemia by histology types can vary considerably depending on coding methods, making comparisons of type-specific incidence rates from different cancer registries difficult (Linet and Cartwright 1996). In the state of Massachusetts during the time period 1982–2000, 33.8% of all leukemia cases were AML, 26.5% were CLL, 12.9% were ALL, 10.7% were CML, and 16.0% were other histology types.

Several occupational exposures have been identified as playing a role in the development of leukemia. For example, exposures to particular chemicals are thought to increase the risk of developing certain kinds of leukemia. Exposure to ionizing radiation, chronic, high-dose

exposure to pesticides, and other chemicals such as benzene, have also been suggested as possible risk factors for leukemia (Linet and Cartwright 1996). Chronic occupational exposure to benzene has been established as a cause of AML. High doses of radiation among survivors of atomic bomb blasts or nuclear reactor accidents are associated with an increased incidence of AML, CML, and ALL, but no association has been established for lower doses such as those used in medical diagnostics.

1. *Age and Gender*

The average age of individuals diagnosed with leukemia in Acton was 56 years. Sixty-three percent (n = 15) were age 50 or older at the time of diagnosis. Three individuals (13%) were between the ages of 0 and 19 years at diagnosis, while approximately four diagnoses were expected for this age group. Overall, there was a slight elevation in the incidence of this cancer type among males in the town, while no females were diagnosed. Among males, increased rates were noted among individuals aged 65–69 years. Females in most age groups were diagnosed with leukemia less often than expected.

In Concord CT 3612, the average age of individuals diagnosed with leukemia was 48 years, and five of the nine cases were age 50 or older at diagnosis. One of the individuals with leukemia was a child between the ages of 0 and 19 years. There were four males and five females diagnosed with leukemia in this census tract during 1982–2000.

2. *Histology*

Of the 24 individuals diagnosed with leukemia in Acton during 1982–2000, 37.5% were diagnosed with the AML subtype, 25.0% were diagnosed with CLL, 16.7% were diagnosed with ALL, 4.2% were diagnosed with CML, and 16.7% were diagnosed with other types of leukemia. This distribution is similar to that seen statewide, with the exception that the CML subtype comprised a relatively larger proportion of leukemia diagnoses. However, considering the relatively small number of total diagnoses in the town of Acton compared to the state as a whole during the 1982–2000 time period, the overall pattern does not appear to be atypical. Two of the three children aged 0 to 19 that were diagnosed with leukemia in Acton were diagnosed with the

ALL subtype, the most common subtype among children. The other child was diagnosed with AML.

Of the nine individuals diagnosed with leukemia in Concord CT 3612 during 1982–2000, two were diagnosed with the AML subtype, three were diagnosed with CLL, one was diagnosed with ALL, and three were diagnosed with CML. While the proportion of CML diagnoses in Concord CT 3612 is also slightly higher than that seen statewide, the pattern does not appear to be unusual considering the ages of the individuals and the number of total leukemia diagnoses in the census tract compared to the state as a whole.

3. Occupation

Review of occupation for individuals diagnosed with leukemia in Acton revealed that two individuals may have worked jobs in which occupational exposures potentially related to the development of leukemia may have been possible. However, information regarding specific job duties that could help to further define exposure potential for these individuals was not available. Occupations reported for the remaining individuals are not likely to be related to an increased risk of this cancer type. Occupation was reported as retired, unknown, or "at home" for 13% of these individuals (n = 3).

An occupation was reported for six of the eight adults diagnosed with leukemia in Concord CT 3612 during 1982-2000. Two of these individuals reported an occupation in which exposures possibly related to development of leukemia may have been present. Occupation was reported as unknown for two adults diagnosed with leukemia in this census tract.

E. Liver Cancer

An estimated 18,510 people in the U.S. (12,600 men and 5,910 women) will be diagnosed with liver cancer in 2006, accounting for approximately 1% of all new cancers (ACS 2006). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Men are at least two to three times more likely to develop liver cancer than women (Yu et al. 2000). Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn 1996). While chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk

factors for developing liver cancer (ACS 2001b), epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2001b; London and McGlynn 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. In addition, arsenic has been associated with an increased risk of liver cancer (ATSDR 2000).

1. Age and Gender

Among the seven individuals diagnosed with liver cancer in Acton during 1982–2000, the average age at diagnosis was 60 years, which is consistent with trends for this cancer type in the general population. The majority of individuals (71%, n = 5) were age 50 or older at the time of diagnosis. Among males, there was approximately one fewer diagnosis than expected. Among females, there was about one additional diagnosis over the expected number.

One individual was diagnosed with liver cancer while living in Concord CT 3612 during 1982–2000. This person was over 50 years of age at the time of diagnosis.

2. Occupation

Occupational information available for three of the seven individuals diagnosed with liver cancer in Acton did not suggest that occupation was likely to be associated with an increased risk of their developing liver cancer. Occupation was unknown for four individuals.

No information on occupation was reported for the individual diagnosed with liver cancer while living in Concord CT 3612.

F. Lung and Bronchus Cancer

According to epidemiological literature, the incidence of lung cancer increases sharply with age and peaks around age 60–70. Only 2% of lung cancers occur before the age of 40. In addition, lung cancer is generally observed more often among men than women (Blot and Fraumeni 1996, MCR 2002).

Lung cancer is divided into two main types: small cell lung cancer and non-small cell lung cancer. Non-small cell lung cancer is further sub-divided into three types: adenocarcinoma, squamous cell carcinoma, and large-cell undifferentiated carcinoma. The different types of lung cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung cancers are adenocarcinomas, 25–30% are squamous cell carcinomas, 20% are small cell cancers, and 10–15% of cases are large cell carcinomas (ACS 2002). Rates in Massachusetts are very similar to those seen nationally.

About 87% of all lung cancers are thought to be caused directly by smoking cigarettes or by exposure to second hand smoke, or environmental tobacco smoke (ACS 2002). An increased prevalence of cigarette smoking among women has produced lung cancer incidence rates that more closely resemble those historically experienced by males. The risk of developing lung cancer depends on the intensity of one's smoking habits (e.g., duration of habit, amount smoked, tar yield of cigarette, and filter type). Smoking cessation decreases the elevated risk by about 50%; however, former smokers still carry a greater risk of developing lung cancer than those who have never smoked.

Several occupational exposures have been identified as playing a role in the development of lung cancer. For example, workplace exposure to asbestos is an established risk factor for this disease (ACS 2002). Underground miners exposed to radon and uranium are also at an increased risk for developing lung cancer (ACS 2002; Samet and Eradze 2000). Other occupations potentially associated with this cancer include chemical workers, talc miners and millers, paper and pulp workers, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers. In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational, and in some cases environmental, risk factors for lung cancer. Occupational/environmental exposure to these compounds in conjunction with cigarette smoking can dramatically increase the risk of developing lung cancer (Blot and Fraumeni 1996).

1. *Age and Gender*

Among the 111 individuals diagnosed with lung and bronchus cancer in Acton during 1982–2000, the average age at diagnosis was 67 years, which is the same as the average age observed statewide. Only one individual (0.9%) was under the age of 40 at the time of diagnosis. Overall, during the 19-year time period 1982–2000, both males and females experienced decreased rates of lung and bronchus cancer compared to state rates, with the rate among males statistically significantly lower than would be expected based on the statewide rate. Males in all age groups were diagnosed with lung and bronchus cancer approximately equal to or less often than expected in Acton during 1982–2000. Females were diagnosed with lung and bronchus cancer at approximately the rate expected or less frequently than expected in most age groups. Females aged 50–54, 70–74, and over 80 years were diagnosed more often than expected.

Age and gender patterns in Concord CT 3612 were also consistent with trends observed statewide. Specifically, the average age of individuals diagnosed with lung and bronchus cancer during 1982–2000 was 66 years. Sixty-five percent (n = 28) of the diagnoses were among males and 35% (n = 15) were among females.

2. *Histology*

Of the 91 lung and bronchus cancer diagnoses in Acton with a specific histology classification, 42% were diagnosed as adenocarcinomas, 27% were squamous cell carcinomas, 19% were small cell cancers, and 12% were large cell carcinomas. The observed distribution is consistent with established prevalence patterns of disease for lung and bronchus cancer in the general population.

The histology distribution of lung and bronchus cancer in Concord CT 3612 was generally similar to state and national trends for this disease, considering the relatively small number of total diagnoses in the census tract compared to the state as a whole during 1982–2000. Ninety-one percent (n = 39) of individuals diagnosed with lung and bronchus cancer in Concord CT 3612 had a specific histology classification. Of these, 46% (n = 18) were diagnosed with adenocarcinomas, 23% (n = 9) were diagnosed with squamous cell carcinomas, 23% (n = 9) were small cell carcinomas, and 8% (n = 3) were large cell carcinomas.

3. *Smoking History*

Of the 111 individuals diagnosed with lung and bronchus cancer in Acton during 1982–2000, 97 reported a smoking status. Of those individuals with a known tobacco history, 92% (n = 89) were current or former smokers and 8% (n = 8) were nonsmokers. This pattern was nearly identical to the statewide pattern of lung cancer during this time period. Specifically, 92% of individuals diagnosed with lung and bronchus cancer in the state of Massachusetts with a known smoking status reported being current or former tobacco users and 8% reported no tobacco use.

In Concord CT 3612, 42 of the 43 individuals diagnosed with lung and bronchus cancer during 1982–2000 reported a smoking status. Of the individuals with a known smoking status, 95% (n = 40) reported being current or former smokers at the time of diagnosis and 5% (n = 2) of individuals reported never using tobacco.

4. *Occupation*

The majority of individuals diagnosed with lung and bronchus cancer in Acton did not report working in jobs likely to have played a role their cancer diagnosis. However, 15 individuals (14%) reported occupations where exposures to asbestos or other chemical compounds possibly associated with lung and bronchus cancer may have been possible. Occupation was reported as retired or unknown for 28% (n = 31) of the individuals. Therefore, it is difficult to determine the role that occupation may have played in the incidence of lung and bronchus cancer in Acton.

In Concord CT 3612, three individuals (7%) reported an occupation where exposures possibly associated with lung and bronchus cancer may have occurred. Occupation was reported as retired or unknown for 12 individuals (28%).

VI. ANALYSIS OF GEOGRAPHIC DISTRIBUTION OF CANCER INCIDENCE

In addition to determining incidence rates for each cancer type, a qualitative evaluation of the geographic pattern of cancer diagnoses was also conducted for the town of Acton, its census tracts, and Concord CT 3612. Place of residence at the time of diagnosis was mapped for each

individual diagnosed with the six cancer types evaluated in this report to assess any possible geographic concentrations of cases in relation to each other or in the vicinity of the W.R. Grace site. As previously mentioned, cancer is one word that describes many different diseases, each with a unique set of risk factors. Therefore, for the purposes of this evaluation, the geographic distribution of each cancer type was evaluated separately to determine whether an atypical pattern of any one type was occurring.

For the majority of cancer types evaluated, review of the geographic distribution of the residences of individuals diagnosed with cancer in Acton revealed no apparent spatial patterns at the neighborhood level that are not likely to be attributed to factors such as areas of higher population density. Brain and CNS cancer was statistically significantly elevated in the town of Acton as a whole during the first time period, 1982–1987, and the diagnoses were fairly evenly distributed throughout the town and seemed to coincide closely with the pattern of population. However, there were a few locations in Acton where two or three individuals with brain and CNS cancer were located in relative close proximity to each other, with some located in the southern area of town near the W.R. Grace site. Therefore, case-specific information available from the MCR was reviewed for individuals in these locations to evaluate whether a common factor might be possible. An evaluation of the dates of diagnosis did not suggest any temporal trends among individuals diagnosed with brain and CNS cancer, and a variety of histology types were represented among individuals located near to each other indicating that a common factor among individuals in these locations was unlikely.

In addition to reviewing case specific information from the MCR, residential histories were constructed for Acton residents diagnosed with brain and CNS cancer between 1982 and 2000 in order to determine how long they lived in their residence prior to diagnosis. Although it is not possible to determine what may have caused any one person's diagnosis with cancer, the length of time in which an individual lived in a particular residence can also help determine the importance that their location might have in terms of exposure to a potential environmental source. Because shorter periods of residence can represent a significant portion of a child's life, residential histories for children ages 0–19 were evaluated separately. Information for residential histories was obtained from annual resident lists for the town of Acton (Town of Acton 1961–2000).

Brain and CNS cancer is often described as having a lengthy latency, or development period, sometimes in excess of 20 years. The latency period is the time between exposure to a potentially harmful substance and the development of the cancer (Preston-Martin & Mack 1996). Based on the information reviewed for the 31 adults diagnosed with brain and CNS cancer in Acton between 1982–2000, 48% (n = 15) lived at their address for less than 10 years prior to diagnosis, 13% (n = 4) lived at their address between 10–19 years prior to diagnosis, and 29% (n = 9) lived at their address for 20 or more years prior to diagnosis. Residential histories could not be confirmed for 10% (n = 3) of the individuals and, therefore, it is probable that these individuals lived at their address for a short time (e.g., less than 1 year) prior to diagnosis. A range in the length of residence was observed among residents diagnosed with brain and CNS cancer who were located in close proximity to each other. This includes those that lived near the W.R. Grace site, who all lived at their reported residence for less than 20 years.

Therefore, a review of case specific information (e.g., dates of diagnosis and/or histology type), together with an evaluation of the length of residence among individuals located in close proximity to each other, did not reveal any common patterns that would suggest that the environment played a primary role among individuals diagnosed with brain and CNS cancer in Acton.

In Concord CT 3612, the geographic distribution of the six cancer types evaluated also seemed to coincide closely with population density, and there were no apparent concentrations of diagnoses in any one area of the census tract. While a statistically significant elevation in leukemia was observed among males and females combined during 1988–1993 in CT 3612, none of the individuals were unusually concentrated in any one area of the census tract during this time period. In addition, none of the six cancer types were concentrated in neighborhoods closest to the Acton town line or in the vicinity of the W.R. Grace site.

VII. DISCUSSION

Most of the cancer types evaluated in this report were selected based on their potential association with contaminants of concern identified at the W.R. Grace site. In general, cancer incidence in Acton and in Concord CT 3612 during the 19-year time period 1982–2000 and the smaller time periods evaluated was approximately at or near expected rates for the six cancer

types. In the town of Acton, one statistically significant elevation was observed. Specifically, in the town as a whole, the incidence of brain and CNS cancer in males and females combined was statistically significantly elevated during the first time period, 1982–1987 (14 diagnoses observed vs. 6.9 expected, SIR = 204, 95% CI = 112–343) (Table 1a). Lung and bronchus cancer occurred statistically significantly less often than expected among males and among males and females combined during 1982–2000. While females in Acton were diagnosed with bladder cancer slightly more often than expected during 1982–2000, the elevation was not statistically significant and there were no consistent trends over time. Residents of most Acton census tracts, including CT 3631.01 where the W.R. Grace site is located, generally experienced the six cancer types near the rates expected during 1982–2000 and during the smaller time periods evaluated in this report. There were no statistically significant elevations among the four Acton census tracts.

In Concord CT 3612, most of the six cancer types evaluated also generally occurred at or near the rates expected and several occurred below expected rates. A statistically significant elevation among males and females diagnosed with leukemia was observed in the census tract during the middle time period, 1988–1993 (6 diagnoses observed vs. 2.2 expected, SIR = 278, 95% CI = 102–606) (Table 6c). Leukemia diagnoses occurred less than or as expected during the other two time periods (Tables 6b, 6d).

Available risk factor information for individuals diagnosed with cancer in Acton and in Concord CT 3612 was compared to known or established trends to assess whether any unexpected patterns exist in these areas. In general, trends observed in Acton and Concord CT 3612 were similar to those seen in the general population. Review of this data suggests that smoking likely played some role in the diagnosis of some cancer types (e.g., cancers of the bladder, kidney, and lung and bronchus) among some individuals. Also, occupational exposures may have been important in the development of cancer among some individuals. However, because of the large number of individuals for whom smoking history and/or occupation was unknown, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in Acton and in Concord CT 3612.

Finally, analysis of the geographic distribution of place of residence for individuals diagnosed with cancer did not reveal any atypical spatial or temporal patterns that would suggest a common factor (environmental or nonenvironmental) played a primary role in the incidence of cancer in Acton or in Concord CT 3612. While there were a few locations in Acton where two or three individuals with brain and CNS cancer were located in relative close proximity to each other, a review of case-specific information (e.g., dates of diagnosis and/or histology type), together with an evaluation of the individuals' length of residence did not reveal any temporal trends or common patterns among diagnoses. Although a statistically significant elevation of leukemia was observed in Concord CT 3612 during 1988–1993, there were no unusual geographic concentrations of diagnoses. In addition, no diagnoses of the six cancer types in CT 3612 were concentrated in neighborhoods closest to the Acton town line or in relation to the W.R. Grace site.

Based on the information reviewed in this evaluation, it does not appear that environmental exposures played a major role in the overall incidence of cancer in the town of Acton, in Concord CT 3612, or in relation to the W. R. Grace site during the 19-year time period 1982–2000.

VIII. LIMITATIONS

This health consultation 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 Acton, its individual census tracts, or Concord census tract 3612.

IX. CONCLUSIONS

- In general, the six cancer types evaluated in this report (leukemia and cancers of the bladder, brain and CNS, kidney, liver, and lung and bronchus) occurred approximately at or near the expected rates for Acton, its individual census tracts, and Concord CT 3612 during the 19-year time period 1982–2000.

- Statistically significant elevations were observed for brain and CNS cancer in the town of Acton as a whole during the first time period, 1982–1987, and for leukemia in Concord CT 3612 during the middle time period, 1988–1993. While females in Acton were diagnosed with bladder cancer slightly more often than expected during 1982–2000, the elevation was not statistically significant and there were no consistent trends over time.

- Review of the geographic distribution of individuals diagnosed with the six cancer types in the town of Acton and in Concord CT 3612 revealed no apparent spatial patterns at the neighborhood level that would suggest that a common factor (environmental or nonenvironmental) played a primary role in the incidence of cancer in these areas or in relation to the W.R Grace site.

- Review of available risk factor information for individuals diagnosed with cancer (e.g., age, gender, smoking history, and occupation) suggests that the trends observed in Acton and in Concord CT 3612 are similar to those seen in the general population. This information also suggests that smoking and, to a lesser extent, occupation, likely played some role in the incidence of some cancer types.

X. RECOMMENDATIONS

- The MDPH/BEH will continue to monitor the incidence of cancer in the town of Acton and in Concord CT 3612 through the Massachusetts Cancer Registry.

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Figure 1: Area of Analysis
Town of Acton and Concord Census Tract (CT) 3612.00
Massachusetts

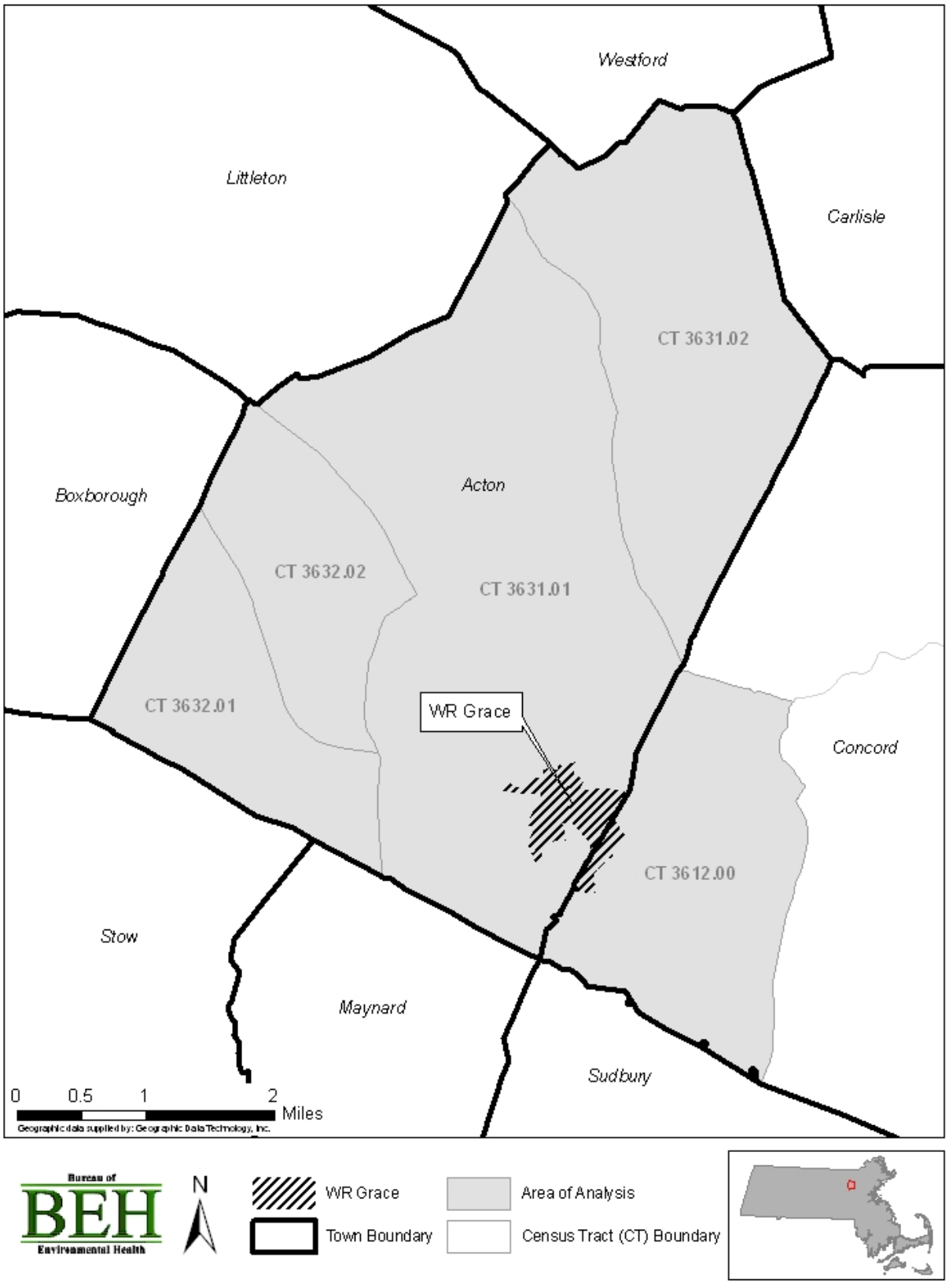

TABLE 1a
Cancer Incidence
Acton, MA
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	39	41.5	94	67 -- 129	24	30.2	79	51 -- 118	15	11.3	133	75 -- 220
Brain & CNS	35	24.4	143	100 -- 199	19	12.8	148	89 -- 231	16	11.6	138	79 -- 224
Kidney & Renal Pelvis	25	29.3	85	55 -- 126	17	18.3	93	54 -- 148	8	11.0	73	31 -- 144
Leukemia	24	25.5	94	60 -- 140	17	14.4	118	69 -- 189	7	11.1	63	25 -- 130
Liver	7	7.0	100	40 -- 207	4	5.2	NC	NC -- NC	3	1.8	NC	NC -- NC
Lung & Bronchus	111	169.8	65*	54 -- 79	47	100.4	47*	34 -- 62	64	69.5	92	71 -- 118

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
CNS = Central Nervous System

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

TABLE 1b
Cancer Incidence
Acton, MA
1982-1987

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	12	11.8	102	52	177	6	8.6	70	25	152	6	3.2	187	68	406
Brain & CNS	14	6.9	204*	112	343	7	3.5	198	79	408	7	3.3	211	84	434
Kidney& Renal Pelvis	4	5.9	NC	NC	NC	2	3.6	NC	NC	NC	2	2.3	NC	NC	NC
Leukemia	4	6.3	NC	NC	NC	4	3.6	NC	NC	NC	0	2.7	NC	NC	NC
Liver	2	1.2	NC	NC	NC	1	0.8	NC	NC	NC	1	0.4	NC	NC	NC
Lung & Bronchus	26	40.9	64*	42	93	12	26.2	46*	24	80	14	14.7	95	52	160

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

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

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

TABLE 1c
Cancer Incidence
Acton, MA
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	13	12.6	103	55 -- 176	7	9.2	76	31 -- 157	6	3.5	173	63 -- 376
Brain & CNS	14	8.3	169	92 -- 284	9	4.2	214	98 -- 407	5	4.1	122	39 -- 286
Kidney & Renal Pelvis	9	9.4	95	44 -- 181	6	6.0	101	37 -- 219	3	3.5	NC	NC -- NC
Leukemia	5	7.2	70	22 -- 163	5	4.1	121	39 -- 283	0	3.0	NC	NC -- NC
Liver	1	1.9	NC	NC -- NC	0	1.4	NC	NC -- NC	1	0.5	NC	NC -- NC
Lung & Bronchus	43	52.5	82	59 -- 110	18	31.4	57*	34 -- 91	25	21.1	119	77 -- 175

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
CNS = Central Nervous System

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

TABLE 1d
Cancer Incidence
Acton, MA
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	14	16.5	85	46 -- 142	11	12.2	90	45 -- 162	3	4.4	NC	NC -- NC
Brain & CNS	7	9.1	77	31 -- 159	3	5.1	NC	NC -- NC	4	4.0	NC	NC -- NC
Kidney & Renal Pelvis	12	14.0	86	44 -- 150	9	8.8	102	47 -- 194	3	5.2	NC	NC -- NC
Leukemia	15	12.3	122	68 -- 201	8	6.9	116	50 -- 229	7	5.4	129	52 -- 265
Liver	4	4.0	NC	NC -- NC	3	3.1	NC	NC -- NC	1	0.9	NC	NC -- NC
Lung & Bronchus	42	75.0	56*	40 -- 76	17	41.8	41*	24 -- 65	25	33.2	75	49 -- 111

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
CNS = Central Nervous System

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

TABLE 2a
Cancer Incidence
CT 3631.01, Acton, MA
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	12	18.0	67	34 -- 117	9	13.7	66	30 -- 125	3	4.3	NC	NC -- NC
Brain & CNS	18	10.9	165	98 -- 261	9	5.9	151	69 -- 288	9	5.0	181	83 -- 344
Kidney & Renal Pelvis	13	13.2	98	52 -- 168	8	8.6	93	40 -- 183	5	4.6	109	35 -- 255
Leukemia	9	11.0	81	37 -- 155	7	6.6	106	43 -- 219	2	4.5	NC	NC -- NC
Liver	3	3.1	NC	NC -- NC	0	2.4	NC	NC -- NC	3	0.7	NC	NC -- NC
Lung & Bronchus	56	76.0	74*	56 -- 96	22	46.8	47*	29 -- 71	34	29.2	116	81 -- 163

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
CNS = Central Nervous System

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

TABLE 2b
Cancer Incidence
CT 3631.01, Acton, MA
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	4.9	NC	NC -- NC	2	3.7	NC	NC -- NC	2	1.2	NC	NC -- NC
Brain & CNS	6	3.1	196	71 -- 426	3	1.6	NC	NC -- NC	3	1.4	NC	NC -- NC
Kidney & Renal Pelvis	1	2.6	NC	NC -- NC	0	1.7	NC	NC -- NC	1	0.9	NC	NC -- NC
Leukemia	2	2.7	NC	NC -- NC	2	1.6	NC	NC -- NC	0	1.1	NC	NC -- NC
Liver	1	0.5	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.1	NC	NC -- NC
Lung & Bronchus	13	17.9	73	39 -- 124	6	11.7	51	19 -- 111	7	6.2	113	45 -- 233

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
CNS = Central Nervous System

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

TABLE 2c
Cancer Incidence
CT 3631.01, Acton, MA
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	1	5.5	NC	NC -- NC	1	4.1	NC	NC -- NC	0	1.3	NC	NC -- NC
Brain & CNS	8	3.7	218	94 -- 429	5	1.9	258	83 -- 601	3	1.7	NC	NC -- NC
Kidney & Renal Pelvis	7	4.2	165	66 -- 340	4	2.8	NC	NC -- NC	3	1.4	NC	NC -- NC
Leukemia	2	3.1	NC	NC -- NC	2	1.9	NC	NC -- NC	0	1.2	NC	NC -- NC
Liver	1	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.2	NC	NC -- NC
Lung & Bronchus	20	24.0	85	52 -- 131	9	14.7	61	28 -- 116	11	8.8	125	62 -- 223

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
CNS = Central Nervous System

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

TABLE 2d
Cancer Incidence
CT 3631.01, Acton, MA
1994-2000

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	7	7.4	95	38	196	6	5.6	107	39	234	1	1.8	NC	NC	NC
Brain & CNS	4	4.1	NC	NC	NC	1	2.3	NC	NC	NC	3	1.8	NC	NC	NC
Kidney & Renal Pelvis	5	6.4	78	25	183	4	4.1	NC	NC	NC	1	2.2	NC	NC	NC
Leukemia	5	5.4	92	30	214	3	3.1	NC	NC	NC	2	2.3	NC	NC	NC
Liver	1	1.8	NC	NC	NC	0	1.4	NC	NC	NC	1	0.4	NC	NC	NC
Lung & Bronchus	23	34.0	68	43	101	7	20.0	36*	14	74	16	14.5	110	63	179

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
CNS = Central Nervous System

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

TABLE 3a
Cancer Incidence
CT 3631.02, Acton, MA
1982-2000

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	13	8.2	159	85	272	8	5.3	152	65	299	5	2.9	173	56	404
Brain & CNS	6	4.8	124	45	270	3	2.3	NC	NC	NC	3	2.5	NC	NC	NC
Kidney & Renal Pelvis	6	5.5	110	40	239	5	3.1	163	53	381	1	2.4	NC	NC	NC
Leukemia	3	5.3	NC	NC	NC	3	2.6	NC	NC	NC	0	2.7	NC	NC	NC
Liver	2	1.3	NC	NC	NC	2	0.9	NC	NC	NC	0	0.4	NC	NC	NC
Lung & Bronchus	19	31.1	61*	37	96	9	16.4	55	25	104	10	14.6	68	33	126

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
CNS = Central Nervous System

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

TABLE 3b
Cancer Incidence
CT 3631.02, Acton, MA
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	2.5	NC	NC -- NC	2	1.6	NC	NC -- NC	2	0.9	NC	NC -- NC
Brain & CNS	3	1.3	NC	NC -- NC	2	0.6	NC	NC -- NC	1	0.7	NC	NC -- NC
Kidney & Renal Pelvis	2	1.1	NC	NC -- NC	2	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Leukemia	1	1.3	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.7	NC	NC -- NC
Liver	1	0.2	NC	NC -- NC	1	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	2	7.4	NC	NC -- NC	1	4.4	NC	NC -- NC	1	3.0	NC	NC -- NC

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

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

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

TABLE 3c
Cancer Incidence
CT 3631.02, Acton, MA
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	2.4	NC	NC -- NC	3	1.5	NC	NC -- NC	1	0.9	NC	NC -- NC
Brain & CNS	2	1.6	NC	NC -- NC	0	0.8	NC	NC -- NC	2	0.9	NC	NC -- NC
Kidney & Renal Pelvis	0	1.7	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.7	NC	NC -- NC
Leukemia	0	1.5	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.7	NC	NC -- NC
Liver	0	0.3	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	12	9.3	129	67 -- 226	6	5.0	121	44 -- 264	6	4.3	139	51 -- 302

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
CNS = Central Nervous System

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

TABLE 3d
Cancer Incidence
CT 3631.02, Acton, MA
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	5	3.5	142	46 -- 332	3	2.4	NC	NC -- NC	2	1.1	NC	NC -- NC
Brain & CNS	1	2.0	NC	NC -- NC	1	1.0	NC	NC -- NC	0	0.9	NC	NC -- NC
Kidney & Renal Pelvis	4	2.9	NC	NC -- NC	3	1.7	NC	NC -- NC	1	1.2	NC	NC -- NC
Leukemia	2	2.7	NC	NC -- NC	2	1.4	NC	NC -- NC	0	1.3	NC	NC -- NC
Liver	1	0.8	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung & Bronchus	5	15.1	33*	11 -- 77	2	7.8	NC	NC -- NC	3	7.3	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
CNS = Central Nervous System

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

TABLE 4a
Cancer Incidence
CT 3632.01, Acton, MA
1982-2000

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	7	7.4	94	38	194	4	5.3	NC	NC	NC	3	2.1	NC	NC	NC
Brain & CNS	6	4.1	147	54	320	3	2.1	NC	NC	NC	3	2.0	NC	NC	NC
Kidney & Renal Pelvis	5	5.1	98	32	230	3	3.1	NC	NC	NC	2	2.0	NC	NC	NC
Leukemia	5	4.4	114	37	265	4	2.4	NC	NC	NC	1	2.0	NC	NC	NC
Liver	1	1.2	NC	NC	NC	1	0.9	NC	NC	NC	0	0.3	NC	NC	NC
Lung & Bronchus	13	30.2	43*	23	74	6	17.4	34*	13	75	7	12.8	55	22	113

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
CNS = Central Nervous System

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

TABLE 4b
Cancer Incidence
CT 3632.01, Acton, MA
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	1	2.1	NC	NC -- NC	1	1.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Brain & CNS	3	1.1	NC	NC -- NC	1	0.6	NC	NC -- NC	2	0.6	NC	NC -- NC
Kidney & Renal Pelvis	1	1.0	NC	NC -- NC	0	0.6	NC	NC -- NC	1	0.4	NC	NC -- NC
Leukemia	0	1.1	NC	NC -- NC	0	0.6	NC	NC -- NC	0	0.5	NC	NC -- NC
Liver	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	3	7.3	NC	NC -- NC	2	4.7	NC	NC -- NC	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
CNS = Central Nervous System

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

TABLE 4c
Cancer Incidence
CT 3632.01, Acton, MA
1988-1993

Cancer Type	Total					Males					Females				
	Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI		Obs	Exp	SIR	95% CI	
Bladder	5	2.3	216	70	505	2	1.6	NC	NC --	NC	3	0.7	NC	NC --	NC
Brain & CNS	1	1.4	NC	NC	NC	1	0.7	NC	NC --	NC	0	0.7	NC	NC --	NC
Kidney & Renal Pelvis	2	1.6	NC	NC --	NC	2	1.0	NC	NC --	NC	0	0.6	NC	NC --	NC
Leukemia	3	1.2	NC	NC --	NC	3	0.7	NC	NC --	NC	0	0.5	NC	NC --	NC
Liver	0	0.3	NC	NC --	NC	0	0.2	NC	NC --	NC	0	0.1	NC	NC --	NC
Lung & Bronchus	4	9.4	NC	NC --	NC	1	5.5	NC	NC --	NC	3	3.9	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
CNS = Central Nervous System

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

TABLE 4d
Cancer Incidence
CT 3632.01, Acton, MA
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	1	2.7	NC	NC -- NC	1	2.0	NC	NC -- NC	0	0.8	NC	NC -- NC
Brain & CNS	2	1.5	NC	NC -- NC	1	0.8	NC	NC -- NC	1	0.7	NC	NC -- NC
Kidney & Renal Pelvis	2	2.3	NC	NC -- NC	1	1.4	NC	NC -- NC	1	0.9	NC	NC -- NC
Leukemia	2	2.0	NC	NC -- NC	1	1.1	NC	NC -- NC	1	0.9	NC	NC -- NC
Liver	1	0.7	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung & Bronchus	6	12.6	47	17 -- 103	3	6.8	NC	NC -- NC	3	5.8	NC	NC -- NC

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

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

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

TABLE 5a
Cancer Incidence
CT 3632.02, Acton, MA
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	7.9	76	28 -- 165	3	6.0	NC	NC -- NC	3	2.0	NC	NC -- NC
Brain & CNS	4	4.6	NC	NC -- NC	3	2.4	NC	NC -- NC	1	2.1	NC	NC -- NC
Kidney & Renal Pelvis	1	5.6	NC	NC -- NC	1	3.6	NC	NC -- NC	0	2.0	NC	NC -- NC
Leukemia	6	4.8	126	46 -- 273	3	2.8	NC	NC -- NC	3	2.0	NC	NC -- NC
Liver	1	1.3	NC	NC -- NC	1	1.0	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	23	32.8	70	44 -- 105	10	19.8	50*	24 -- 93	13	12.9	100	53 -- 172

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
CNS = Central Nervous System

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

TABLE 5b
Cancer Incidence
CT 3632.02, Acton, MA
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	2.3	NC	NC -- NC	1	1.8	NC	NC -- NC	1	0.6	NC	NC -- NC
Brain & CNS	1	1.4	NC	NC -- NC	0	0.7	NC	NC -- NC	1	0.7	NC	NC -- NC
Kidney & Renal Pelvis	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
Leukemia	1	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.5	NC	NC -- NC
Liver	0	0.2	NC	NC -- NC	0	0.2	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	8	8.4	95	41 -- 187	3	5.4	NC	NC -- NC	5	3.0	165	53 -- 384

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
CNS = Central Nervous System

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

TABLE 5c
Cancer Incidence
CT 3632.02, Acton, MA
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	3	2.5	NC	NC -- NC	1	1.8	NC	NC -- NC	2	0.6	NC	NC -- NC
Brain & CNS	3	1.6	NC	NC -- NC	3	0.8	NC	NC -- NC	0	0.8	NC	NC -- NC
Kidney & Renal Pelvis	0	1.8	NC	NC -- NC	0	1.2	NC	NC -- NC	0	0.7	NC	NC -- NC
Leukemia	0	1.4	NC	NC -- NC	0	0.8	NC	NC -- NC	0	0.6	NC	NC -- NC
Liver	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	7	10.3	68	27 -- 140	2	6.3	NC	NC -- NC	5	4.0	125	40 -- 292

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
CNS = Central Nervous System

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

TABLE 5d
Cancer Incidence
CT 3632.02, Acton, MA
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	1	2.9	NC	NC -- NC	1	2.3	NC	NC -- NC	0	0.7	NC	NC -- NC
Brain & CNS	0	1.6	NC	NC -- NC	0	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC
Kidney & Renal Pelvis	1	2.4	NC	NC -- NC	1	1.6	NC	NC -- NC	0	0.9	NC	NC -- NC
Leukemia	5	2.1	235	76 -- 549	2	1.2	NC	NC -- NC	3	0.9	NC	NC -- NC
Liver	1	0.7	NC	NC -- NC	1	0.6	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	8	13.2	61	26 -- 119	5	7.7	65	21 -- 152	3	5.5	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
CNS = Central Nervous System

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

TABLE 6a
Cancer Incidence
CT 3612, Concord, MA
1982-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	8	14.4	55	24 -- 109	5	11.3	44	14 -- 104	3	3.2	NC	NC -- NC
Brain & CNS	8	7.2	111	48 -- 219	4	4.0	NC	NC -- NC	4	3.2	NC	NC -- NC
Kidney & Renal Pelvis	10	9.5	105	50 -- 194	6	6.3	95	35 -- 206	4	3.1	NC	NC -- NC
Leukemia	9	7.8	116	53 -- 220	4	4.8	NC	NC -- NC	5	3.0	166	54 -- 388
Liver	1	2.3	NC	NC -- NC	1	1.8	NC	NC -- NC	0	0.5	NC	NC -- NC
Lung & Bronchus	43	57.2	75	54 -- 101	28	36.8	76	51 -- 110	15	20.4	73	41 -- 121

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
CNS = Central Nervous System

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

TABLE 6b
Cancer Incidence
CT 3612, Concord, MA
1982-1987

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	4.1	NC	NC -- NC	2	3.1	NC	NC -- NC	0	1.0	NC	NC -- NC
Brain & CNS	4	2.1	NC	NC -- NC	1	1.1	NC	NC -- NC	3	1.0	NC	NC -- NC
Kidney & Renal Pelvis	4	2.0	NC	NC -- NC	2	1.3	NC	NC -- NC	2	0.7	NC	NC -- NC
Leukemia	1	2.0	NC	NC -- NC	1	1.2	NC	NC -- NC	0	0.8	NC	NC -- NC
Liver	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	13	14.4	91	48 -- 155	5	9.5	53	17 -- 123	8	4.9	165	71 -- 325

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
CNS = Central Nervous System

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

TABLE 6c
Cancer Incidence
CT 3612, Concord, MA
1988-1993

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	4.4	NC	NC -- NC	2	3.5	NC	NC -- NC	2	1.0	NC	NC -- NC
Brain & CNS	3	2.4	NC	NC -- NC	2	1.3	NC	NC -- NC	1	1.1	NC	NC -- NC
Kidney & Renal Pelvis	2	3.0	NC	NC -- NC	1	2.1	NC	NC -- NC	1	1.0	NC	NC -- NC
Leukemia	6	2.2	278 *	102 -- 606	2	1.3	NC	NC -- NC	4	0.8	NC	NC -- NC
Liver	1	0.6	NC	NC -- NC	1	0.5	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung & Bronchus	13	17.6	74	39 -- 126	10	11.4	87	42 -- 161	3	6.2	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
CNS = Central Nervous System

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

TABLE 6d
Cancer Incidence
CT 3612, Concord, MA
1994-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	5.6	NC	NC -- NC	1	4.4	NC	NC -- NC	1	1.2	NC	NC -- NC
Brain & CNS	1	2.8	NC	NC -- NC	1	1.7	NC	NC -- NC	0	1.1	NC	NC -- NC
Kidney & Renal Pelvis	4	4.5	NC	NC -- NC	3	3.0	NC	NC -- NC	1	1.4	NC	NC -- NC
Leukemia	2	3.8	NC	NC -- NC	1	2.3	NC	NC -- NC	1	1.4	NC	NC -- NC
Liver	0	1.3	NC	NC -- NC	0	1.0	NC	NC -- NC	0	0.3	NC	NC -- NC
Lung & Bronchus	17	24.7	69	40 -- 110	13	15.0	86	46 -- 148	4	9.7	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
CNS = Central Nervous System

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

APPENDIX A

CODING DEFINITIONS OF CANCER SITE/TYPE

Appendix A

Cancer Site / Type	*ICD-O-1 and Other Pre-ICD-O-2 Codes*		*ICD-O-2 Codes*		*ICD-O-3 Codes*	
	Site code	***Histology code***	***Site code***	***Histology code***	***Site code***	***Histology code***
Bladder	188.0-188.9	except 9590-9980	C67.0-C67.9	except 9590-9989	C67.0-C67.9	except 9590-9989
Brain & Central Nervous System (CNS)	191.0-192.9	*See Table 1 below*	C70.0-C72.9	*See ICD-O codes in Table 1 below*	C70.0-C72.9	*See Table 2 below*
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

**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 (1995-2000 data).*

Table 1: Histology codes for Brain and Central Nervous System (pre-ICD-O-3)

ICD-O O 9370, 9380, 9381, 9382, 9390, 9391, 9392, 9400, 9401, 9403, 9410, 9411, 9420, 9421, 9422, 9423, 9424, 9430, 9440, 9441, 9442, 9443, 9450, 9451, 9460, 9470, 9471, 9472, 9473, 9480, 9481, 9490, 9500, 9501, 9502, 9503, 9530, 9539, 9540, 9560, 9561.

SNOP P 9363, 9383, 9393, 9403, 9413, 9423, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9533, 9543, 9563.

HLTHSTT B 9383, 9393, 9403, 9433, 9443, 9453, 9463, 9473, 9483, 9493, 9503, 9530, 9533, 9537, 9543, 9563.

Table 2: Histology codes for Brain and Central Nervous System (ICD-O-3)

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

APPENDIX B

RISK FACTOR INFORMATION FOR SELECTED CANCER TYPES

Bladder Cancer

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

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

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

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

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS, 2006a; Silverman et al., 1996). Exposure to chlorinated by-products in drinking water has also been suggested to increase bladder cancer risk. However, a recent population-based study found that an association was present only among smokers (Cantor et al., 1998).

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Brain and CNS Cancer

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

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

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

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

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

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

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

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

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

In addition, rare cases of brain and spinal cord cancer run in some families. Brain tumors in some persons are associated with genetic disorders such as neurofibromatosis types I and II, Li-Fraumeni syndrome, and tuberous sclerosis. Neurofibromatosis type I (von Recklinghausen's disease) is the most common inherited cause of brain or spinal cord tumors and occurs in about one out of every 3,000 people (Preston-Martin and Mack 1996). The disease may be associated with optic gliomas or other gliomas of the brain or spinal cord (ACS 2006b). Of those afflicted with the disease, about 5-10% will develop a central

nervous system tumor (Preston-Martin and Mack 1996). In addition, von Hippell-Lindau disease is associated with an inherited tendency to develop blood vessel tumors of the cerebellum (ACS 2006b). However, malignant (or cancerous) brain tumors are rare in these disorders; inherited syndromes that predispose individuals to brain tumors appear to be present in fewer than 5% of brain tumor patients (Preston-Martin and Mack 1996).

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Kidney Cancer

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

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

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

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

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al. 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary risk factors may be important in the development of kidney cancer (ACS 2006; McLaughlin et al. 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS 2006; Linehan et al. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS

2006). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al. 1997; McLaughlin et al. 1996).

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

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Liver Cancer

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

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

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

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

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that is the result of scar tissue formation on the liver, which can lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al. 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS 2006a). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS 2006a). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al. 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands 1999). Long-term exposure to aflatoxin can also cause

liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS 2006a; London and McGlynn 1996). These chemicals may also increase the risk of cholangiocarcinoma and HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS 2006a). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS 2006a; 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 2006a; London and McGlynn 1996). Long-term anabolic steroid use may slightly increase the risk of HCC (ACS 2006a). 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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Leukemia

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

In 2006, leukemia is expected to affect approximately 35,070 individuals in the United States (20,000 males and 16,730 females) in the United States, resulting in 22,280 deaths. Acute cases of leukemia are slightly more common that chronic, 15,860 and 14,520 respectively. In Massachusetts, approximately 770 individuals will be diagnosed with the disease in 2006, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML (approximately 11,700 cases) and CLL (approximately 9,560 cases). Incidences of ALL have increased approximately 1.8% per year since 1988 while incidences of CLL have decreased approximately 1.9% each year since 1988. Leukemia is the most common type of childhood cancer, accounting for about 30% of all cancers diagnosed in children. The majority (74%) of these cases are of the ALL type (ACS 2006a).

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

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2006c). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al. 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS 2006c). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and

hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection. However, no specific virus has been identified as related to ALL (Linet and Cartwright 1996). Reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care do not support this hypothesis (Greaves MF 1997; Kinlen and Balkwill 2001; Rosenbaum et al. 2000).

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

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

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 2006e). Twice as many men as women are affected by this type of leukemia (Deisseroth et al. 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS 1999; Weinstein and Tarbell 1997). It is thought that individuals with a family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright 1996). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al. 1997).

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

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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 2005). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

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

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

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS 2005). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (Samet and Eradze 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni 1996; Pohlabeln et al. 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS 2005; Blot and Fraumeni 1996). Industrial sand workers exposed to crystalline

silica are also at an increased risk for lung cancer (Rice et al. 2001; Steenland and Sanderson 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni 1996).

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

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

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

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

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CERTIFICATION

The Health Consultation, *Assessment of Cancer Incidence in Acton and Concord Census Tract 3612: 1982-2000, Acton, Middlesex County, Massachusetts* was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated. Editorial review was completed by the cooperative agreement partner.

-FOR-

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

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