Massachusetts
Department Of
Public Health

Health Consultation: Evaluation of Environmental Concerns at Ginn Field and Childhood Cancer Incidence from 2000-2008 in Winchester, Middlesex County, Massachusetts

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I. SUMMARY

Introduction: This health consultation was conducted because a resident of the community of Winchester, Massachusetts, and the Winchester Board of Health was concerned about suspected increases of childhood cancer and potential health risks related to surface soil at Ginn Field (as well as surface water and sediment contamination of the section of the Aberjona River that abuts the field).

Overview: The MDPH has reached several important conclusions about the incidence of childhood cancer in Winchester and potential environmental exposures at Ginn Field.

Conclusion 1: The MDPH concluded that accidentally eating or touching soil at Ginn Field is not expected to harm people's health.

Basis for Decision: Past activities at the Industri-Plex and Wells G & H Superfund sites in Woburn, MA resulted in chemical contaminants in the surface water and sediment of the Aberjona River. Based on soil sampling conducted directly downstream of Ginn Field, surface soil on or near the field is likely impacted by contamination deposited by flood waters of the abutting Aberjona River. As a result, adults, adolescents, and younger children could come into contact with chemical contaminants while playing sports at or visiting the field either in the past or presently. Based on available information and conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into a child's or an adult's body are below levels that would harm their health.

Because the maximum concentration of lead in surface soil was above health-based comparison values and children may have been exposed while playing at Ginn Field, the MDPH evaluated data on blood lead levels among children who resided in Winchester between July 1999 and June 2010. No unusual concentration of children with elevated blood lead levels was noted in areas proximate to the field. In addition, the MDPH used the USEPA Integrated Exposure Uptake Biokinetic (IEUBK) model to predict blood lead levels based on lead intake via various sources. Using conservative assumptions, this model predicted that about 0.6% of a hypothetical population of children under the age of 7 years who trespass/play on the site would have blood lead levels greater than 10 µg/dl, which the Centers for Disease Control and Prevention (CDC) define as a level of concern. The prediction of less than a 1% risk of blood lead levels above 10 µg/dl is below the USEPA Office of Solid Waste and Emergency Response's specified level of protectiveness of no more than a 5% risk of an elevated blood lead level for a given scenario. Based upon

this information, children who played or visited Ginn Field are unlikely to have experienced elevated blood lead levels as a result of potential exposure to lead in surface soil at the site.

In the future, deposition of contaminants from flood waters onto the surface soil at Ginn Field is expected to be minimal after the channel in this section of the Aberjona River is deepened as part of a series of flood mitigation projects that are currently underway. Therefore, exposure of adults, adolescents or children to contaminants in surface soil deposited by flood waters was eliminated as an exposure pathway in the future.

Conclusion 2: The MDPH concluded that accidentally eating or touching sediment while playing or wading in the Aberjona River near Ginn Field is not expected to harm people's health.

Basis for Decision: Based on available sampling data, sediment in the section of the Aberjona River directly adjacent to Ginn Field contains chemical contaminants. Adults, adolescents, and younger children could come into contact with contaminants in the sediment while wading or playing in the section of the Aberjona River that abuts Ginn Field in the past or present. Based on conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into a child's or an adult's body are below levels that would harm their health. Likewise, future exposures to constituents in sediment are unlikely to cause health concerns given that there are no known ongoing sources and, hence, similar or lower concentrations are expected in the future.

Conclusion 3: The MDPH concluded that accidentally drinking or touching surface water while playing or wading in the Aberjona River near Ginn Field is not expected to harm people's health.

Basis for Decision: Sampling conducted directly upstream of Ginn Field demonstrates that surface water in the stretch of the Aberjona River adjacent to the field contains chemical contaminants. Adults, adolescents, and younger children could come into contact with contaminants while wading or playing in the section of the Aberjona River that abuts Ginn Field in the past, present and future. Based on available information and conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into a child's or an adult's body during either base flow or storm flow conditions are below levels that would harm their health.

Conclusion 4: The MDPH concluded that tap water in the community of Winchester is not impacted by contaminants related to the Aberjona River and, therefore, is not expected to harm people's health.

Basis for Decision: Groundwater in the community of Winchester is not used as a source of drinking water. The drinking water for the community of Winchester is supplied by local reservoirs and the Massachusetts Water Resources Authority. Therefore, exposure of individuals to contaminants in groundwater was eliminated as a pathway.

Conclusion 5: The MDPH concluded that accidentally touching pesticides that were sprayed in the past at Ginn Field is not expected to harm people's health. No pesticides have been applied to Ginn Field since 2005.

Basis for Decision: Based on the restricted frequency of use as well as the toxicological characteristics and carcinogenicity of the active ingredients of the pesticides used at Ginn Field, post-application exposure of children or adults is not expected to harm their health.

Due to the possible association between 2,4-D and non-Hodgkin's lymphoma (NHL), the MDPH reviewed the incidence of NHL among adults and children in the community of Winchester during the time period 2000-2005. During this six-year time period, the number of observed diagnoses was slightly greater than expected but not statistically significant. No unusual spatial or temporal trends were observed.

Conclusion 6: The MDPH concluded that the incidence of childhood cancer was slightly elevated in the community of Winchester during the 6-year time period 2000-2005 but was not statistically significant. The incidence among females was slightly less than expected, whereas, among males, it was more than expected but the difference was not statistically significant. A qualitative review of childhood cancer in Winchester for 2006-2008 was also conducted. No unusual trends emerged when the overall age, gender and histology patterns were examined in more detail for the time period 2000-2008.

Basis for Decision: To determine whether the incidence of childhood cancer in Winchester was elevated, the observed number of cancer diagnoses in the community was compared to the number that would be expected based on the statewide cancer rate. Between 2000 and 2005, 7 diagnoses were reported when approximately 6 would be expected. This difference was not statistically significant. The incidence among females was slightly less than expected (1 observed compared to about 3 expected), whereas, among males, it was more than expected (6 observed compared to about 3 expected) but not statistically significant. During this time period, the following four types of cancer were diagnosed among children in Winchester: CNS tumors, leukemia, neuroblastoma, and malignant gonadal germ cell tumors.

A qualitative review of childhood cancer in Winchester for more recent years was also conducted. Between 2006 and 2008, six other children in Winchester were diagnosed with the following five types of cancer: leukemia, malignant gonadal germ cell tumors, cancer of the bone, soft tissue sarcoma, and an unspecified carcinoma.

Overall, the distribution of cancer types diagnosed among children in Winchester during 2000-2008 was generally consistent with state and national trends. Furthermore, analysis of the geographic distribution of place of residence for children diagnosed with cancer in Winchester during 2000-2008 did not reveal any atypical spatial patterns. Although some diagnoses during 2006-2008 occurred among children whose residences at the time of diagnosis were in relative close proximity to one another, the overall geographic distribution was generally consistent with population density.

Next Steps:

❖ The MDPH recommends no further investigation of childhood cancer incidence in Winchester at this time.

For More Information:

If you have concerns about your health, you should contact your health care provider. You may also call the MDPH at 617-624-5757 and ask for information on the Ginn Field site.

For more information about pesticides, contact the National Pesticide Information Center at 1-800-858-7378 or the Pesticide Program at the Massachusetts Department of Agricultural Resources at (617) 626-1776.

II. INTRODUCTION

At the request of a concerned resident and the Winchester Board of Health (BOH), the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), reviewed the incidence of childhood cancer (i.e., ages 0-19) for the community of Winchester and conducted an evaluation of potential environmental exposures related to Ginn Field[1]. Concerns focused on possible health risks from exposure to contaminants in the surface water and sediment of the Aberjona River, which abuts the athletic field and floods periodically.

This Health Consultation (HC) provides both quantitative and qualitative evaluations of childhood cancer (all types) for all children living in Winchester who were diagnosed between 2000–2008 to determine if childhood cancer may be occurring in an unusual pattern in the community. This investigation also provides a review of potential pathways of exposure to contaminants detected in surface soil near Ginn Field as well as surface water and sediment in the stretch of the Aberjona River adjacent to the field. Additionally, a review of pesticides used at Ginn Field was conducted.

III. BACKGROUND

The community of Winchester is located 8 miles northwest of Boston in Middlesex County. Ginn Field, which is managed by the Winchester Department of Public Works (DPW), encompasses 5 acres and is located on Bacon Street, off of Mystic Valley Parkway, in southeastern Winchester (Figure 1). To the east, Ginn Field directly abuts the Aberjona River, which is a small stream approximately 15 to 20 feet wide in this stretch, and a narrow strip of woods. Farther east, residential properties are located on Mystic Valley Parkway. Across Bacon Street to the south is the Wedgemere MBTA station and wooded wetlands. Farther south is the inlet to Upper Mystic Lake. The western edge of Ginn Field abuts the MBTA commuter rail line. Farther west, residential

[1] This report was supported in part by funds from a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Health and Human Services. This document has not been reviewed and cleared by ATSDR.

properties are located on Ginn Road. To the north, the field is bordered by a parking area and the Aberjona River with residential properties nearby.

Two sites that are listed on the National Priorities List for Uncontrolled Hazardous Waste Sites (also referred to as Superfund sites) by the U.S. Environmental Protection Agency (USEPA) are located north of Ginn Field in the community of Woburn and are hydraulically connected by the Aberjona River. The Industri-Plex Superfund site encompasses 245 acres and is located approximately one mile north of the Wells G & H Superfund site, which itself encompasses 330 acres and is located about 3.5 miles north of Ginn Field (Figure 2). Wells G & H were two municipal wells developed in 1964 and 1967 to supplement the water supply for the City of Woburn. Both wells were shut down in 1979 after contamination was detected. Five separate properties were found to be contributing sources of contamination to the aquifer that supplied water to these two wells. Groundwater was contaminated with volatile organic compounds (VOCs) and soils were contaminated with polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), VOCs and pesticides. The Industri-Plex Superfund site was a former chemical and glue manufacturing facility for over 100 years until it was developed for industrial use during the 1970s. The manufacturing by-products and wastes contaminated the groundwater with VOCs as well as ammonia and metals such as arsenic. Soils were contaminated with heavy metals, including arsenic, chromium, and lead. The Aberjona River flows through both of these Superfund sites and continues south approximately six river miles before discharging to the Mystic Lakes in Winchester (Tetra Tech 2005; USEPA 2005a, 2009a, 2009b). Just before discharging to Upper Mystic Lake, it passes Ginn Field, which is located at an elevation lower than that of the surrounding streets, houses and rail line, and, as a result, is subject to periodic flooding. At such times, surface water and sediment of the Aberjona River may be transported by flood waters and deposited on the surface soil at Ginn Field.

In 2002, the USEPA combined the separate surface water and sediment investigations that were being performed at the two Superfund sites into one study called the Multiple Source Groundwater Response Plan (MSGRP) Remedial Investigation to more efficiently evaluate contamination and assess potential risk for the entire Aberjona River. As part of

the MSGRP Remedial Investigation, a baseline risk assessment was conducted. The findings were used to formulate a comprehensive strategy to address human health and ecological risks. Remediation activities at the Superfund sites, which are still on-going, have included removing contaminated soils, placing a protective cap over more than 100 acres of contaminated soils, dredging and disposing of contaminated sediment, and treating groundwater from a contaminated aquifer. While remediation efforts have been proceeding, significant portions of the sites have been redeveloped. The sites are currently occupied by numerous retail, commercial, and industrial businesses as well as the Anderson Regional Transportation Center, a 33-acre commuter transportation hub, which was constructed in the late 1990s and opened in May 2001 (Tetra Tech 2005; USEPA 2009a,b).

IV. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS AND HEALTH CONCERNS

An evaluation of potential pathways of exposure was conducted to determine whether contaminants in the stretch of the Aberjona River adjacent to Ginn Field or pesticides applied to the field could impact children playing at the field in the past, present, or future. In general, five conditions must be present for exposure to occur. First, there must be a source of the chemical. Second, an environmental medium must be contaminated by either the source or by chemicals transported away from the source. Third, there must be a location where a person can potentially contact the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, or dermal absorption. Finally, a population of individuals that could potentially be exposed must be present (ATSDR 2005). A completed exposure pathway exists when all five elements are present and indicates that exposure to humans occurred in the past, is occurring in the present, or will occur in the future. A potential exposure pathway exists when one or more of the five elements is either missing or uncertain and indicates that exposure to a contaminant could have occurred in the past, could be occurring in the present, or could occur in the future. An exposure pathway can be eliminated if at least one of the five elements is missing and will not likely be present in the future.

In order to evaluate concerns about potential environmental exposures to contaminants from the section of the Aberjona River that abuts Ginn Field and contaminants possibly deposited by flood waters, the MDPH contacted the USEPA to obtain and review comprehensive and up-to-date environmental information related to the Wells G & H and Industri-Plex Superfund sites. For the purposes of the MSGRP Remedial Investigation, the USEPA divided a six-mile stretch of the Aberjona River into seven reaches that extend from the town line between Wilmington and Woburn to the Upper and Lower Mystic Lakes in Arlington. The section of the Aberjona River that borders Ginn Field in Winchester is located within Reach 5, which extends from the Mill Pond outlet south to the Upper Mystic Lake inlet (Figure 3). Sampling data available for this reach include soil, sediment, and surface water samples (Tetra Tech 2005). Additional sediment data specific to the section of the Aberjona River immediately adjacent to Ginn Field were available from sampling efforts related to a series of flood mitigation projects that are currently underway (AECOM 2010).

The USEPA concluded in the MSGRP Remedial Investigation that heavy metals are the principal contaminants of concern in surface water and sediment throughout the entire study area, with arsenic representing the most significant metal present at elevated concentrations throughout the system. The principal source of contamination is the soils underlying the Industri-Plex Superfund site that are impacting groundwater, which in turn discharges to surface water. Organic compounds originating from the Industri-Plex site were shown to attenuate within the first reach of the Aberjona River whereas metals were shown to migrate farther downstream through dissolved phases and sediment transport mechanisms during both base flow and storm flow conditions. It was determined that the impacted media in Reach 5 consist of sediment and surface water with metals as the contaminants of concern. Specifically, arsenic, iron and lead were the surface soil and sediment contaminants determined to exceed USEPA Region 9 Preliminary Remediation Goals, which were used by the USEPA as comparison values (Tetra Tech 2005). Based on this information, the MDPH conducted its evaluation with a focus on these three metal contaminants in this HC.

The maximum concentrations of contaminants detected in the various types of environmental media were identified and compared to health-based comparison values (ATSDR 2005, 2010a, 2010b; USEPA 2010; MDEP 2010). Comparison values are developed based on health guidelines and assumed situations that represent conservative estimates of human exposure. Contaminant concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, contaminant concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a compound to impact one's health, it must not only be present in the environmental media, but one must also come in contact with it. Therefore, if a contaminant concentration is greater than the comparison value, the potential for exposure should be further evaluated (ATSDR 2005). Concentration levels that are considered typical or "background" were also used in the analysis (USGS 1984; MDEP 2002). In addition, essential nutrients for which comparison values were not available were not retained for further analysis (ATSDR 2005, USEPA 1989).

In order to evaluate concerns about potential exposure to pesticides applied on Ginn Field, the MDPH contacted the Winchester DPW to obtain available information regarding current and historical applications. The chemical ingredients of each pesticide were identified from their Material Safety Data Sheets (MSDS). Information about the toxicity and carcinogenicity of each active ingredient was obtained from a variety of sources, including the International Agency for Research on Cancer (IARC) and the USEPA Office of Pesticide Programs.

A. Exposure to Soil

In Reach 5, surface soil samples (0 to 6 inches) were collected by the USEPA in 2004 from nine locations along the bank of a ponded area of the Aberjona River south of Bacon Street, downstream of Ginn Field. The purpose of collecting these samples was to investigate potential metals contamination deposited by flood waters. Levels of arsenic and lead in at least one sample exceeded both soil comparison values and typical background values (Tetra Tech 2005; ATSDR 2010a; USEPA 2010). The maximum concentration of iron (34,000 ppm) detected did not exceed the applicable soil

comparison value. Although the maximum concentration of chromium (90 ppm) detected exceeded the soil comparison value for children, it is within the range of typical background values and, therefore, further analysis was not conducted. A summary of the maximum and average concentration of contaminants detected in surface soil samples that exceeded comparison values is presented in Table 1.

Because Ginn Field is located in a low-lying area subject to flooding, contaminants in the surface water and sediment of the Aberjona River may periodically be transported by flood waters and deposited on the surface soil at Ginn Field. As a result, it is possible that children participating in sports, adult spectators, and others visiting Ginn Field may have experienced exposure opportunities to arsenic and lead via incidental ingestion of or dermal contact with contaminated floodplain soils at Ginn Field either in the past or presently. In order to evaluate the potential for carcinogenic health effects, exposure doses were estimated and compared to health guideline values for cancer. It should be noted that exposure to subsurface soil is not expected due to its depth below ground surface and, therefore, was not evaluated.

The USEPA and the IARC have classified inorganic arsenic as a known carcinogen based on sufficient evidence in humans. Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. The degree of risk depends on the intensity and frequency of exposure. It is of note that the majority of the data on the effects of exposure of humans to arsenic has focused on adults, with few studies specific to children. Also, data linking inorganic arsenic to cancer or noncancer health effects are primarily from studies evaluating daily exposure to inorganic arsenic in drinking water over many years. Furthermore, dermal uptake of inorganic arsenic is considered to be sufficiently limited such that other routes of exposure, such as ingestion, are almost always expected to be of greater concern (ATSDR 2007a).

Under the conservative assumption that adults (ages 18 and above), adolescents (ages 12 through 17) and younger children (under age 11) incidentally ingested surface soil with the maximum concentration of arsenic that was detected in Reach 5 (98 mg/kg) for 5 days/week for 22 weeks per year during the warmer months of the year (from May

through September) for either 11 years for the child, 17 years for the adolescent or 30 years for the adult, the estimated exposure would not result in an unusual cancer risk. Also, the estimated exposure level would be below the ATSDR Minimal Risk Level (MRL). The MRL is an estimate of daily exposure to a contaminant below which adverse noncancer health outcomes are unlikely to occur. Since the estimated exposure doses for adults, adolescents and younger children are below the MRL, noncancer health effects would not be expected. Under the same exposure conditions and assumptions described above, dermal exposure to the maximum concentration of arsenic detected in surface soil samples is also not expected to result in an unusual cancer risk or adverse noncancer health effects in adults, adolescents or younger children. See Appendices A and B for more information on the exposure dose and cancer risk calculations. It should be noted that the analysis provided here examines metals deposited by flood waters as represented by the ponded area located south of Bacon Street, downstream of Ginn Field.

In humans, the main target for lead toxicity is the nervous system. Lead exposure is of most concern for children six years of age or younger because they absorb more lead than older children or adults, have more hand to mouth activity that results in greater incidental ingestion of soil, and are more susceptible to health effects from lead exposure. In order to evaluate potential health concerns related to exposure opportunities to lead in surface soil at Ginn Field, the MDPH used the USEPA Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children (USEPA 2007). This model is widely used throughout the country to predict blood lead levels based on lead intake via various sources (e.g., soil, food, water). Environmental data specific to a given scenario are input into the model in order to predict blood lead levels for young children (aged 6 months to 7 years). The model generally uses typical or average concentrations in the various source media, assumes daily exposures, and predicts blood lead concentrations based on chronic exposures (e.g., 1 year or more).

To be conservative, the MDPH used the IEUBK model with the assumptions that young children visited the site 5 days each week for 22 weeks of the year for 7 years, that the average lead concentration detected in surface soil (298 mg/kg) within Reach 5 reflects the range of contaminant concentrations that would likely have been ingested over time,

and that half of a child's typical daily incidental soil ingestion occurred during the time spent on the site. Using these assumptions, the predicted mean blood lead concentration was 3.1 micrograms per deciliter (μg/dL). The IEUBK model predicted that about 0.6% of this hypothetical population of children under the age of 7 years who trespass/play on the site would have blood lead levels greater than 10 μg/dL, which the Centers for Disease Control and Prevention (CDC) define as a level of concern (ATSDR 2007b). The prediction of less than a 1% risk of blood lead levels above 10 μg/dL is below the USEPA Office of Solid Waste and Emergency Response's specified level of protectiveness of no more than a 5% risk of an elevated blood lead level for a given scenario (USEPA 2007). Thus, it appears unlikely that young children trespassing/playing at Ginn Field would have blood lead levels above the current CDC level of concern given exposure opportunities at the site. In addition, it should be noted that the average concentration of lead detected in surface soil is within the range of typical background values.

To further address concerns of past exposure to lead in surface soil at Ginn Field, the MDPH evaluated readily available data on blood lead levels among children living in Winchester. Data were obtained from the MDPH BEH Childhood Lead Poisoning Prevention Program (CLPPP). CLPPP was established for the prevention, screening, diagnosis, and treatment of lead poisoning in children residing in Massachusetts. The Massachusetts Lead Law requires that all children be tested for blood lead levels once between the ages of 9 months and 12 months, and again at the ages of 2 and 3 years (CLPPP 2010a).

Blood lead level testing data were obtained for the community of Winchester from July 1999 though June 2010, the time period for which the most recent data were available from the CLPPP at the initiation of this analysis[2]. During this time period, there were 18 children living in Winchester with blood lead levels equal to or greater than 10 μg/dL. None of these children lived adjacent to Ginn Field and only one lived within a quarter-mile radius of the field. Therefore, there was no geographic pattern of higher blood lead

[2] The data summarized in this report are drawn from data entered into the CLPPP before December 21, 2010.

levels closer to Ginn Field that would suggest that exposure to lead from the site could have occurred resulting in adverse health effects.

The most important source of elevated blood lead levels in Massachusetts children is lead paint in older homes. Many homes built before 1978 have lead paint on the interior and exterior of the building (CLPPP 2010a). Of the 18 children living within Winchester that had an elevated blood lead level during the time period of analysis, 17 lived in a house that was built before 1978. In addition, records indicate that lead violations were found and/or de-leading was conducted at six of the 18 houses (CLPPP 2010b). Therefore, exposure to residential lead paint likely contributed to the children's elevated blood lead levels.

With regard to possible future exposures to contaminants in surface soil at Ginn Field, it is important to note that the Town of Winchester is currently implementing a series of flood mitigation projects. These projects aim to reduce the frequency and severity of flooding along the Aberjona River by increasing its flood storage capacity and removing constrictions to flow. Specifically, one of these projects involves re-grading and deepening the channel between Manchester Road and Bacon Street, which includes the section of the Aberjona River that abuts Ginn Field (AECOM 2010). This project is currently underway and is expected to be completed by mid-winter (Marra 2011). As a result of this effort, it is expected that future deposition of contaminants from flood waters onto surface soil at Ginn Field would be minimal, and hence, future exposure opportunities to such contaminants are not expected.

B. Exposure to Sediment

Sediment samples were collected from the Aberjona River at four locations directly adjacent to Ginn Field in 2008 for the town of Winchester as part of a proposal for a flood mitigation program. Samples were analyzed for metals, VOCs, PAHs, PCBs and total petroleum hydrocarbons (TPH) (AECOM 2010). Because ATSDR comparison values do not exist for sediment, soil comparison values were used for screening purposes. Levels of the PAH benzo(a)pyrene in at least one sample exceeded both soil comparison values and typical background values (AECOM 2010; ATSDR 2010a;

USEPA 2010). Other PAHs were detected within background levels. Although the maximum concentration of arsenic detected (32.7 ppm) exceeds soil comparison values, it is within the range of typical background levels and, therefore, further analysis was not conducted. The maximum concentration of lead detected (191 ppm) does not exceed the applicable soil comparison value. Sediment samples were not tested for iron. A summary of the maximum and average concentrations of contaminants detected in sediment samples that exceed comparison values is presented in Table 2.

During a site visit by CAP staff in January 2011, it appeared that access from Ginn Field to the abutting section of the Aberjona River was possible despite the presence of a chain link fence. A large break in the fence was noted between the front and back baseball fields in what is presumed to be a heavily trafficked area during the warmer months of the year. It is reasonable to assume that older children, including elementary aged children and adolescents, may play or wade in this section of the Aberjona River while baseball, soccer and other athletic games are being played at Ginn Field. In addition, access is also possible from a paved pathway along Mystic Valley Parkway on the opposite side of the river. Therefore, it is possible that older children could have been exposed to benzo(a)pyrene via incidental ingestion of or dermal contact with sediment while playing or wading in the Aberjona River near Ginn Field in the past or present.

Under the highly conservative assumption that adults, adolescents and younger children incidentally ingested sediment with the maximum concentration of benzo(a)pyrene that was detected (3.5 mg/kg) for 5 days/week for 22 weeks per year during the warmer months of the year for either 11 years for the child, 17 years for the adolescent or 30 years for the adult, the estimated exposure would not result in an unusual cancer risk. Adverse noncancer health effects are also unlikely to occur. Likewise, dermal exposure to the maximum concentration of benzo(a)pyrene detected in sediment is also not expected to result in an unusual cancer risk or adverse noncancer health effects. See Appendices C and D for more information on the exposure dose and cancer risk calculations.

Dredging is planned as part of the effort to deepen the channel in this section of the river to mitigate future flooding. Because PAHs are the products of incomplete combustion

and are ubiquitous in the environment, there is no reason to believe that the concentration of benzo(a)pyrene in sediment would significantly change after dredging occurs (ATSDR 1995a). Hence, health effects are not expected to result from future exposure opportunities.

C. Exposure to Surface Water

Several investigations were conducted between 1995 and 2002 to evaluate the presence and transport of dissolved and suspended contaminants in surface water throughout the MSGRP Remedial Investigation study area. In Reach 5, surface water samples were collected in 2001 and 2002 during base flow and storm flow conditions at a USGS gauging station (Station #01102500) located immediately upstream of Ginn Field and analyzed for total suspended solids and metals (Figure 3). An additional sample was collected at this station in 1995 and analyzed for metals, VOCs and several additional water quality parameters (Tetra Tech 2005). It should be noted that streamflow measured at this station during the investigation period was consistent with the 63-year averages recorded at this site, with the exception of the timing of the spring snowmelt peak.

Due to the lack of health-based comparison values for surface water, results were compared to Massachusetts standards for public drinking water supplies (MDEP 2010). Water from the Aberjona River is not used as a drinking water source; and thus, this is a highly conservative approach because exposures to contaminants in surface water that is not used for drinking water are expected to be much less than exposures to those in drinking water consumed every day. If Massachusetts drinking water standards were not available, results were compared to ATSDR drinking water comparison values (ATSDR 2010b). In the absence of both Massachusetts drinking water standards and ATSDR drinking water comparison values, results were compared to USEPA Regional Screening Levels (RSLs) for tap (drinking) water (USEPA 2010).

Levels of sodium and thallium in at least one surface water sample collected during base flow conditions exceed drinking water comparison values. The maximum concentrations of arsenic (maximum = 6.6 ppb; MMCL = 10 ppb), iron (maximum = 1,570 ppb; USEPA RSL = 26,000 ppb) and lead (maximum = 10.3 ppb; MMCL = 15 ppb) detected are

below comparison values (Tetra Tech 2005; MDEP 2010b). A summary of the maximum and average concentrations of contaminants detected in surface water samples collected during base flow conditions that exceed comparison values is presented in Table 3.

As discussed previously, it appears that access from Ginn Field to the abutting section of the Aberjona River is possible either through a large break in the chain link fence that separates the field from the river or from a paved pathway along Mystic Valley Parkway on the opposite side of the river. It is reasonable that some children may access the river during the warmer months of the year while athletic games are being played at Ginn Field. Therefore, it is possible that children could have been exposed via incidental ingestion of or dermal contact with sodium and thallium in the river.

Sodium is a naturally occurring element found in water and soil. It is an essential mineral, which is necessary for the normal functioning of the body and maintenance of body fluids. The Massachusetts guideline of 20 ppm in drinking water represents a level of sodium that sodium-sensitive individuals and their physicians should be aware of in cases where sodium intake is carefully controlled. People who have difficulty regulating fluid volume as a result of several diseases such as hypertension and kidney failure are particularly affected by elevated levels of sodium in drinking water (MDPH 2007). Since the water from the Aberjona River is not used as a drinking water source and any ingestion would be inadvertent and of small amounts while playing or wading, exposure to sodium in the surface water of the Aberjona River is not expected to result in adverse health effects.

Thallium is a naturally occurring element found in trace amounts in the earth's crust. Under a very conservative assumption that an adult, adolescent or younger child incidentally ingested surface water with the maximum concentration of thallium that was detected in Reach 5 (5.2 ppb) for one hour per day, 5 days per week during the warmer months of the year (May through September), the estimated exposures would not be expected to result in adverse noncancer health effects. See Appendix E for more information on the exposure calculations. With regard to dermal contact, no information

is available on the health effects of skin contact with thallium in either people or animals (ATSDR 1995b). Therefore, no further evaluation was conducted.

During storm flow conditions, several inorganic constituents (aluminum, arsenic, cadmium, chromium, copper, iron, lead, manganese, mercury, sodium, vanadium and zinc) had levels detected in at least one surface water sample that exceeded drinking water comparison values. A summary of the maximum and average concentrations of these inorganics is presented in Table 4.

Based on site conditions, it is not expected that children would play or wade in the section of the Aberjona River abutting Ginn Field during storms when water flow is heavier and sediment with inorganic constituents is stirred up on a regular basis. Therefore, exposure of adolescents or younger children to contaminants via incidental ingestion of or dermal contact with surface water while playing or wading in the Aberjona River during storm flow conditions is expected to occur very infrequently or on rare occasions, if ever.

Despite the low probability of exposure opportunities during storm events, MDPH did consider the unlikely exposure scenario of children exposed to surface water one day per week for 22 weeks per year during the warmer months of the year. Assuming the maximum concentration of each constituent, the estimated exposures would not result in adverse noncancer health effects or unusual cancer concerns. See Appendix F for more information on the exposure calculations.

D. Exposure to Groundwater

No groundwater investigations were conducted in Reaches 2 through 6 as part of the MSGRP Remedial Investigation (Tetra Tech 2005). Drinking water in the community of Winchester is supplied by local reservoirs and the Massachusetts Water Resources Authority (MWRA). All area residents use municipal water for their potable water supply. Groundwater is not used as a source of drinking water (J. Gibbons, Town of Winchester, personal communication, 2009). As a result, the MDPH concluded that no

further evaluation of groundwater near Ginn Field was necessary as this exposure pathway could be eliminated.

E. Exposure to Pesticides

The MDPH evaluated past and present pesticide use at Ginn Field as potential exposure sources. Records of pesticides that were applied to Ginn Field were available from 2003-2004 and obtained from the Winchester DPW. Three different herbicides were applied to Ginn Field during this time period, all of which are applied as a spray: Vanquish, Confront and Millennium Ultra. No pesticides have been applied to Ginn Field since 2005.

All pesticides sold or distributed in the United States must be registered by the USEPA indicating that they can be used without posing unreasonable risks to people or to the environment, based on scientific studies. As part of this process, the USEPA develops any mitigation measures or regulatory controls that are needed to effectively reduce the risks of the pesticide and requires that they be specified on the product label. Accordingly, it is a violation of federal law to use a pesticide in a manner inconsistent with the instructions on the product label. Furthermore, federal law also requires that the active ingredients of a pesticide be identified by name on the product label together with their percentage by weight. An active ingredient is defined as one which "prevents, destroys, repels or mitigates a pest, or is a plant regulator, defoliant, desiccant or nitrogen stabilizer." The active ingredients of the three herbicides used at Ginn Field during 2003-2004 consist of salts associated with clopyralid (3,6-dichloro-2-pyridinecarboxylic acid), triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid), dicamba (3,6-dichloro-o-anisic acid) and 2,4-D (2,4-dichlorophenoxyacetic acid). Information about the toxicity and carcinogenicity of each of these active ingredients was obtained from the IARC and the USEPA Office of Pesticide Programs (See summary in Table 5).

Clopyralid (an active ingredient of Confront and Millennium Ultra) and triclopyr (an active ingredient of Confront) belong to the pyridine family of herbicides, which are used to target broadleaf plants. Both compounds are moderately persistent in soil and are degraded primarily by microbial activity. Whereas triclopyr can also be broken down by

sunlight or water, clopyralid can not. Neither is volatile (Tu et al 2001, USEPA 1998). The application of these two compounds is restricted depending on the particular herbicide as specified on the product label. For non-agricultural uses, a maximum of two broadcast applications of Millennium Ultra is allowed per year per treatment site and the application of Confront is limited to a maximum of 4 pints per acre per year of treatment. In addition, restrictions on the label of both products specify that people are not allowed on the treatment area during application of these herbicides and must wait to enter such areas until the spray has dried (Dow AgroSciences 2007, 2008, Nufarm Americas 2006; USEPA 2002). This restriction reduces any potential exposure of individuals by minimizing possible contact. At Ginn Field, as well as at other fields managed by the Town of Winchester, signs are posted after a field has been sprayed to indicate that a pesticide has been applied (J. Gill, Town of Winchester, personal communication, 2009). Although potential exposures of children or adults who enter treated sites shortly after application may still exist, the USEPA determined that a post-application exposure assessment of triclopyr was not warranted because of its very low dermal toxicological characteristics (USEPA 1998). Similarly, clopyralid is not readily absorbed through the skin and also has low dermal toxicity (SERA 2004). Based on the application and use restrictions as well as the low dermal toxicity of these compounds, potential exposures from application to recreational turf are expected to be short-term in duration with possible acute health effects consisting mainly of eye and/or skin irritation. Therefore, long-term noncancer health effects are not expected to occur in either children or adults as a result of potential exposures from application of clopyralid or triclopyr to recreational turf at Ginn Field.

Finally, clopyralid has been classified by the USEPA as “not likely to be carcinogenic to humans.” Triclopyr has been classified by the USEPA as a Group D agent, indicating that insufficient information is available for classification. Group D is generally used for agents with inadequate human and animal evidence of carcinogenicity or for which no data are available (USEPA 2009c). In this case, it was based on marginal animal evidence and the absence of additional support from structurally similar compounds. Based on this information, it is not expected that exposure to either clopyralid or triclopyr at Ginn Field would result in an unusual cancer risk for either children or adults.

Dicamba (an active ingredient of Millennium Ultra and Vanquish) and 2,4-D (an active ingredient of Millennium Ultra) belong to the chlorophenoxy family of herbicides, which are used to selectively control broadleaf weeds. Both compounds are primarily broken down by microbial activity in soil with 2,4-D degrading rapidly and dicamba being moderately persistent. Neither is significantly broken down by sunlight or water in soil. Whereas dicamba has low volatility, 2,4-D rapidly dissipates from foliage (NPIC 2002, 2008; Nufarm Americas 2008, 2009; USEPA 2005b). As with clopyralid and triclopyr, the application of dicamba and 2,4-D is restricted depending on the particular herbicide as specified on the product label. As previously mentioned, a maximum of two broadcast applications of Millennium Ultra is allowed per year per treatment site for non-agricultural uses. Repeat applications of Vanquish may be made as needed on lawns and recreational turf but a maximum application rate of 2 pints per acre may not be exceeded during the growing season. Furthermore, only protected handlers are allowed on the treatment area during application of either Vanquish or Millennium Ultra and both herbicides should be applied in a manner such that they will not contact people, either directly or through drift (Nufarm Americas 2004, 2006; USEPA 2005c, 2006). Despite these restrictions, if post-application exposure of children or adults to dicamba or 2,4-D were to occur, the potential exposures are expected to be short-term in duration with possible acute health effects consisting of irritation of the skin, eyes and/or respiratory tract. While both dicamba and 2,4-D may be absorbed through the skin, both have low dermal toxicity (NPIC 2002, 2008; USEPA 2005d). Therefore, long-term noncancer health effects are not expected from post-application exposure of children or adults to dicamba or 2,4-D at Ginn Field.

With regard to carcinogenic effects, chlorophenoxy herbicides have been classified as Group 2B carcinogens by the IARC, indicating that they are possibly carcinogenic to humans. This category is used to denote an agent for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in laboratory animals (IARC 2009).

Dicamba, itself, was classified by the USEPA in 1996 as a Group D agent, indicating that it is not classifiable as to human carcinogenicity. However, it was later considered to be

"Not likely to be Carcinogenic to Humans" in accordance with the EPA Final Guidelines for Carcinogen Risk Assessment released in 2005. This designation was based on negative cancer studies in rats and mice which were tested at adequate dose levels to assess the carcinogenicity of dicamba (USEPA 2005d). As a result, it is not expected that exposure to dicamba at Ginn Field would result in an unusual cancer risk for either children or adults.

2,4-D was evaluated by the USEPA in 1988, 1992, and in 2004 due to concerns of a link with non-Hodgkin's lymphoma (NHL). Each evaluation concluded that "the data are not sufficient to conclude that there is a cause and effect relationship between exposure to 2,4-D and non-Hodgkin's lymphoma." In 2004, the USEPA categorized 2,4-D as a Group D agent, indicating that it is not classifiable as to human carcinogenicity due to insufficient data. Because 2,4-D is often mixed with other herbicides, it is difficult to determine whether carcinogenic effects may be linked to 2,4-D itself or to another ingredient (USEPA 2005b). MDPH examined the incidence of NHL in the community of Winchester and the geographic distribution of the addresses at the time of diagnosis to assess whether any unusual patterns might be evident in relation to Ginn Field. This evaluation of cancer incidence is discussed in Section V of this HC.

It is probably worthwhile to note that these four ingredients are common to many types of herbicides widely used on athletic fields and other recreational areas throughout the Commonwealth in accordance with label directions and with federal and state approval for using these herbicides in this manner (S. Kenyon, MDAR, personal communication, 2010). Thus, it is not expected that children at Ginn Field would be at greater risk of exposure opportunities or health effects because of the use of these herbicides than other children throughout the state.

V. ANALYSIS OF CANCER INCIDENCE

The community of Winchester has an approximate area of 6.3 square miles and a population of 20,500 (U.S. Census Bureau 2000). Childhood cancer incidence rates were calculated for the community of Winchester for the years 2000–2005, 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[3]. A qualitative review of childhood cancer in Winchester for more recent years (i.e., 2006-2008) was also conducted.

A. Methods for Analyzing Cancer Incidence

The term "childhood cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth in individuals aged 0 through 19 years. These diseases are classified using the International Classification of Childhood Cancers (ICCC) which is based primarily on the tissue or cell type of the cancer (histology). This is different from the system used to classify adult cancers, the International Classification of Diseases for Oncology (ICD-O), which is based on the location in the body where the cancer originated (primary site). The distinction between classification systems is important to consider, as the majority of pediatric cancers are disseminated when they are diagnosed and only the tissue of origin can be determined (Bleyer et al 2006). When evaluating cancer patterns in adults, primary site cancers are treated as different diseases because most have different causes and risk factors associated with their development. However, some childhood cancers may have similar etiologies and, therefore, are often grouped together to reflect similarities in histology or cell type [e.g., leukemias, lymphomas (including Hodgkin's disease and NHL), tumors of the central nervous system, and soft tissue sarcomas] (Birch and Marsden 1987; MCR 2003a; Steliarova-Foucher et al 2005).

As part of this investigation, the CAP reviewed incidence data available from the MCR for childhood cancer in Winchester. The 6-year period from 2000-2005 constituted the most recent and complete cancer incidence data that were available at the time of this report. [Coding for cancer types in this report follows the International Classification of Childhood Cancer (ICCC) system. See Appendix F for the incidence coding definitions used in this report.] The MCR, a division within the MDPH Bureau of Health Information, Statistics, Research, and Evaluation, is a population-based surveillance

[3] The data summarized in this report are drawn from data entered into the MCR before April 24, 2009. 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.

system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain in situ (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111. s 111b). The MCR also gathers background information (e.g. gender, age, and address at time of 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. This process corrects misclassification of data (i.e., city/town misassignment) and deletes duplicate diagnosis reports. Once these steps are finished, the data for that year are considered "complete." Due to the high volume of data collected, 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 be a minimum of two years prior to the current date. At the initiation of this analysis, complete data records available from the MCR included diagnoses that occurred from 1/1/1982 – 12/31/2005. However, it is possible to review diagnosis reports for more recent years (i.e., 2006-2008), which can provide a qualitative review of cancer patterns in a given area.

It should be noted that although some non-cancerous (i.e., benign) tumors are reported to the MCR (e.g., those diagnosed in the brain and central nervous system), these cancers were not included in the data summarized here. In addition, the MCR research file may contain duplicate reports of individuals diagnosed with cancer. Duplicate cases are additional reports of the same primary site cancer diagnosis. In Winchester, no duplicate reports were identified during the years 2000–2008. However, reports of individuals with multiple primary site cancers were included as separate diagnoses. 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 as an earlier cancer, if diagnosed in the same primary site more than two months after the initial diagnosis (MCR 2003b). Cancers that occur as a result of a primary site cancer spreading to another location in the body (i.e., metastasis) are not considered separate cancers and, therefore, were not included in this analysis.

To assess whether the incidence of childhood cancer in Winchester is unusual, a statistic called the standardized incidence ratio (SIR) was calculated using data from the MCR.

The SIR is a comparison of the number of diagnoses in the community to the number of expected diagnoses based on the statewide rate. Specifically, an SIR is the ratio of the observed number of cancer diagnoses in an area to the expected number of diagnoses multiplied by 100. Age-specific statewide incidence rates were applied to the population distribution of the community to calculate the number of expected cancer diagnoses.[4]

An SIR of 100 indicates that the number of cancer diagnoses observed in the population being evaluated is equal to the number of cancer diagnoses expected. An SIR greater than 100 indicates that more cancer diagnoses occurred than expected and an SIR less than 100 indicates that fewer cancer diagnoses occurred than expected. Accordingly, an SIR of 150 is interpreted as 50% more diagnoses than the expected number; an SIR of 90 indicates 10% fewer diagnoses than expected. To help interpret an SIR, the statistical significance of an SIR can be assessed by calculating a 95% confidence interval (CI) to determine if the observed number of diagnoses is "statistically significantly different" from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). When an SIR is statistically significant, there is less than a 5% percent chance that the observed difference (either increase or decrease) in the rate is the result of random fluctuation in the number of observed cancer diagnoses. It should be noted that SIRs and 95% CIs are not calculated when the observed number of diagnoses is fewer than five. Appendix G provides a more detailed explanation of SIRs and 95% CIs.

An SIR for childhood cancer (all types) for the community of Winchester was calculated for the 6-year period 2000-2005. Because childhood cancer is relatively rare, this report includes rates on all childhood cancers combined in the community of Winchester. This method was used to increase the power of comparisons of the incidence of childhood cancer in Winchester with that of the statewide experience. In addition, because statewide data for the years 2006-2008 were not complete when this analysis was begun, expected numbers of diagnoses and incidence ratios could not be calculated for this more recent time period. However, a qualitative review of childhood cancer diagnoses in Winchester during 2006-2008 was conducted.

[4] Using 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.

Address at the time of diagnosis was mapped for each child diagnosed with cancer in Winchester from 2000 to 2008 using a computerized geographic information system (GIS) (ESRI 2009). This allowed for a qualitative evaluation of the spatial distribution of the residence of children diagnosed with cancer at a smaller geographic level within the community (i.e., neighborhoods). The MDPH is bound by law not to make public the names or any other information (e.g., place of residence) that could personally identify individuals with cancer whose diagnoses have been reported to the MCR (M.G.L. c.111. s. 24A). Therefore, for confidentiality reasons, it is not possible for the MDPH to release maps showing the locations of individuals diagnosed with cancer in public reports. However, a summary of the evaluation of geographic distribution with any notable findings is presented in this report.

To better characterize the pattern of childhood cancer incidence in Winchester, diagnosis-specific information available from the MCR relating to the type of cancer, date of diagnosis, age at diagnosis, and gender was reviewed for each child diagnosed with cancer during 2000-2008. This information is discussed in the context of known or established cancer risk factors and incidence patterns in the general population. It should be noted, however, that very little is known about the etiology of childhood cancer. Unlike many cancers of adults, there are no avoidable risk factors (such as smoking or exposure to hazardous chemicals in the workplace) that are known to influence a child's risk of developing cancer.

B. Results of Cancer Incidence Analysis

1. Childhood Cancer Incidence in Winchester, 2000-2005

Table 6 summarizes the incidence of childhood cancer for the community of Winchester during the six-year time period of 2000-2005. Overall, cancer occurred slightly more often than expected among children aged 0-19 years in Winchester during this time period (7 diagnoses observed compared to about 6 expected). This difference was not statistically significant (SIR = 121, 95% CI = 49-250). A separate evaluation by gender revealed that a greater number of males were diagnosed with cancer than expected.

Among males, six diagnoses were observed compared to slightly more than three expected (SIR = 195). This elevation was not statistically significant (95% CI = 71-423).

The cancer types diagnosed among these 7 children during 2000-2005 included Central Nervous System and Miscellaneous Intracranial and Intraspinal Neoplasms [CNS tumors (n = 3)], Leukemias, Myeloproliferative Diseases and Myelodysplastic Diseases (n = 2), Neuroblastomas and Other Peripheral Nervous Cell Tumors (n = 1) and Germ Cell, Trophoblastic and Other Gonadal Neoplasms (n = 1). The number of diagnoses per year varied between zero and three with at least one diagnosis occurring in five of the six years.

The geographic distribution of the place of residence at the time of diagnosis for these 7 children was generally consistent with the population density in Winchester. None of the children lived within a ½-mile radius of Ginn Field at the time of their diagnosis. No unusual patterns were observed relative to space (i.e., in the vicinity of Ginn field) or time (i.e., dates of diagnosis).

2. Childhood Cancer Incidence in Winchester, 2006-2008

As stated earlier, expected numbers of diagnoses and SIRs cannot be calculated for more recent years because the statewide data for the years 2006 to present were not complete at the time of this analysis. This section, however, provides a qualitative review of childhood cancer in Winchester from 2006 to 2008.

From January 2006 to December 2008, six children (four males and two females) reported to the MCR as residents of Winchester were diagnosed with cancer. The cancer types diagnosed among these six children included Leukemias, Myeloproliferative Diseases and Myelodysplastic Diseases (n = 2), Malignant Bone Tumors (n = 1), Soft Tissue and Other Extraosseous Sarcomas (n = 1), Germ Cell, Trophoblastic and Other Gonadal Neoplasms (n = 1), and Other Malignant Epithelial Neoplasms and Malignant Melanomas (n = 1). Of the six diagnoses that were reported during this three-year time period, three occurred during 2006, one occurred in 2007, and two occurred in 2008.

Although some diagnoses occurred among children whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution of the place of residence at the time of diagnosis for these 6 children was generally consistent with the population density in Winchester. Three of the children lived within a ½-mile radius of Ginn Field at the time of their diagnosis. It should be noted that the places of residence for these three diagnoses were all located within areas of high population density. Overall, no unusual spatial patterns or concentrations of diagnoses were noted in the vicinity of the field or elsewhere in the community.

3. Incidence in Winchester by Type of Childhood Cancer

In general, several of the types of cancer diagnosed among children in Winchester during 2000-2008 are among the most common cancer types diagnosed among children (i.e., ages 0-19). Specific information on each of the seven cancer types diagnosed among children in Winchester during the time period evaluated is provided in the following sections.

a. Leukemias, Myeloproliferative Diseases and Myelodysplastic Diseases

Leukemia is a cancer of the bone marrow and blood. It is the most common type of childhood cancer, accounting for slightly more than 30% of all cancers diagnosed in children (ACS 2009a). Leukemia was the most common cancer type diagnosed among children in Winchester during 2000-2008. Of the 13 children diagnosed with cancer during this time period, four (31%) were diagnosed with leukemia. No temporal trends were observed among these diagnoses.

In children, leukemia is classified into four major subtypes: lymphoid leukemia, acute myeloid leukemia, chronic myeloproliferative disease, and myelodysplastic syndrome (Steliarova-Foucher et al 2005). In Massachusetts, the majority of childhood leukemia diagnoses are of the lymphoid leukemia subtype (MCR 2003a). During 2000-2008, all four diagnoses of leukemia in Winchester were lymphoid leukemias. According to the American Cancer Society (ACS), the risk for lymphoid leukemia is highest in children between 2 and 4 years of age. The risk then declines slowly until the mid-20s (ACS

2009b). One of the four children diagnosed in Winchester fits the pattern suggested by the ACS and was diagnosed between the ages of 2 and 4, whereas two children were between the ages of 5 and 10 years of age at diagnosis and the fourth child was between the ages of 11 and 19 years old at diagnosis.

b. Central Nervous System and Miscellaneous Intracranial and Intraspinal Neoplasms

Central nervous system and miscellaneous intracranial and intraspinal neoplasms (CNS tumors) include tumors that arise from the brain, spinal cord, and other sites within the skull and spinal cord. Nationally and statewide, these tumors are the second most common type of cancer in children. CNS tumors account for about 21% of all childhood cancers in the United States and about 17% of all childhood cancers in Massachusetts (MCR 2003a; ACS 2009c). In Winchester, three (23%) children were diagnosed with a CNS tumor from 2000-2008. No temporal trends were observed, with the number of diagnoses per year varying between zero and two.

There are several different types of CNS tumors. Gliomas are a general classification of CNS tumors that include a variety of types, named for the cells from which they arise: astrocytomas, oligodendrogliomas, and ependymomas. According to the ACS, gliomas account for approximately 80% of all malignant brain and CNS tumors and astrocytomas account for about half of all childhood brain tumors. Additional types of CNS tumors include primitive neuroectodermal tumors (PNETs), medulloblastomas, and other rare types (ACS 2009c). In Winchester, two children were diagnosed with gliomas and one child was diagnosed with an astrocytoma. According to the American Brain Tumor Association (ABTA), CNS tumors typically affect children under 15 years of age and older adults (ABTA 2008). Of the three children diagnosed with neoplasms of the CNS in Winchester from 2000-2008, all were diagnosed at ages younger than 15.

c. Germ Cell, Trophoblastic and Other Gonadal Neoplasms

Germ cell (egg or sperm), trophoblastic (cells outside an early embryo) and other gonadal (ovarian or testicular) neoplasms arise from reproductive cells. This group of cancers

accounts for about 7% of all cancers occurring among children less than 20 years old in the United States. The highest incidence rates of these types of neoplasms occur in adolescents between 15 and 19 years old, accounting for 16% of all cancers diagnosed in this age group. The majority (about 60%) of germ cell, trophoblastic, and other gonadal neoplasms are gonadal germ cell tumors (Ries et al 1999). The Massachusetts statewide experience is consistent with these national statistics (MCR 2003a). In Winchester, two children were diagnosed with malignant gonadal germ cell tumors during 2000-2008. Both were adolescents between the ages of 15 and 19 years old at the time of diagnosis.

d. Neuroblastoma and Other Peripheral Nervous Cell Tumors

Neuroblastoma occurs in the sympathetic nervous system, which regulates involuntary activities of the heart muscle, smooth muscle, and glands. It develops in certain types of developing nerve cells found in an embryo or fetus. Neuroblastoma accounts for over 97% of sympathetic nervous system tumors (Ries et al 1999). Nationally and statewide, neuroblastoma accounts for about 7% of all cancers in children (ACS 2008, 2009c; MCR 2003a). Neuroblastoma is by far the most common cancer in infants (less than 1 year old) and is rarely found in children older than 10 years (ACS 2008). In Winchester, one child was diagnosed with neuroblastoma during 2000-2008. This child was less than 1 year of age at the time of diagnosis.

e. Malignant Bone Tumors

Malignant bone tumors account for about 5% of all childhood cancers in Massachusetts, which is consistent with national statistics (MCR 2003a; Ries et al 1999). Osteosarcoma is the most common type of cancer that develops in bone and comprises about 66% of malignant bone tumors diagnosed in children in Massachusetts (ACS 2009d; MCR 2003a). Most osteosarcomas occur in children and young adults between the ages of 10 and 30. Teenagers comprise the most commonly affected age group and are at the highest risk during their “growth spurt” (ACS 2009d). In Winchester, one child was diagnosed with osteosarcoma during 2000-2008. This child was a teenager at the time of diagnosis.

f. Soft Tissue and Other Extraosseous Sarcomas

Soft tissue sarcomas are cancers that develop in connective tissues, such as muscle, fat and blood vessels, and can occur at any site throughout the body. Between 1990 and 1999, soft tissue sarcomas accounted for about 7% of all childhood cancers in Massachusetts, which coincides with national trends (MCR 2003a; Ries et al 1999). There are many different types of soft tissue sarcomas. The most common type of childhood soft tissue sarcoma is rhabdomyosarcoma, which develops in skeletal muscle. About 3% of childhood cancers are rhabdomyosarcomas (ACS 2007). According to the National Cancer Institute (NCI), the incidence of soft tissue sarcomas is highest among young children during infancy and children aged 15 to 19 years old (Ries et al 1999). In Winchester, one child was diagnosed with a soft-tissue sarcoma (rhabdomyosarcoma) during 2000-2008. This child was less than 1 year of age at the time of diagnosis.

g. Other Malignant Epithelial Neoplasms and Malignant Melanomas

This group of cancers includes several different types including adrenocortical carcinomas, thyroid carcinomas, nasopharyngeal carcinomas, malignant melanoma, and skin cancers. It also includes a category of cancers referred to as other and unspecified carcinomas (Steliarova-Fucher et al 2005). Carcinomas are many different cancers that develop from epithelial cells that form the lining of organs. The age-specific incidence rate of carcinomas and other malignant epithelial neoplasms among children in Massachusetts was highest among adolescents 15-19 years old (MCR 2003a). Of the 13 children diagnosed with cancer in Winchester during 2000-2008, one was diagnosed with an unspecified carcinoma as an adolescent. It was noted that this child also had a previous cancer diagnosis prior to the time period evaluated.

4. Incidence of Non-Hodgkin's Lymphoma in Winchester, 2000-2005

The incidence of non-Hodgkin's lymphoma among adults and children in the community of Winchester during 2000-2005 was reviewed in response to concerns of a possible association with the active pesticide ingredient 2,4-D. During this time period, the incidence among both males and females was slightly greater than expected based on the

statewide cancer experience, but neither elevation was statistically significant. A total of 20 males were diagnosed during this time period compared to about 16 expected (SIR = 124, 95% CI = 76-191). Among females, 18 diagnoses were observed and about 16 diagnoses were expected (SIR = 113, 95% CI = 67-179). No temporal trends were observed, with the number of diagnoses per year varying between four and eleven.

The geographic distribution of the place of residence at the time of diagnosis for those diagnosed with NHL during this time period was generally consistent with the population density pattern in Winchester. Six individuals lived within a ½-mile radius of Ginn Field at the time of their diagnosis. However, the places of residence for these six individuals were all located within areas of high population density. In addition, the ages at the time of diagnosis of those individuals diagnosed in Winchester during this time period followed what would be expected based on national trends. According to the ACS, approximately 90% to 95% of diagnoses of NHL occur in adults. The average age at diagnosis is in the 60s, and around half of patients are older than 65 (ACS 2009e). In Winchester, all of the diagnoses during this time period occurred in adults. The average age was 68 years and 61% of those diagnosed were older than age 65. Overall, no unusual spatial patterns or concentrations of diagnoses were noted in the vicinity of Ginn Field or elsewhere in the community.

VI. DISCUSSION

At the request of a concerned resident and the Winchester BOH, the MDPH conducted an evaluation of possible environmental exposures related to Ginn Field and the incidence of cancer in the community of Winchester. A detailed evaluation of the pattern of cancer in children was the primary focus. Concerns focused on potential exposures to contaminants in the surface water and sediment of the section of the Aberjona River that abuts the field as well as to contaminants in surface soil deposited by flood waters.

As part of this HC, the MDPH conducted a quantitative and qualitative evaluation of childhood cancer for the community of Winchester during 2000-2008 and examined the pattern of childhood cancer to identify any unusual concentrations of diagnoses. In addition, available environmental information for the stretch of the Aberjona River near

Ginn Field was assessed to determine possible pathways of exposure for adults and children visiting or playing at the field and available records of pesticide applications to the field were reviewed to determine if any active ingredients have been associated with long-term noncancer or carcinogenic effects.

Some potential exposure pathways relative to surface soil and sediment may have occurred in the past and could occur presently. However, based on highly conservative exposure assumptions, adverse health effects or unusual cancer risks are not expected to occur from exposure opportunities to constituents in sediment or surface soil. In the future, exposure to contaminants in surface soil deposited by flood waters may be eliminated as a pathway based on current projects to deepen the section of the Aberjona River abutting Ginn Field to reduce the frequency and severity of any future flooding. Likewise, future exposures to constituents in sediment are unlikely to cause health concerns given that there are no known ongoing sources and, hence, similar or lower concentrations are expected in the future.

It should be noted that no surface soil sample data specific to Ginn Field were available for review in this HC. The analyses conducted in this HC are based on the assumption that the concentrations of contaminants detected in the surface soil samples collected from floodplain soils located south of Bacon Street are representative of the floodplain soils at Ginn Field. It should also be noted that Ginn Field is primarily covered by grass with only a few areas of exposed dirt, such as the baseball diamonds, which would minimize incidental ingestion of soil by an individual.

Some potential exposure pathways relative to surface water may have occurred in the past and could occur presently or in the future. During base flow conditions, opportunities for exposure to sodium and thallium in surface water may be possible. During storm flow conditions, opportunities for exposure to several constituents may be possible, though unlikely. However, based on highly conservative exposure assumptions, adverse health effects or unusual cancer risks are not expected to result from any of these potential pathways.

Potential groundwater pathways were eliminated because it was not and is not used as a source of drinking water in Winchester. Drinking water is provided by a combination of local reservoirs and the MWRA.

Three different herbicides, containing a total of four active ingredients, were applied to Ginn Field in 2003-2004 (no pesticides have been applied since then). These ingredients are common to many types of herbicides and widely used on athletic fields and other recreational areas throughout Massachusetts. Long-term noncancer health effects are not expected to occur from opportunities for post-application exposure of children or adults to pesticides applied at Ginn Field during this time period due to the restricted frequency of use as well as the toxicological characteristics of clopyralid, triclopyr, dicamba and 2,4-D. The MDPH examined the incidence of NHL within the community of Winchester during 2000-2005. During this 6-year time period, the incidence of NHL among both males and females was slightly greater than expected but not statistically significant. No temporal trends were observed and no unusual spatial patterns were noted in the vicinity of the field or elsewhere in the community.

As part of this investigation, the CAP reviewed incidence data available from the MCR for childhood cancer in Winchester during the six-year time period, 2000-2005. This was the time period for which the most recent and complete cancer incidence data were available at the initiation of this analysis. A qualitative review of childhood cancer in Winchester for more recent years (i.e., 2006-2008) was also conducted.

In general, Winchester experienced a slightly elevated incidence of childhood cancer during 2000-2005 that was not statistically significant. The incidence among females was slightly less than expected, whereas that among males was more than expected but not statistically significant. Between 2000 and 2008, a total of 13 children in Winchester were diagnosed with the following seven types of cancer: leukemia, CNS tumors, malignant gonadal germ cell tumors, neuroblastoma, cancer of the bone, soft tissue sarcoma, and unspecified carcinoma.

In general, the types of cancers that occur in children vary greatly from those seen in adults. Leukemias, brain and CNS tumors, lymphomas, bone cancers, soft tissue

sarcomas, kidney cancers and eye cancers are the most common cancers in children. In contrast, the most common cancers among adults include skin, prostate, breast, lung, and colorectal cancers (ACS 2009f). In Massachusetts, a total of 1,720 children between the ages of 0 and 19 were diagnosed with cancer between 2000 and 2005. Leukemia was the most common type of childhood cancer, followed by cancers of the brain and CNS. Other common childhood cancers among Massachusetts children include Hodgkin's disease, NHL, soft tissue sarcoma, neuroblastoma, and some bone cancers. For the most part, the distribution of cancer types diagnosed among children in Winchester during 2000-2008 was consistent with state and national trends.

In addition, analysis of the geographic distribution of place of residence for children in Winchester diagnosed with cancer during 2000-2008 did not reveal any atypical spatial patterns that would suggest a common factor (environmental or non-environmental) is related to the incidence. That is, no apparent concentrations of diagnoses were observed in the vicinity of Ginn Field or elsewhere in the community that might suggest an association with a common environmental factor. Although some diagnoses during 2006-2008 occurred among children whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution was generally consistent with population density.

Unlike many cancers of adults, there are no avoidable risk factors (such as smoking or exposure to hazardous chemicals in the workplace) that are known to influence a child's risk of developing cancer. Unfortunately, the causes of childhood cancers are largely unknown. A few conditions, such as Down syndrome, other specific chromosomal and genetic abnormalities, and ionizing radiation exposures, explain a small percentage of diagnoses (NCI 2008). According to the NCI, environmental causes of childhood cancer have long been suspected but difficult to identify, partly because cancer in children is rare and because it is difficult to identify past exposure levels in children, particularly during potentially important periods such as pregnancy or even prior to conception. In addition, each of the distinctive types of childhood cancers develops differently, with a potentially wide variety of causes (NCI 2008).

According to the NCI, cancer is more common in male children than in female children. Based on data from the 1990s, childhood cancers are most prevalent among the white population followed by the Hispanic, Asian/Pacific Islander, African-American, and American Indian populations in the United States (Bleyer et al 2006). In addition, the types of cancer diagnosed among younger children (0-5 years of age) often vary from those diagnosed among older adolescents (15-19 years of age). For example, leukemias are most frequently diagnosed in younger children (with most diagnoses occurring before the age of 5) whereas lymphomas are the most frequently diagnosed type of cancer among adolescents (MCR 2003a; Bleyer et al 2006).

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

VII. CHILD HEALTH CONSIDERATIONS

The MDPH recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination in their environment. Children are at a greater risk than adults for certain kinds of exposure to hazardous

substances emitted from waste sites. They are more likely to be exposed because they play outdoors and because they often bring food into contaminated areas. Because of their smaller stature, they might breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of contaminant exposure per body weight. The developing body systems of children can sustain permanent damage if certain toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

The incidence and patterns of cancer among children in Winchester are discussed in Section V ("Analysis of Cancer Incidence") of this report. As mentioned previously, risk of exposure to contaminants in surface soil exists for children playing sports or visiting Ginn Field. Risk of exposure to contaminants in sediment and surface water also exists for children who wade or play in the section of the Aberjona River that abuts Ginn Field. However, it is unlikely that anyone would have contact with surface soil, sediment or surface water for a sufficient frequency and duration of time to result in health effects.

In addition, past exposure of children to pesticides that were sprayed at Ginn Field prior to 2006 may have been possible. However, based on the restricted frequency of use as well as the toxicological characteristics and carcinogenicity of the active ingredients of the pesticides used at Ginn Field, post-application exposure of children is not expected to harm their health.

VIII. LIMITATIONS

As part of this HC, descriptive health outcome data for cancer was analyzed to determine whether the pattern or occurrence of childhood cancer in the community of Winchester is unusual. The pattern of diagnoses of childhood cancer was evaluated in a geographical context in relation to available information about risk factors to determine whether further investigation seems warranted. Information from descriptive analyses, which may suggest a common etiology (or cause) is possible, can serve to identify areas where further analyses may be needed. Inherent limitations in the available data and this type of

analysis make it impossible to determine the precise casual relationships or synergistic roles that may have contributed to the development of individual cancers in this community. Cancers in general have a variety of risk factors known or suggested to be related to the etiology of the disease that could not be evaluated in this investigation. It is beyond the scope of this investigation to determine the causal relationship of these factors and the development of childhood cancer in Winchester. Also, this type of analysis cannot determine what may have caused cancer in any one particular individual.

IX. CONCLUSIONS

Based on the MDPH's evaluation of the available environmental data, the exposure pathway analysis, and risk factor information related to childhood cancer, MDPH concludes that:

- **Incidentally eating or touching soil at Ginn Field in the past, present or future** is not expected to harm the health of adults, adolescents or younger children playing at or visiting the field. The reason for this is because, based on available information and conservative assumptions about the frequency and duration of potential exposures, levels of metal contaminants that could get into an adult's or a child's body in the past or present are below levels that would harm their health. In the future, deposition of contaminants from flood waters onto the soil at Ginn Field is expected to be minimal after the channel is deepened in this section of the Aberjona River.

- **Incidentally eating or touching sediment while playing or wading in the Aberjona River near Ginn Field in the past, present or future** is not expected to harm the health of adults, adolescents or younger children. The reason for this is because, based on available information and conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into an adult's or a child's body in the past or present are below levels that would harm their health. Likewise, future exposures to constituents in sediment are unlikely to cause health concerns given that there are

no known ongoing sources and, hence, similar or lower concentrations are expected in the future.

- **Incidentally drinking or touching surface water while playing or wading in the Aberjona River near Ginn Field in the past, present or future** is not expected to harm the health of adults, adolescents or younger children. The reason for this is because, based on available information and conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into an adult's or a child's body during either base flow or storm flow conditions are below levels that would harm their health.

- **Drinking tap water in the community of Winchester was/is not impacted by contaminants from Superfund sites and the Aberjona River in the past, present or future** and, therefore, is not expected to harm people's health. The reason for this is because groundwater in the community of Winchester is not used as a source of drinking water. Rather, the drinking water is supplied by local reservoirs and the Massachusetts Water Resources Authority.

- **Incidentally touching pesticides that were sprayed at Ginn Field in the past** is not expected to harm people's health. The reason for this is because, based on the restricted frequency of use as well as the toxicological characteristics of the pesticides used at Ginn Field, post-application exposure of adults or children is not expected to harm their health. No pesticides have been applied to Ginn Field since 2005.

- **Within the community of Winchester, the incidence of childhood cancer during 2000-2005** occurred slightly more often than expected. This difference was not statistically significant. The incidence among females was slightly less than expected, whereas that among males was more than expected but not statistically significant. The histologies (cell types) and ages at diagnosis were generally consistent with state and national trends.

- **Within the community of Winchester, the geographic distribution of place of residence for children diagnosed with cancer during 2000-2008** did not reveal any atypical spatial patterns. Although some diagnoses during 2006-2008 occurred among children whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution of the place of residence at the time of diagnosis was generally consistent with population density.

X. RECOMMENDATIONS

The MDPH recommends no further investigation of childhood cancer incidence in Winchester at this time.

XI. REFERENCES

AECOM. 2010. Massachusetts Environmental Policy Act, Final Environmental Impact Report, EOEA File No. 13046, Aberjona River Flood Mitigation Program, Town of Winchester, MA. February 12.

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

ATSDR. 1995b. ToxFAQ for Thallium. Atlanta: U.S. Department of Health and Human Services. September.

ATSDR. 2005. Public health assessment guidance manual. Atlanta: U.S. Department of Health and Human Services.

ATSDR. 2007a. Toxicological Profile for Arsenic. Atlanta: U.S. Department of Health and Human Services. August.

ATSDR. 2007b. ToxFAQ for Lead. Atlanta: U.S. Department of Health and Human Services. August.

ATSDR. 2010a. Soil comparison values. Atlanta: U.S. Department of Health and Human Services. Last updated September 15.

ATSDR. 2010b. Drinking water comparison values. Atlanta: U.S. Department of Health and Human Services. Last updated June 30.

American Brain Tumor Association (ABTA). 2008. A Primer of Brain Tumors: Facts and Statistics. Available at: http://www.abta.org.

American Cancer Society (ACS). 2007. Detailed Guide: Rhabdomyosarcoma. Available at: http://www.cancer.org. Last updated December 18.

ACS. 2008. Detailed Guide: Neuroblastoma. Available at: http://www.cancer.org. Last updated October 22.

ACS. 2009a. Detailed Guide: Leukemia – Children's. Available at: http://www.cancer.org. Last updated May 14.

ACS. 2009b. Detailed Guide: Leukemia – Acute Lymphocytic (ALL). Available at: http://www.cancer.org. Last updated May 14.

ACS. 2009c. Detailed Guide: Brain / CNS Tumors in Children. Available at: http://www.cancer.org. Last updated May 14.

ACS. 2009d. Detailed Guide: Osteosarcoma. Available at: http://www.cancer.org. Last updated January 14.

ACS. 2009e. Detailed Guide: Lymphoma, Non-Hodgkin Type. Available at http://www.cancer.org. Last updated July 17.

ACS. 2009f. Detailed Guide: Cancer in Children. Available at: http://www.cancer.org. Last updated May 14.

Birch JM and Marsden HB. 1987. A classification scheme for childhood cancer. Int J Cancer; 40(5):620-624.

Bleyer A, O'Leary M, Barr R and Ries LAG (eds). 2006. Cancer Epidemiology in Older Adolescents and Young Adults 15 to 29 Years of Age, Including SEER Incidence and Survival: 1975-2000. National Cancer Institute, NIH Pub. No. 06-5767. Bethesda, MD.

Childhood Lead Poisoning Prevention Program (CLPPP). 2010a. Massachusetts Department of Public Health, Bureau of Environmental Health, Childhood Lead Poisoning Prevention Program. Cited December 21. Available at: http://www.state.ma.us/dph/clppp.

CLPPP. 2010b. Lead Safe Homes - Database for Lead Inspected Homes. Available at: http://webapps.ehs.state.ma.us/Leadsafehomes/default.aspx. Accessed on December 28.

Dow AgroSciences LLC. 2007. Material Safety Data Sheet: Confront Herbicide. July 5.

Dow AgroSciences LLC. 2008. Specimen Label: Confront Specialty Herbicide. June 20.

Environmental Systems Research Institute (ESRI). 2009. ArcGIS, ArcView license, ver. 9.3.1, Redlands, California.

International Agency for Research on Cancer (IARC). 2009. Agents Reviewed by the IARC Monographs (Volumes 1-100A). April.

Marra, D. 2011. Winchester river-widening project falls behind. Winchester Patch. November 16.

Massachusetts Cancer Registry (MCR). 2003a. Childhood Cancer in Massachusetts, 1990-1999. Massachusetts Department of Public Health, Bureau of Health Statistics, Research, and Evaluation. Boston; March.

MCR. 2003b. Massachusetts cancer registry abstracting and coding manual for hospitals. 5th ed. Massachusetts Department of Public Health, Bureau of Health Information, Statistics, Research, and Evaluation. Boston; December. Includes revisions through July 2007.

Massachusetts Department of Environmental Protection (MDEP). 2002. Background levels of polycyclic aromatic hydrocarbons and metals in soil. Office of Research and Standards.

MDEP. 2010. Standards and guidelines for contaminants in Massachusetts drinking waters. Office of Research and Standards.

Massachusetts Department of Public Health (MDPH). 2007. Bureau of Environmental Health. Sodium in Drinking Water Fact Sheet. Revised May 2007.

National Cancer Institute (NCI). 2008. Childhood Cancer: Questions and Answers. Available at: http://www.cancer.gov. Last updated January 10.

National Pesticide Information Center (NPIC). 2002. Dicamba: Technical Fact Sheet. Available at: http://npic.orst.edu/factsheets/dicamba_tech.pdf. Last updated January.

NPIC. 2008. 2,4-D: Technical Fact Sheet. Available at: http://npic.orst.edu/factsheets/2,4-DTech.pdf. Last updated November.

Nufarm Americas Inc. 2004. Specimen Label: Vanquish Herbicide. November 2.

Nufarm Americas Inc. 2006. Specimen Label: Millennium Ultra. February 27.

Nufarm Americas Inc. 2008. Material Safety Data Sheet: Millennium Ultra. May 14.

Nufarm Americas Inc. 2009. Material Safety Data Sheet: Vanquish Herbicide. July 1.

Ries LG, Smith MA, Gurney JG, Linet M, Tamra T, Young JL, Bunin GR (eds). 1999. Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975-1995. National Cancer Institute, SEER Program. NIH Pub. No. 99-4649. Bethesda, MD.

Rothman KJ and Boice JD. 1982. Epidemiologic analysis with a programmable calculator. Boston: Epidemiology Resources, Inc.

Steliarova-Foucher E, Stiller C, Lacour B and Kaatsch P. 2005. International Classification of Childhood Cancer, Third edition. Cancer, 103, 1457-1467.

Syracuse Environmental Research Associates, Inc. (SERA). 2004. Clopyralid - Human Health and Ecological Risk Assessment - Final Report. SERA TR 04-43-17-03c. December 5.

Tetra Tech NUS, Inc. (Tetra Tech). 2005. Draft Final MSGRP Remedial Investigation Report: Industri-Plex Site, Woburn, Massachusetts. March.

Tu M, Hurd C, Robinson R and Randall JM. 2001. Weed Control Methods Handbook, The Nature Conservancy. http://www.invasive.org/gist/products/handbook/methods-handbook.pdf. April.

United States Census Bureau. 2000. 2000 census of population: general population characteristics, Massachusetts. Washington, D.C.: U.S. Department of Commerce.

United States Environmental Protection Agency (USEPA). 1989. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part A). December.

USEPA. 1998. Reregistration Eligibility Decision (RED): Triclopyr. EPA 738-98-011. October.

USEPA. 2002. Clopyralid; Pesticide Tolerance. Federal Register. 67:186. p. 60152-60161. September 25.

USEPA. 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). July.

USEPA. 2005a. Aberjona River Study: Comprehensive Multiple Source Groundwater Response Plan Remedial Investigation. April. Available at: http://www.epa.gov/ne/superfund/sites/industriplex/213095.pdf.

USEPA. 2005b. Reregistration Eligibility Decision for 2,4-D. EPA 738-R-05-002. June.

USEPA. 2005c. 2,4-D: HED's Revised Human Health Risk Assessment for the Reregistration Eligibility Decision (RED) Revised to Reflect Public Comments. May 12.

USEPA. 2005d. Dicamba: HED Chapter of the Reregistration Eligibility Decision Document (RED) – Phase 1. September 13.

USEPA. 2006. Reregistration Eligibility Decision for Dicamba and Associated Salts. June 8.

USEPA. 2007. User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows. May. Office of Superfund Remediation and Technology Innovation.

USEPA. 2009a. Waste Site Cleanup and Reuse in New England: Industri-Plex, Woburn, Massachusetts. Available at: http://www.epa.gov/region1/superfund/sites/industriplex. Last updated June 23.

USEPA. 2009b. Waste Site Cleanup and Reuse in New England: Wells G & H, Woburn, Massachusetts. Available at: http://www.epa.gov/region1/superfund/sites/wellsgh. Last updated January 2.

USEPA. 2009c. Office of Pesticide Programs List of Chemicals Evaluated for Carcinogenic Potential. September 3.

USEPA. 2009d. Child-Specific Exposure Factors. September. Office of Research and Development. Available at: http://cfpub.epa.gov/ncea/CFM/recordisplay.cfm?deid=199243.

USEPA. 2010. Human Health Risk Assessment: Regional Screening Levels. Available at: http://www.epa.gov/reg3hwmd/risk/human/index.htm. Last updated May.

United States Geological Survey (USGS). 1984. Shacklette HT, Boerngen JG. Element concentrations in soils and other surficial materials of the conterminous United States. USGS Professional Paper 1270. Washington, D.C.: U.S. Government Printing Office.

FIGURES

Figure 1
Site Location
Ginn Field, Winchester, Massachusetts

Figure 2
Location of Ginn Field, Wells G & H and the Industri-Plex Site
Winchester and Woburn, Massachusetts

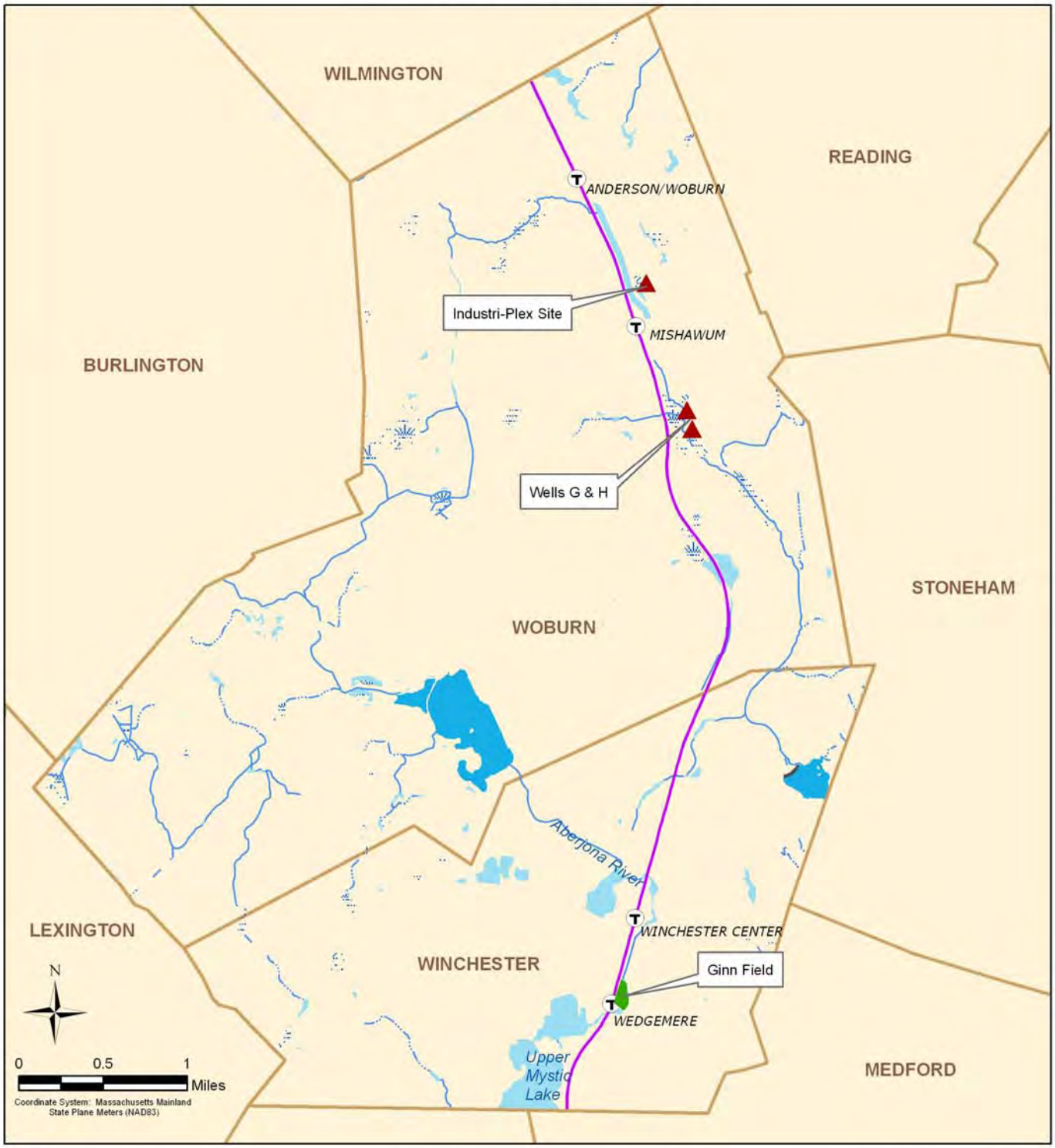

Figure 3
Reach 5 of the Aberjona River
Winchester, Massachusetts

TABLES

Table 1
Maximum and Average Concentration of Contaminants Detected in Surface Soil Samples that Exceed Comparison Values
Aberjona River, Reach 5
Winchester, Massachusetts

Contaminant	Frequency of Detection	Maximum Concentration (ppm)	Sample Name	Average Concentration (ppm)	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Arsenic	7 / 9	98	AJRW22	46.4	CREG = 0.5 Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200	<0.1 - 73* 20 (natural soil)†
Chromium	9 / 9	90	AJRW18	39.9	Chronic EMEG (child) = 50 Chronic EMEG (adult) = 700	1 - 1,000* 30 (natural soil)†
Lead	9 / 9	930	AJRW16	298	EPA RSL (residential) = 400	<10 - 300* 100 (natural soil)†

*Observed Range for the Eastern United States (east of 96th meridian). USGS. 1984. Shacklette HT, Boerngen JG. Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States. U.S. Geological Survey Professional Paper 1270.

†MDEP. 2002. Background Levels of Polycyclic Aromatic Hydrocarbons and Metals in Soil. Office of Research and Standards.

ppm = parts per million

< = less than

Comparison Values:

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2005, 2010a)

CREG = Cancer Risk Evaluation Guide for 1 x 10^{-6} excess cancer risk (ATSDR 2005, 2010a)

EPA RSL = EPA Region 3 Regional Screening Level for soil (USEPA 2010)

Data Sources:

Tetra Tech NUS, Inc. (Tetra Tech). 2005. Draft Final MSGRP Remedial Investigation Report: Industri-Plex Site, Woburn, Massachusetts. March.

Table 2
Maximum and Average Concentration of Contaminants Detected in Sediment Samples that Exceed Comparison Values
Aberjona River
Winchester, Massachusetts

Contaminant	Frequency of Detection	Maximum Concentration (ppm)	Sample Name	Average Concentration (ppm)	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Arsenic	4 / 4	32.7	SDISCA-1	18.2	CREG = 0.5 Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200	<0.1 - 73* 20 (natural soil)††
Benzo(a)anthracene	4 / 4	3.7	SDISCA-1	2.7	EPA RSL (residential) = 0.15 CREG = 1**	0.005 - 0.02 (rural soil)† 0.169 - 59 (urban soil)† 2 (natural soil)††
Benzo(a)pyrene	4 / 4	3.5	SDISCA-1	2.4	CREG = 0.1	0.002 - 1.3 (rural soil)† 0.165 - 0.22 (urban soil)† 2 (natural soil)††
Benzo(b)fluoranthene	4 / 4	3.4	SDISCB-3	2.4	EPA RSL (residential) = 0.15 CREG = 1**	0.02 - 0.03 (rural soil)† 15 - 62 (urban soil)† 2 (natural soil)††
Benzo(k)fluoranthene	4 / 4	3.4	SDISCA-1	2.0	EPA RSL (residential) = 1.5 CREG = 1**	0.01 - 0.11 (rural soil)† 0.3 - 26 (urban soil)† 1 (natural soil)††
Chromium	4 / 4	119	SDISCA-1	68	Chronic EMEG (child) = 50 Chronic EMEG (adult) = 700	1 - 1,000* 30 (natural soil)††
Dibenzo(a,h)anthracene	4 / 4	0.44	SDISCB-3	0.31	EPA RSL (residential) = 0.015 CREG = 0.02**	0.5 (natural soil)††
Indeno(1,2,3-cd)pyrene	4 / 4	2.1	SDISCA-1	1.4	EPA RSL (residential) = 0.15 CREG = 1**	0.01 - 0.015 (rural soil)† 8.0 - 61 (urban soil)† 1 (natural soil)††

*Observed range for the Eastern United States (east of 96th meridian). USGS. 1984. Shacklette HT, Boerngen JG. Element Concentrations in Soils and Other Surficial Materials of the conterminous United States. U.S. Geological Survey Professional Paper 1270.

† ATSDR. 1995. Toxicological profile for polyaromatic hydrocarbons. Atlanta: U.S. Department of Health and Human Services.

†† MDEP. 2002. Background levels of polycyclic aromatic hydrocarbons and metals in soil. Office of Research and Standards.

ppm = parts per million

< = less than

Comparison Values:

Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2005, 2010a)

CREG = Cancer Risk Evaluation Guide for 1 x 10^{-6} excess cancer risk (ATSDR 2005, 2010a)

CREG** = Estimated CREG using toxicity equivalency factors relative to benzo(a)pyrene developed by USEPA.

EPA RSL = EPA Region 3 Regional Screening Levels for soil (USEPA 2010)

Data Sources:

AECOM. 2010. Massachusetts Environmental Policy Act, Final Environmental Impact Report, EOEA File No. 13046, Aberjona River Flood Mitigation Program, Town of Winchester, MA. February 12.

Table 3
Maximum and Average Concentrations of Contaminants Detected in Surface Water Samples During Base Flow Conditions that Exceed Comparison Values
Aberjona River, Reach 5
Winchester, Massachusetts

Contaminant	Frequency of Detection	Maximum Concentration (ppb)	Date of Sample	Average Concentration (ppb)	Drinking Water Comparison Value (ppb)
Sodium	17 / 17	141,000 (J)	2/15/2002	80,100	MDEP Guideline = 20,000
Thallium	1 / 16	5.2	1/4/2002	2.0	MDEP MMCL = 2

(J) Exact value not quantified in laboratory analytical results

ppb = parts per billion

Comparison Values:
MDEP Guideline = Massachusetts Department of Environmental Protection Drinking Water Guideline (MDEP 2010)
MDEP MMCL = Massachusetts Department of Environmental Protection Massachusetts Maximum Contaminant Level (MDEP 2010)

Data Sources:
Tetra Tech NUS, Inc. (Tetra Tech). 2005. Draft Final MSGRP Remedial Investigation Report: Industri-Plex Site, Woburn, Massachusetts. March.

Notes:
Due to the lack of health-based comparison values for surface water, results were compared to Massachusetts drinking water standards. When a Massachusetts drinking water standard was not available, results were compared to ATSDR drinking water comparison

Table 4
Maximum and Average Concentrations of Contaminants Detected in Surface Water Samples During Storm Flow Conditions that Exceed Comparison Values
Aberjona River, Reach 5
Winchester, Massachusetts

Contaminant	Frequency of Detection	Maximum Concentration (ppb)	Date of Sample	Average Concentration (ppb)	Drinking Water Comparison Value (ppb)
Aluminum	51 / 88	58,300	5/16/2002	1,490	Chronic EMEG (child) = 10,000 Chronic EMEG (adult) = 40,000
Arsenic	85 / 88	427 (J)	5/16/2002	15.6	MDEP MMCL = 10
Cadmium	4 / 88	25.5 (J)	5/16/2002	0.63	MDEP MMCL = 5
Chromium	70 / 88	1,070	5/16/2002	26.7	MDEP MMCL = 100
Copper	72 / 88	1,320	5/16/2002	39.6	MDEP MMCL, Action Level = 1,300
Iron	88 / 88	145,000	5/16/2002	4,330	EPA RSL = 26,000
Lead	65 / 88	1,420	5/16/2002	38.5	MDEP MMCL, Action Level = 15
Manganese	88 / 88	14,600	5/16/2002	478	RMEG (child) = 500 RMEG (adult) = 2,000
Mercury	25 / 83	3.7	5/16/2002	0.12	MDEP MMCL = 2
Sodium	88 / 88	80,000	4/26/2002	38,400	MDEP Guideline = 20,000
Vanadium	42 / 88	170	5/16/2002	4.9	Int. EMEG (child) = 100 Int. EMEG (adult) = 400
Zinc	88 / 88	5,070	5/16/2002	153	Chronic EMEG (child) = 3,000 Chronic EMEG (adult) = 10,000

(J) Exact value not quantified in laboratory analytical results
ppb = parts per billion

Comparison Values:
Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2005, 2010b)
Intermediate EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures between 14 days and 1 year) (ATSDR 2005, 2010b)
MDEP Guideline = Massachusetts Department of Environmental Protection Drinking Water Guideline (MDEP 2010)
MDEP MMCL = Massachusetts Department of Environmental Protection Massachusetts Maximum Contaminant Level (MDEP 2010)
EPA RSL = EPA Region 3 Regional Screening Levels for tap water (USEPA 2010)

Data Sources:
Tetra Tech NUS, Inc. (Tetra Tech). 2005. Draft Final MSGRP Remedial Investigation Report: Industri-Plex Site, Woburn, Massachusetts. March.

Notes:
Due to the lack of health-based comparison values for surface water, results were compared to Massachusetts drinking water standards. When a Massachusetts drinking water standard was not available, results were compared to ATSDR drinking water comparison

Table 5: Carcinogenic Classifications of Active Ingredients of Pesticides Applied to Ginn Field, 2003 - 2004

Pesticide Name	Active Ingredient (% by Weight)	CAS No.	IARC Classification	USEPA Classification
Confront Herbicide	Triclopyr Triethylamine (TEA) Salt (33%)	57213-69-1	Not classified	Class D - Not Classifiable as to Human Carcinogenicity
	Clopyralid Triethylamine (TEA) Salt (12.1%)	119308-91-7	Not classified	Not Likely to be Carcinogenic to Humans
Millennium Ultra Herbicide	Dimethylamine Salt of 2,4-Dichlorophenoxyacetic Acid (2,4-D) (37.32%)	2008-39-1	Group 2B*	Class D - Not Classifiable as to Human Carcinogenicity
	Monoethanolamine Salt of Clopyralid (3,6-Dichloro-2-Pyridinecarboxylic Acid) (2.54%)	57754-85-5	Not classified	Not Likely to be Carcinogenic to Humans
	Dimethylene Salt of Dicamba (3,6-Dichloro-o-anisic Acid) (4.65%)	2300-66-5	Group 2B*	Not Likely to be Carcinogenic to Humans
Vanquish Herbicide	Diglycolamine Salt of Dicamba (3,6-Dichloro-o-anisic Acid) (56.8%)	1040440-79-1	Group 2B*	Not Likely to be Carcinogenic to Humans

*The IARC lists chlorophenoxy herbicides as Group 2B carcinogens.

IARC Classification:
Group 1 - The agent is carcinogenic to humans.
Group 2A - The agent is probably carcinogenic to humans.
Group 2B - The agent is possibly carcinogenic to humans.
Group 3 - The agent is not classifiable as to its carcinogenicity to humans.
Group 4 - The agent is probably not carcinogenic to humans.

Data Sources:
Dow AgroSciences LLC. 2007. Material Safety Data Sheet: Confront Herbicide. July 5.
International Agency for Research on Cancer (IARC). 2009. Agents Reviewed by the IARC Monographs (Volumes 1-100A). April.
Nufarm Americas Inc. 2009. Material Safety Data Sheet: Vanquish Herbicide. July 1.
Nufarm Americas Inc. 2008. Material Safety Data Sheet: Millenium Ultra. May 14.
USEPA. 2009e. Office of Pesticide Programs List of Chemicals Evaluated for Carcinogenic Potential. September 3.
USEPA. 2005b. Dicamba: HED Chapter of the Reregistration Eligibility Decision Document (RED) – Phase 1. September 13.

TABLE 6
Cancer Incidence
Winchester, Massachusetts
2000-2005

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
All Childhood Cancer	7	5.8	121	49 -- 250	6	3.1	195	71 -- 423	1	2.7	NC	NC -- NC

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

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

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

APPENDICES

APPENDIX A: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA INGESTION OF SURFACE SOIL

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Soil Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{soil} \times IR \times NC\_EF \times CF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose:

$$C\_D = \frac{[C]_{soil} \times IR \times C\_EF \times CF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{soil}$	= Maximum Analyte Concentration in Soil (mg/kg)
IR	= Soil Ingestion Rate (mg/day)
CF	= Conversion Factor (kg/mg)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum concentration of arsenic and lead detected in surface soil samples was assumed as the soil concentration.
3. The amount of soil ingested was assumed to be 100 milligrams per day for the adult receptor and 200 milligrams per day for the child and adolescent receptors.
4. The exposure factor was determined assuming the receptors were exposed to surface soil 5 days per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
5. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.
6. The cancer exposure dose for an adolescent was added to that of a child to represent the situation in which an adolescent was also exposed as a child.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Soil Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Ingestion of Surface Soil Containing Arsenic:

1. **Adult (age 18 and over)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.3 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000042\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.13$$

$$\text{Cancer Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.13 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000018\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000018 \times 1.5 = 0.000027$$

2. **Adolescent (ages 12 to less than 18)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30 \times 10^{-6}\ \text{kg/mg}}{50\ \text{kg}} = 0.00012\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.03$$

$$\text{Cancer Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.03 \times 10^{-6}\ \text{kg/mg}}{50\ \text{kg}} = 0.00001\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00001 \times 1.5 = 0.000015$$

3. **Child (under age 12)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30 \times 10^{-6}\ \text{kg/mg}}{30\ \text{kg}} = 0.00020\ \text{mg/kg/day}$$

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Soil
Ginn Field, Winchester, Massachusetts

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

$$\text{Cancer Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.05 \times 10^{-6}\ \text{kg/mg}}{30\ \text{kg}} = 0.000031\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000031 \times 1.5 = 0.000046$$

4. Adolescent and Child

Cancer Risk = Cancer Risk of Child + Cancer Risk of Adolescent

Cancer Risk = 0.000015 + 0.000046 = 0.000061

NOTES:

1. The ATSDR Chronic Oral MRL for arsenic is 0.0003 mg/kg/day.
2. The USEPA Oral Cancer Slope Factor for arsenic is 1.5 $mg/kg/day^{-1}$.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Soil
Ginn Field, Winchester, Massachusetts

Exposure Dose and Noncancer Risk Calculations for Ingestion of Surface Soil Containing Lead:

1. **Adult (age 18 and over)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{930\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.30\ \text{x}\ 10^{-6}}{70\ \text{kg}} = 0.0004\ \text{mg/kg/day}$$

2. **Adolescent (ages 12 to less than 18)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{930\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30\ \text{x}\ 10^{-6}}{50\ \text{kg}} = 0.0011\ \text{mg/kg/day}$$

3. **Child (under age 12)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{930\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30\ \text{x}\ 10^{-6}}{30\ \text{kg}} = 0.0019\ \text{mg/kg/day}$$

NOTES:

1. There is no ATSDR MRL or USEPA RfD available for lead. The calculated exposure dose for lead was input into the USEPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows® (IEUBKwin, Lead Model Version 1.1, Build 9) to estimate blood lead (PbB) levels in children exposed to lead-contaminated media. The IEUBK model results indicated that exposure to the average concentration of lead detected in surface soil (298 mg/kg) would not result in a predicted mean blood lead concentration above 10 μg/dL, which the CDC defines as a level of concern.
2. The USEPA has categorized lead as a probable human carcinogen; however, they have concluded that existing scientific information cannot determine whether or not exposure to lead can cause cancer in humans; thus, no USEPA Oral Cancer Slope Factor has been developed for lead. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for lead.

APPENDIX B: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA DERMAL CONTACT WITH SURFACE SOIL

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Surface Soil Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{soil} \times SAF \times SA \times AF \times CF \times NC\_EF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose:

$$C\_D = \frac{[C]_{soil} \times SAF \times SA \times AF \times CF \times C\_EF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{soil}$	= Maximum Analyte Concentration in Surface Soil (mg/kg)
SAF	= Soil Adherence Factor (mg/cm^2)
SA	= Exposed Body Surface Area (cm^2)
AF	= Absorption Factor (Dermal) (unitless)
CF	= Conversion Factor (kg/mg)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum concentration of arsenic and lead detected in surface soil samples was assumed as the soil concentration.
3. The exposure factor was determined assuming the receptors were exposed to surface soil 5 days per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
4. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.
5. The cancer exposure dose for an adolescent was added to that of a child to represent the situation in which an adolescent was also exposed as a child.

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Surface Soil Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Dermal Contact with Surface Soil Containing Arsenic:

1. Adult (age 18 and above)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 5700\text{cm}^2 \times 0.03 \times 10^{-6}\ \text{kg/mg} \times 0.30}{70\ \text{kg}} = 0.000022\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.13$$

$$\text{Cancer Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 5700\ \text{cm}^2 \times 0.03 \times 10^{-6}\ \text{kg/mg} \times 0.13}{70\ \text{kg}} = 0.0000093\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000079 \times 1.5 = 0.000014$$

2. Adolescent (ages 12 to less than 18)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 4266\text{cm}^2 \times 0.03 \times 10^{-6}\ \text{kg/mg} \times 0.30}{50\ \text{kg}} = 0.000023\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.03$$

$$\text{Cancer Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 0.3\ \text{mg/cm2} \times 4266\ \text{cm2} \times 0.03 \times 10^{-6}\ \text{kg/mg} \times 0.03}{50\ \text{kg}} = 0.000002\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000002 \times 1.5 = 0.000003$$

3. Child (under age 12)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{98\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 2625\text{cm}^2 \times 0.03 \times 10^{-6}\ \text{kg/mg} \times 0.30}{30\ \text{kg}} = 0.000016\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Surface Soil Ginn Field, Winchester, Massachusetts

$$\text{Cancer Effects Exposure Dose} = \frac{98\,\text{mg/kg} \times 0.2\text{mg/cm2} \times 2625\text{cm2} \times 0.03 \times 10^{-6}\,\text{kg/mg} \times 0.05}{30\,\text{kg}} = 0.0000024\,\text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000024 \times 1.5 = 0.0000037$$

4. Adolescent and Child

Cancer Risk = Cancer Risk of Child + Cancer Risk of Adolescent

Cancer Risk = 0.000003 + 0.0000037 = 0.000067

NOTES:

1. The ATSDR Chronic MRL for arsenic is 0.0003 mg/kg/day.
2. The USEPA Oral Cancer Slope Factor for arsenic is 1.5 $mg/kg/day^{-1}$.

APPENDIX C: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA INGESTION OF SEDIMENT

APPENDIX C

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Sediment Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{soil} \times IR \times NC\_EF \times CF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose:

$$C\_D = \frac{[C]_{soil} \times IR \times C\_EF \times CF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{soil}$	= Maximum Analyte Concentration in Sediment (mg/kg)
IR	= Sediment Ingestion Rate (mg/day)
CF	= Conversion Factor (kg/mg)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum concentration of benzo(a)pyrene detected in sediment samples was assumed as the sediment concentration.
3. The amount of sediment ingested was assumed to be 100 milligrams per day for the adult receptor and 200 milligrams per day for the child and adolescent receptors.
4. The exposure factor was determined assuming the receptors were exposed to sediment 5 days per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
5. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.
6. The cancer exposure dose for an adolescent was added to that of a child to represent the situation in which an adolescent was also exposed as a child.

APPENDIX C

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Sediment Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Ingestion of Sediment Containing Benzo(a)pyrene:

1. Adult (age 18 and over)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.30 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000015\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.13$$

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.13 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00000065\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000065 \times 7.3 = 0.0000047$$

2. Adolescent (ages 12 to less than 18)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30 \times 10^{-6}\ \text{kg/mg}}{50\ \text{kg}} = 0.0000042\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.03$$

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.03 \times 10^{-6}\ \text{kg/mg}}{50\ \text{kg}} = 0.0000004\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000004 \times 7.3 = 0.000003$$

3. Child (under age 12)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.30 \times 10^{-6}\ \text{kg/mg}}{30\ \text{kg}} = 0.000007\ \text{mg/kg/day}$$

APPENDIX C

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Sediment Ginn Field, Winchester, Massachusetts

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.05 \times 10^{-6}\ \text{kg/mg}}{30\ \text{kg}} = 0.000001\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000001 \times 7.3 = 0.000007$$

4. Adolescent and Child

Cancer Risk = Cancer Risk of Child + Cancer Risk of Adolescent

Cancer Risk = 0.000003 + 0.000007 = 0.00001

NOTES:

1. The USEPA RfD for pyrene (0.03 mg/kg/day) was used to evaluate noncancer health effects from exposure to benzo(a)pyrene.
2. The USEPA Oral Cancer Slope Factor for benzo(a)pyrene is 7.3 $mg/kg/day^{-1}$.

APPENDIX D: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA DERMAL CONTACT WITH SEDIMENT

APPENDIX D

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Sediment Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{soil} \times SAF \times SA \times AF \times CF \times NC\_EF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose:

$$C\_D = \frac{[C]_{soil} \times SAF \times SA \times AF \times CF \times C\_EF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{soil}$	= Maximum Analyte Concentration in Sediment (mg/kg)
SAF	= Soil Adherence Factor (mg/cm^2)
SA	= Exposed Body Surface Area (cm^2)
AF	= Absorption Factor (Dermal) (unitless)
CF	= Conversion Factor (kg/mg)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum concentration of benzo(a)pyrene detected in sediment samples was assumed as the sediment concentration.
3. The exposure factor was determined assuming the receptors were exposed to sediment 5 days per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
4. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.
5. The cancer exposure dose for an adolescent was added to that of a child to represent the situation in which an adolescent was also exposed as a child.

APPENDIX D

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Sediment Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Dermal Contact with Sediment Containing Benzo(a)pyrene:

1. **Adult (age 18 and over)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 5700\text{cm}^2 \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.30}{70\ \text{kg}} = 0.0000033\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.13$$

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 5700\ \text{cm}^2 \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.13}{70\ \text{kg}} = 0.0000014\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000014 \times 7.3 = 0.00001$$

2. **Adolescent (ages 12 to less than 18)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.3\ \text{mg/cm}^2 \times 4266\text{cm}^2 \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.30}{50\ \text{kg}} = 0.0000035\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.03$$

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.3\ \text{mg/cm2} \times 4266\ \text{cm2} \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.03}{50\ \text{kg}} = 0.0000003\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000003 \times 7.3 = 0.000002$$

3. **Child (under age 12)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 2625\text{cm}^2 \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.30}{30\ \text{kg}} = 0.0000024\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.05$$

APPENDIX D

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with Sediment Ginn Field, Winchester, Massachusetts

$$\text{Cancer Effects Exposure Dose} = \frac{3.5\ \text{mg/kg} \times 0.2\ \text{mg/cm2} \times 2625\ \text{cm2} \times 0.13 \times 10^{-6}\ \text{kg/mg} \times 0.05}{30\ \text{kg}} = 0.0000004\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000004 \times 7.3 = 0.000003$$

4. Adolescent and Child

Cancer Risk = Cancer Risk of Child + Cancer Risk of Adolescent

Cancer Risk = 0.000002 + 0.000003 = 0.000005

NOTES:

1. The USEPA RfD for pyrene (0.03 mg/kg/day) was used to evaluate noncancer health effects from exposure to benzo(a)pyrene.
2. The USEPA Oral Cancer Slope Factor for benzo(a)pyrene is 7.3 $mg/kg/day^{-1}$.

APPENDIX E: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA INGESTION OF SURFACE WATER DURING BASE FLOW CONDITIONS

APPENDIX E

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Water During Base Flow Conditions
Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{surface\ water} \times IR \times NC\_EF}{BW}$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{surface\ water}$	= Maximum Analyte Concentration in Surface Water (mg/L)
IR	= Surface Water Ingestion Rate (L/day)
BW	= Body Weight (kg)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum concentration of thallium detected in surface water during base flow conditions was assumed as the surface water concentration.
3. The amount of surface water ingested was assumed to be 0.05 liters per day.
4. The exposure factor was determined assuming the receptors were exposed to surface water 5 days per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
5. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.

APPENDIX E

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Water During Base Flow Conditions
Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Ingestion of Surface Water Containing Thallium:

1. **Adult (age 18 and over)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.0052\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.30}{70\ \text{kg}} = 0.0000011\ \text{mg/kg/day}$$

2. **Adolescent (ages 12 to less than 18)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.0052\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.30}{50\ \text{kg}} = 0.0000016\ \text{mg/kg/day}$$

3. **Child (under age 12)**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{110\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.30$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.0052\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.30}{30\ \text{kg}} = 0.0000026\ \text{mg/kg/day}$$

NOTES:

1. The USEPA RfD for thallium sulfate (0.0008 mg/kg/day) was used to evaluate noncancer health effects from thallium detected in Reach 5.
2. The USEPA has not classified thallium with respect to its cancer causing potential and has not developed a USEPA Oral Cancer Slope Factor for thallium. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for thallium.

APPENDIX F: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA INGESTION OF SURFACE WATER DURING STORM FLOW CONDITIONS

APPENDIX F

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Water During Storm Flow Conditions
Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

$$NC\_EF = \frac{F \times ED}{ED \times 365\ days}$$

Noncancer Health Effects Exposure Dose:

$$NC\_D = \frac{[C]_{surface\ water} \times IR \times NC\_EF}{BW}$$

Cancer Effects Exposure Factor:

$$C\_EF = \frac{F \times ED}{70\ years \times 365\ days}$$

Cancer Effects Exposure Dose:

$$C\_D = \frac{[C]_{surface\ water} \times IR \times C\_EF}{BW}$$

Cancer Risk:

$$CR = C\_D \times CSF$$

Where:

NC_EF	= Noncancer Exposure Factor (unitless)
F	= Frequency of Exposure (days/year)
ED	= Years of Exposure (years)
NC_D	= Noncancer Exposure Dose (mg/kg/day)
$[C]_{surface\ water}$	= Maximum Analyte Concentration in Surface Water (mg/L)
IR	= Surface Water Ingestion Rate (L/day)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1. The receptors evaluated were children (under age 12), adolescents (ages 12 to less than 18) and adults (age 18 and above).
2. The maximum contaminant concentration detected in surface water during storm flow conditions was assumed as the surface water concentration.
3. The amount of surface water ingested was assumed to be 0.05 liters per day.
4. The exposure factor was determined assuming the receptors were exposed to surface water 1 day per week, for 22 weeks per year over a 30 year time period for an adult, a 6 year time period for an adolescent and an 11 year time period for a child.
5. The average body weight was assumed to be as follows: adult = 70 kilograms; adolescent = 50 kilograms; and child = 30 kilograms.
6. The cancer exposure dose for an adolescent was added to that of a child to represent the situation in which an adolescent was also exposed as a child.

APPENDIX F

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Water During Storm Flow Conditions
Ginn Field, Winchester, Massachusetts

Exposure Dose and Cancer Risk Calculations for Ingestion of Surface Water Containing Arsenic:

1. Adult (age 18 and over)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{22\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.427\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.06}{70\ \text{kg}} = 0.000018\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{22\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.026$$

$$\text{Cancer Effects Exposure Dose} = \frac{0.427\ \text{mg/kg} \times 0.05\ \text{L/day} \times 0.026}{70\ \text{kg}} = 0.0000079\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000079 \times 1.5 = 0.000012$$

2. Adolescent (ages 12 to less than 18)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{22\ \text{days/year} \times 6\ \text{years}}{6\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.427\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.06}{50\ \text{kg}} = 0.000026\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{22\ \text{days/year} \times 6\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.005$$

$$\text{Cancer Effects Exposure Dose} = \frac{0.427\ \text{mg/kg} \times 0.05\ \text{L/day} \times 0.005}{50\ \text{kg}} = 0.0000021\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000021 \times 1.5 = 0.0000032$$

3. Child (under age 12)

$$\text{Noncancer Health Effects Exposure Factor} = \frac{22\ \text{days/year} \times 11\ \text{years}}{11\ \text{years} \times 365\ \text{days}} = 0.06$$

APPENDIX F

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of Surface Water During Storm Flow Conditions
Ginn Field, Winchester, Massachusetts

$$\text{Noncancer Health Effects Exposure Dose} = \frac{0.427\ \text{mg/L} \times 0.05\ \text{L/day} \times 0.06}{30\ \text{kg}} = 0.000043\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{22\ \text{days/year} \times 11\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.009$$

$$\text{Cancer Effects Exposure Dose} = \frac{0.427\ \text{mg/kg} \times 0.05\ \text{L/day} \times 0.009}{30\ \text{kg}} = 0.0000064\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000064 \times 1.5 = 0.000010$$

4. Adolescent and Child

Cancer Risk = Cancer Risk of Child + Cancer Risk of Adolescent

Cancer Risk = 0.0000032 + 0.000010 = 0.000013

NOTES:

1. The ATSDR Chronic Oral MRL for arsenic is 0.0003 mg/kg/day.
2. The USEPA Oral Cancer Slope Factor for arsenic is 1.5 mg/kg/day^{-1}.
3. The calculations shown above for arsenic are provided as an example of those for the other constituents that had levels detected in at least one surface water sample that exceeded drinking water comparison values (aluminum, cadmium, chromium, copper, iron, lead, manganese, mercury, sodium, vanadium and zinc).

APPENDIX G: CODING DEFINITIONS OF CHILDHOOD CANCER

Appendix G
Coding Definitions of Childhood Cancer

ICCC Diagnostic Group	ICD-O-3** codes	
	Morphology	**Topography**
I. Leukemias, myeloproliferative diseases, and myelodysplastic diseases		
a. Lymphoid leukemias	9820, 9823, 9826, 9827, 9831-9837, 9940, 9948	
b. Acute myeloid leukemias	9840, 9861, 9866, 9867, 9870-9874, 9891, 9895-9897, 9910, 9920, 9931	
c. Chronic myeloproliferative diseases	9863, 9875, 9876, 9950, 9960-9964	
d. Myelodysplastic syndrome and other myeloproliferative diseases	9945, 9946, 9975, 9980, 9982-9987, 9989	
e. Unspecified and other specified leukemias	9800, 9801, 9805, 9860, 9930	
II. Lymphomas and reticuloendothelial neoplasms		
a. Hodgkin Lymphomas	9650-9655, 9659, 9661-9665, 9667	
b. Non-Hodgkin lymphomas (except Burkitt lymphoma)	9591, 9670, 9671, 9673, 9675, 9678-9680, 9684, 9689-9691, 9695, 9698-9702, 9705, 9708, 9709, 9714, 9716-9719, 9727-9729, 9731-9734, 9760-9762, 9764-9769, 9970	
c. Burkitt lymphomas	9687	
d. Miscellaneous lymphoreticular neoplasms	9740-9742, 9750, 9754-9758	
e. Unspecified lymphomas	9590, 9596	
III. Central nervous system and miscellaneous intracranial and intraspinal neoplasms		
a. Ependymomas and choroid plexus tumors	9383, 9390-9394[a]	
b. Astrocytomas	9380[a]	C72.3
	9384, 9400-9411, 9420, 9421-9424, 9440-9442[a]	
c. Intracranial and intraspinal embryonal tumors	9470-9474, 9480, 9508[a]	
	9501-9504[a]	C70.0-C72.9
d. Other gliomas	9380[a]	C70.0-C72.2, C72.4-C72.9, C75.1, C75.3
	9381, 9382, 9430, 9444, 9450, 9451, 9460[a]	
e. Other specified intracranial and intraspinal neoplasms	8270-8281, 8300, 9350-9352, 9360-9362, 9412, 9413, 9492, 9493, 9505-9507, 9530-9539, 9582[a]	

* Chart from: Steliarova-Foucher E, Stiller C, Lacour B and Kaatsch P. 2005. International Classification of Childhood Cancer, Third edition. *Cancer*, **103**, 1457-1467.

** *International Classification of Diseases for Oncology, 3rd Ed.*

f. Unspecified intracranial and Intraspinal neoplasms	8000-8005[a]	C70.0-C72.9, C75.1-C75.3
IV. Neuroblastoma and other peripheral nervous cell tumors		
a. Neuroblastoma and ganglioneuroblastoma	9490, 9500	
b. Other peripheral nervous cell tumors	8680-8683, 8690-8693, 8700, 9520-9523	
	9501-9504	C00.0-C69.9, C73.9-C76.8, C80.9
V. Retinoblastoma	9510-9514	
VI. Renal Tumors		
a. Nephroblastoma and other nonepithelial renal tumors	8959, 8960, 8964-8967	
	8963, 9364	C64.9
b. Renal carcinoma	8010-8041, 8050-8075, 8082, 8120-8122, 8130-8141, 8143, 8155, 8190-8201, 8210, 8211, 8221-8231, 8240, 8241, 8244-8246, 8260-8263, 8290, 8310, 8320, 8323, 8401, 8430, 8440, 8480-8490, 8504, 8510, 8550, 8560-8576	C64.9
	8311, 8312, 8316-8319, 8361	
c. Unspecified malignant renal tumors	8000-8005	C64.9
VII. Hepatic Tumors		
a. Hepatoblastoma	8970	
b. Hepatic carcinoma	8010-8041, 8050-8075, 8082, 8120-8122, 8140, 8141, 8143, 8155, 8190-8201, 8210, 8211, 8230, 8231, 8240, 8241, 8244-8246, 8260-8264, 8310, 8320, 8323, 8401, 8430, 8440, 8480-8490, 8504, 8510, 8550, 8560-8576	C22.0, C22.1
	8160-8180	
c. Unspecified malignant hepatic tumors	8000-8005	C22.0, C22.1
VIII. Malignant bone tumors		
a. Osteosarcomas	9180-9187, 9191-9195, 9200	C40.0-C41.9, C76.0-C76.8, C80.9
b. Chondrosarcomas	9210, 9220, 9240	C40.0-41.9, 76.0-76.8, 80.9
	9221, 9230, 9241-9243	
c. Ewing tumor and related sarcomas of bone	9260	C40.0-C41.9, C76.0-76.8, C80.9
	9363-9365	C40.0-C41.9
d. Other specified malignant bone tumors	8810, 8811, 8823, 8830	C40.0-C41.9
	8812, 9250, 9261, 9270-9275, 9280-9282, 9290, 9300-9302, 9310-9312, 9320-9322, 9330, 9340-9342, 9370-9372	C40.0-C41.9
e. Unspecified malignant bone tumors	8000-8005, 8800, 8801, 8803-8805	C40.0-C41.9

IX. Soft tissue and other extraosseous sarcomas		
a. Rhabdomyosarcomas	8900-8905, 8910, 8912, 8920, 8991	
b. Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	8810, 8811, 8813-8815, 8821, 8823, 8834-8835	C00.0-C39.9, C44.0-76.8, C80.9
c. Kaposi sarcoma	9140	
d. Other specified soft tissue sarcomas	8587, 8710-8713, 8806, 8831-8833, 8836, 8840-8842, 8850-8858, 8860-8862, 8870, 8880, 8881, 8890-8898, 8921, 8982, 8990, 9040-9044, 9120-9125, 9130-9133, 9135, 9136, 9141, 9142, 9161, 9170-9175, 9231, 9251, 9252, 9373, 9581	
	8830	C00.0-C39.9, C44.0-C76.8, C80.9
	8963	C00.0-C63.9, C65.9-C69.9, C73.9-C76.8, C80.9
	9180, 9210, 9220, 9240	C49.0-C49.9
	9260	C00.0-C39.9, C47.0-C75.9
	9364	C00.0-C39.9, C47.0-C63.9, C65.9-C69.9, C73.9-C76.8, C80.9
	9365	C00.0-C39.9, C47.0-C63.9, C65.9-C76.8, C80.9
e. Unspecified soft tissue sarcomas	8800-8805	C00.0-C39.9, C44.0-C76.8
X. Germ cell tumors, trophoblastic tumors, and neoplasms of gonads		
a. Intracranial and Intraspinal germ cell tumors	9060-9065, 9070-9072, 9080-9085, 9100, 9101[a]	C70.0-C72.9, C75.1-C75.3
b. Malignant extracranial and extragonadal germ cell tumor	9060-9065, 9070-9072, 9080-9085, 9100-9105	C00.0-C55.9, C57.0-C61.9, C63.0-C69.9, C73.9-C75.0, C75.4-C76.8, C80.9
c. Malignant gonadal germ cell tumors	9060-9065, 9070-9073, 9080-9085, 9090, 9091, 9100, 9101	C56.9, C62.0-C62.9
d. Gonadal carcinomas	8010-8041, 8050-8075, 8082, 8120-8122, 8130-8141, 8143, 8190-8201, 8210, 8211, 8221-8241, 8244-8246, 8260-8263, 8290, 8310, 8313, 8320, 8323, 8380-8384, 8430, 8440, 8480-8490, 8504, 8510, 8550, 8560-8573, 9000, 9014, 9015	C56.9, C62.0-C62.9
	8441-8447, 8450, 8451, 8460-8473	
e. Other and unspecified malignant gonadal tumors	8590-8671	
	8000-8005	C56.9, C62.0-C62.9

Appendix G
Coding Definitions of Childhood Cancer

XI. Other malignant epithelial neoplasms and malignant melanomas		
a. Adrenocortical carcinomas	8370-8375	
b. Thyroid carcinomas	8010-8041, 8050-8075, 8082, 8120-8122, 8130-8141, 8190, 8200, 8201, 8211, 8230, 8231, 8244-8246, 8260-8263, 8290, 8310, 8320, 8323, 8430, 8440, 8480, 8481, 8510, 8560-8573	C73.9
	8330-8337, 8340-8347, 8350	
c. Nasopharyngeal carcinomas	8010-8041, 8050-8075, 8082, 8083, 8120-8122, 8130-8141, 8190, 8200, 8201, 8211, 8230, 8231, 8244-8246, 8260-8263, 8290, 8310, 8320, 8323, 8430, 8440, 8480, 8481, 8500-8576	C11.0-C11.9
d. Malignant melanomas	8720-8780, 8790	
e. Skin carcinomas	8010-8041, 8050-8075, 8078, 8082, 8090-8110, 8140, 8143, 8147, 8190, 8200, 8240, 8246, 8247, 8260, 8310, 8320, 8323, 8390-8420, 8430, 8480, 8542, 8560, 8570-8573, 8940, 8941	C44.0-C44.9
f. Other and unspecified carcinomas	8010-8084, 8120-8157, 8190-8264, 8290, 8310, 8313-8315, 8320-8325, 8360, 8380-8384, 8430-8440, 8452-8454, 8480-8586, 8588-8589, 8940, 8941, 8983, 9000, 9010-9016, 9020, 9030	C00.0-C10.9, C12.9-C21.8, C23.9-C39.9, C48.0-C48.8, C50.0-C55.9, C57.0-C61.9, C63.0-C63.9, C65.9-C72.9, C75.0-C76.8, C80.9
XII. Other and unspecified malignant neoplasms		
a. Other specified malignant tumors	8930-8936, 8950, 8951, 8971-8981, 9050-9055, 9110	
	9363	C00.0-C39.9, C47.0-C75.9
b. Other unspecified malignant tumors	8000-8005	C00.0-C21.8, C23.9-C39.9, C42.0-C55.9, C57.0-C61.9, C63.0-C63.9, C65.9-C69.9, C73.9-C75.0, C75.4-C80.9

APPENDIX H: EXPLANATION OF STANDARDIZED INCIDENCE RATIO (SIR) AND 95% CONFIDENCE INTERVAL

Appendix H
Explanation of a Standardized Incidence Ratio (SIR) And 95% Confidence Interval

To determine whether an elevation is occurring among individuals diagnosed with cancer in a community or census tract (CT), cancer incidence data are tabulated by gender according to eighteen age groups to compare the observed number of cancer diagnoses to the number that would be expected based on the statewide cancer rate.
Specifically, an SIR is the ratio of the observed number of cancer diagnoses in an area to the expected number of diagnoses multiplied by 100. Age-specific statewide incidence rates are applied to the population distribution of a community to calculate the number of expected cancer diagnoses. The SIR is a comparison of the number of diagnoses in the specific area (i.e., community or census tract) to the number of expected diagnoses based on the statewide rate. Comparison of SIRs between communities or census tracts is not possible because each of these areas has different population characteristics.

To calculate an SIR, it is necessary to obtain accurate population information. Population is interpolated based on U.S. census data for the community of interest. Midpoint population estimates are calculated for each time period evaluated. To estimate the population between census years, an assumption is made that the change in population occurs at a constant rate throughout the ten-year interval between each census.

A CT is a geographic subdivision of a city or town designated by the United States Census Bureau. Because age group and gender-specific population information is necessary to calculate incidence rates, the CT is the smallest geographic area for which cancer rates can be accurately calculated. Specifically, a CT is a smaller statistical subdivision of a county as defined by the U.S. Census Bureau. CTs usually contain between 1,500 and 8,000 persons and are designed to be homogenous with respect to population characteristics (U.S. DOC 2000).

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

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

To help interpret or measure the stability of an SIR, the statistical significance of an SIR can be assessed by calculating a 95% confidence interval (95% CI) to determine if the observed number of diagnoses is "statistically 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 has 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 5% percent chance that the observed difference (either increase or decrease) in the rate is the result of random fluctuation in the number of observed cancer diagnoses.

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

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 diagnoses, statistical significance is not assessed when fewer than five diagnoses are observed.