Massachusetts Department Of Public Health

Health Consultation: Evaluation of Four Facilities within the New Bedford Business Park and Cancer Incidence in Two Census Tracts in New Bedford, Bristol County, Massachusetts

Former BorgWarner TorqTransfer Systems, Inc., Former Polymerine, Former Tallyrand, and Former Polaroid

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I. SUMMARY

Introduction:	This health consultation was conducted because residents and former health officials of the community of New Bedford, Massachusetts, were concerned about possible environmental exposures from the New Bedford Business Park and potential health impacts (i.e., cancer) in two nearby neighborhoods.
Overview:	The MDPH has reached several important conclusions about possible environmental exposures from the New Bedford Business Park and cancer incidence in the census tracts/neighborhoods of concern.
Conclusion 1:	The MDPH concluded that health effects in nearby residents are not expected to result from environmental exposures associated with impacts to drinking water or indoor air at the former BorgWarner, Polymerine, Tallyrand, and Polaroid sites in the past, present, or future.
Basis for Decision:	Groundwater at the former BorgWarner, Polymerine, Tallyrand and Polaroid sites was not used as a source of drinking water historically, nor is it at present. The surrounding neighborhoods are generally served by the City of New Bedford Water Department. Although private drinking water wells may exist on Braley Road, they are located upgradient. Therefore, ingestion of or dermal contact with contaminants in groundwater was eliminated as an exposure pathway in the past, present and future. In addition, exposure of nearby residents to groundwater contaminants via vapor intrusion is not expected in the past, present and future due to the distance to the nearest residences.
Conclusion 2:	The MDPH concluded that incidentally eating or touching soil at the former Polymerine or former Tallyrand sites in the past, present or future is not expected to result in health effects.
Basis for Decision:	Past activities at the former Polymerine and Tallyrand sites resulted in chemical contaminants in on-site soil. While it is possible that some trespassers could have come into contact with chemical contaminants at either site, the available information does not suggest health impacts would be expected. Conservative assumptions about the frequency and duration of potential exposures demonstrate that the levels of chemical contaminants that could get into an older child's body are below levels that would result in adverse non-cancer or cancer health effects. Remediation at the former Polymerine and Tallyrand sites included the removal of contaminated soil. Confirmatory sampling at the former Polymerine site indicated that concentrations of PCBs still remain in on-site soils at levels above the applicable regulatory clean-up standards.

Although PCB concentrations at the former Tallyrand site are not expected to exceed the USEPA action level, it is possible that exceedances of health-based comparison values could still occur. Using conservative assumptions about the frequency and duration of potential exposures, present and future incidental ingestion of or dermal contact with PCBs in soil by trespassers at either the former Polymerine or Tallyrand sites are not expected to result in adverse non-cancer or cancer health effects.

Conclusion 3: The MDPH concluded that incidentally eating or touching soil at the former BorgWarner or Polaroid sites in the past, present or future is not expected to result in health effects.

Basis for Decision: Due to the depth below ground surface at which contamination was detected at the former BorgWarner and Polaroid sites, no exposure pathways were complete in the past, present or future. In addition, remedial activities at both sites included the excavation and disposal of contaminated soil.

Conclusion 4: The MDPH concluded that breathing chemicals in outdoor air impacted by emissions from the former Polaroid facility was possible for nearby residents when the facility was in operation. While acute impacts at that time may have been possible, there are no ambient air data available to evaluate whether facility emissions may have resulted in health impacts in the past. Based on available hospital discharge data, the number of asthma hospitalizations within zip code 02745 during 2000-2003 does not appear to be related to the level of emissions of MEK, a respiratory irritant, from the former Polaroid facility.

Basis for Decision: Since there are no historical ambient air monitoring data available for the former Polaroid facility or the surrounding neighborhoods, it is difficult to evaluate whether facility emissions may have resulted in chemical concentrations in ambient air greater than health-based comparison values. However, since MEK is a respiratory irritant, the MDPH examined hospital discharge data for asthma hospitalizations of residents of zip code 02745 during 2000-2003. This four-year time period reflects the most complete data available through 2003, after which point MEK was no longer emitted from the former Polaroid facility. Overall, there does not appear to be a relationship between the level of emissions of MEK from the former Polaroid facility and the number of asthma hospitalizations within the zip code. The level of emissions of MEK generally decreased over this time period and a corresponding decrease in the number of asthma hospitalizations was not observed.

Conclusion 5: The MDPH concluded that the incidence of bladder and breast cancer in the census tract (CT) that contains the Briarwood Development was either less than or about as expected during 1996-2000 and 2001-2005 compared

to the statewide cancer experience. Although no elevations were statistically significant, the incidence of kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate cancer was slightly elevated or elevated during one or both time periods evaluated.

Basis for Decision: To determine whether the incidence of cancer in CT 6501.01 was elevated, the observed number of cancer diagnoses in the CT was compared to the number that would be expected based on the statewide cancer rate. In each of the two time periods evaluated (1996-2000 and 2001-2005), the incidence of bladder and breast occurred either less than or about as expected in both genders.

The incidence of kidney/renal pelvis cancer was about as expected among males in both time periods. Among females, kidney/renal pelvis cancer was slightly elevated during 1996-2000 (5 diagnoses observed compared to about 2 expected) but the difference was not statistically significant.

Among males, the incidence of liver/intrahepatic bile duct cancer was slightly elevated during both 1996-2000 (2 diagnoses observed compared to 1 expected) and 2001-2005 (3 diagnoses observed compared to about 1 expected). Neither difference was statistically significant. Among females, the incidence of this cancer type was as expected during both time periods.

The incidence of lung and bronchus cancer was either less than or about as expected among females during both time periods evaluated. Among males, the incidence of lung and bronchus cancer was elevated during 1996-2000 (19 diagnoses observed compared to about 13 expected) but the elevation was not statistically significant. During 2001-2005, the incidence of lung and bronchus cancer among males was about as expected.

The incidence of prostate cancer was less than expected during 1996-2000 but greater than expected during 2001-2005 (31 diagnoses observed compared to about 25 expected). This difference was not statistically significant.

Age at diagnosis, histology (cell type), and the temporal pattern of diagnoses were evaluated separately for those individuals diagnosed with kidney/renal pelvis cancer, liver/intrahepatic bile duct, lung and bronchus cancer, or prostate cancer. No unusual patterns emerged. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution generally followed the population density of the CT.

Review of risk factor information suggests that tobacco use likely played some role in the development of kidney/renal pelvis and lung and bronchus cancer among some individuals. Occupational exposures may have also been important in the development of these cancer types among some individuals. It should also be noted that exposure to radon has been identified as the second leading cause of lung and bronchus cancer, and the leading cause among nonsmokers. Testing of individual homes is the only way to find out the radon level in a home.

Conclusion 6: The MDPH concluded that the incidence of bladder, breast, and lung and bronchus cancer in the census tract (CT) that contains the Pine Hill Acres neighborhood occurred either less than or about as expected during 1996-2000 and 2001-2005 compared to the statewide cancer experience. Although no elevations were statistically significant, the incidence of kidney/renal pelvis, liver/intrahepatic bile duct and prostate cancer was either slightly elevated or elevated during one of the two time periods evaluated.

Basis for Decision: To determine whether the incidence of cancer in CT 6501.02 was elevated, the observed number of cancer diagnoses in the CT was compared to the number that would be expected based on the statewide cancer rate. In each of the two time periods evaluated (1996-2000 and 2001-2005), the incidence of bladder, breast, and lung and bronchus cancer in both genders occurred either less than or about as expected.

The incidence of kidney/renal pelvis cancer among females occurred either less than or about as expected during both time periods. Among males, the incidence of kidney/renal pelvis cancer was slightly elevated during 1996-2000 (4 diagnoses observed compared to about 2 expected). This elevation, however, was not statistically significant. During 2001-2005, the incidence of kidney/renal pelvis cancer among males was about as expected.

In CT 6501.02, no diagnoses of liver/intrahepatic bile duct cancer were observed during 1996-2000. During 2001-2005, the incidence of liver/intrahepatic bile duct cancer among males was about as expected (1 observed diagnosis compared to about 1 expected) whereas that among females was slightly elevated (1 observed diagnosis compared to 0 expected). These differences were not statistically significant.

The incidence of prostate cancer was less than expected during 1996-2000 but greater than expected during 2001-2005 (19 diagnoses observed compared to about 16 expected). This slight elevation was not statistically significant.

Age at diagnosis, histology (cell type), and the temporal pattern of diagnoses were evaluated separately for those individuals diagnosed with kidney/renal pelvis, liver/intrahepatic bile duct and prostate cancer. No unusual patterns emerged for these cancer types. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution generally followed the population density of the CT.

Next Steps:

❖ The MDPH recommends no further investigation of cancer incidence in CTs 6501.01 and 6501.02 at this time.

❖ The MDPH recommends that all residences in Massachusetts be tested for radon. The only way to determine if your home has a radon problem is to do a radon test. For further questions about radon, you may contact MDPH/BEH's Radiation Control Program toll free at (800) 723-6695 for advice on home testing.

❖ The MDPH recommends that residents who would like more information about quitting smoking contact the Massachusetts Tobacco Cessation and Prevention Program at 1-800-Quit-Now or 1-800-784-8669 or visit the website http://makesmokinghistory.org/.

For More Information: If you have questions about this report, you may call the MDPH/BEH at 617-624-5757.

II. Introduction and Statement of Issues

At the request of community members and former New Bedford health officials, the Community Assessment Program (CAP) within the Massachusetts Department of Public Health's (MDPH) Bureau of Environmental Health (BEH) conducted an evaluation of possible environmental exposures related to the New Bedford Business Park and cancer incidence for two New Bedford census tracts in the vicinity of the business park (see Figure 1)[1]. Community members were concerned about cancer in two neighborhoods in northern New Bedford near the business park as well as possible dumping, possible groundwater and surface water contamination, and possible impacts of air emissions from a former Polaroid Corporation (Polaroid) manufacturing facility.

The New Bedford Business Park was established in 1960 and currently consists of more than 40 businesses, including, but not limited to, a solar energy facility, a machine shop, a steel fabricator, print shops, and manufacturers of thin-film photovoltaic material, golf balls, electric capacitors, plastics, and circuit boards (Davis 2003; Cohen 2008; Brown 2011; GNBIF 2011). A number of properties within the New Bedford Business Park have had releases of oil or other hazardous material that have been reported to the Massachusetts Department of Environmental Protection (MDEP) under the statewide hazardous waste site cleanup program. This program, referred to as the Massachusetts Contingency Plan (MCP), was established in 1983 under Chapter 21E of Massachusetts General Laws (M.G.L. c21E, 310 CRM 40.0000). It authorizes the MDEP to enforce regulations governing the investigation and cleanup of oil and hazardous material release sites, known as "21E sites." Releases can vary widely with respect to the source, materials involved and the amount released, and the geographic extent of contamination. Many of the releases that occurred within the New Bedford Business Park were addressed with an immediate action response and, soon thereafter, were considered by the MDEP to pose no significant risk of harm to health, safety, public welfare and the environment.

[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.

At the time that this evaluation was initiated, three properties within the New Bedford Business Park had a release of oil or hazardous material that was currently being investigated under the MCP and cleanup activities were ongoing: the former BorgWarner TorqTransfer Systems, Inc. facility (BorgWarner), the former Polymerine site and the former Tallyrand site. Furthermore, two releases have occurred at the former Polaroid facility (MDEP 2009). Although they have been closed out under the MCP, the releases at the former Polaroid facility were included in this investigation since this facility was of specific interest to community members.

To address community concerns about possible environmental exposures, the CAP reviewed available environmental data for the sites of the former BorgWarner, Polymerine, Tallyrand, and Polaroid facilities. In addition, the CAP considered potential ways that people may come into contact with contaminants in soil, surface water, sediment, groundwater, and indoor and outdoor air associated with these sites. The available environmental data were compared to health-based screening values to determine whether there may be potential health impacts to nearby residents.

The U.S. Census Bureau subdivides the City of New Bedford into 31 smaller geographic areas known as census tracts (CTs). To address community concerns about cancer incidence, the CAP reviewed the incidence of six cancer types (bladder, breast, kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate cancer) in the two CTs (6501.01 and 6501.02) in which the neighborhoods of concern are located (see Figure 2). The six cancer types evaluated were chosen based on residents' concerns over suspected elevations. The incidence of each of these cancer types was compared to the incidence in the state of Massachusetts as a whole. Cancer incidence data for New Bedford were obtained from the Massachusetts Cancer Registry (MCR). At the time of this evaluation, the most recent and complete cancer incidence data available from the MCR were through the year 2005. Cancer incidence data reported for the years 1996 through 2005 were evaluated. Additionally, available information about known or suspected risk factors for developing these cancers was evaluated.

III. Objectives

The specific objectives of this investigation were as follows:

- To evaluate the extent to which contamination and/or emissions related to the former BorgWarner, Polymerine, Tallyrand and Polaroid facilities at the New Bedford Business Park could result in exposure to people in the area and whether adverse health effects might be possible, if exposure occurred.

- To discuss possible exposures related to contamination and/or emissions related to the former BorgWarner, Polymerine, Tallyrand and Polaroid facilities in the context of the available scientific and medical literature on cancer to determine whether further investigation or public health actions may be warranted.

- To review the incidence of six cancer types (bladder, breast, kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate cancer) in the two CTs (6501.01 and 6501.02) containing the neighborhoods of concern.

- To evaluate the geographic distribution of individuals diagnosed with these six cancer types in CTs 6501.01 and 6501.02 to see if there are any unusual spatial patterns.

- To review descriptive information available from the MCR for individuals diagnosed with these six cancer types in CTs 6501.01 and 6501.02 to see if there are any unusual patterns related to known or suspected risk factors for developing these diseases.

IV. Evaluation of Potential Community Exposure Pathways and Health Concerns

This evaluation was initiated in response to community concerns about cancer in two neighborhoods in northern New Bedford and possible environmental exposures from the nearby New Bedford Business Park (see Figure 1). In particular, community members were concerned about possible dumping, possible groundwater and surface water contamination, and possible impacts of air emissions from the Polaroid facility.

In order to address concerns of possible dumping prior to the construction of the Briarwood Estates development, the CAP contacted the New Bedford Department of Environmental

Stewardship (NBDES). Staff in NBDES reported that no documentation could be located that indicated any dumping had occurred. In addition, no record of a cease and desist order, which reportedly had been placed on the developer during construction, could be found (S. Alfonse, New Bedford Department of Environmental Stewardship, personal communication, 2008).

At the initiation of this evaluation, three properties within the New Bedford Business Park had a reported release of oil or hazardous material that was being investigated under the MCP and cleanup activities were still underway: the former BorgWarner, Polymerine and Tallyrand sites. The former Polaroid facility has had two releases, both of which were remediated and have been closed out under the MCP (MDEP 2009). These releases were included in this investigation because community residents were specifically concerned about the former Polaroid facility.

The release at the former BorgWarner facility (Release Tracking Number 4-0389), located at 200 Theodore Rice Boulevard, was reported to the MDEP in 1987 and involved petroleum contamination in groundwater and soil due to an underground storage tank (UST). Possible sources of petroleum also included metal parts drainage bins and underground piping associated with former USTs, all of which were removed from the site. Approximately 2,500 cubic yards of soils heavily impacted by petroleum were removed during remedial activities in 1987 (M&A 1999). A dual-phase product recovery/groundwater treatment system was installed in 2000 to recover subsurface light non-aqueous phase liquid (LNAPL). In 2006, the system was shut down for modification and began re-operating in September 2010 as a total fluid recovery system that discharges treated groundwater to the New Bedford Publicly Owned Treatment Works (POTW) via a sewer. In the interim, absorbent media was deployed in the recovery wells to continue product recovery. It should be noted that polychlorinated biphenyls (PCBs) were detected in LNAPL at the site but all groundwater monitoring results for PCBs have been non-detect. Currently, the release is being regulated under the MCP and has a Remedy Operation Status (REMOPS), indicating that a remedial system is being operated for the purpose of achieving a permanent solution (M&A 2010; MDEP 2008, 2009). Contamination identified at the site is reportedly completely below grade and limited to within the boundaries of the property. Therefore, residents of nearby neighborhoods or trespassers who may visit the Business Park are not expected to have direct contact with contaminated soil or groundwater. In addition, the

surrounding neighborhoods are generally served by the City of New Bedford Water Department, which obtains its drinking water from surface water bodies (M&A 1999). While it is possible that private drinking water wells may exist on Braley Road, they are located upgradient of the former BorgWarner site. The groundwater beneath the site flows west/southwest toward Duchaine Boulevard (B. Sylvia, New Bedford Department of Inspectional Services, personal communication, 2009; New Bedford Department of Public Infrastructure, personal communication, 2009; M&A 1999). The groundwater beneath the site has also been identified by the MDEP as a Non-Potential Drinking Water Source area. Due to the distance from the site to the nearest residences, exposure of nearby residents to contaminants via vapor intrusion (i.e., the volatilization of chemicals from groundwater through soil and into the indoor air of a building) would not have been expected in the past, nor in the present or future (M&A 1999; ITRC 2007; USEPA 2002). As a result, there are no completed soil or groundwater exposure pathways for nearby residents and this release was not evaluated further.

With respect to the former Polymerine, Tallyrand and Polaroid sites, the CAP contacted the MDEP to obtain available environmental information pertaining to these three sites. Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify those substances that may need to be considered for further analysis to determine potential health impacts to residents. The screening analysis identified maximum concentrations of contaminants detected in various environmental media (i.e., soil and water) and compared those concentrations to health-based comparison values. Comparison values are set well below levels that are known or anticipated to result in adverse health effects. Contaminant concentrations that exceed comparison values will not necessarily affect one's health. For a contaminant 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 concentration of a contaminant is greater than the appropriate comparison value, the potential for exposure to the contaminant should be further evaluated to determine whether exposure is occurring and whether health effects might be possible as a result of that exposure. An evaluation of potential exposure pathways was conducted to determine whether contamination identified at the sites could impact the health of nearby residents of New Bedford in the past, present, or future.

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

A. Former Polymerine Site

Site Description

The former Polymerine site is located at 241 Duchaine Boulevard within the New Bedford Business Park in New Bedford. The property is currently owned by the City of New Bedford and was acquired through a tax taking in 2006. The 8-acre property consists of a single-story manufacturing building with 33,757 square feet that was built circa 1960, an asphalt-paved parking area, landscaped areas, undeveloped wooded and wetland areas, and an abandoned railroad spur (New Bedford 2008a). A portion of the on-site building is currently leased by New England Plastics Corporation (NEP) and operates as a plastics manufacturing facility. The remaining portion of the building is vacant. Until recently, it had been used as a warehouse by Aquapoint, Inc., a wastewater treatment system manufacturer (Roy F. Weston 1998; Tighe & Bond 2008).

The property is bordered to the east by Duchaine Boulevard and Alberox Corporation (also known as Morgan Advanced Ceramics), to the south by Black Pond and the Titleist Acushnet Company Ball Plant III, to the west by Hobomock Swamp and Conrail Railroad tracks, and to the north by undeveloped woods and wetlands (Roy F. Weston 1998; Tighe & Bond 2008). The nearest residence is located approximately 1,300 feet (0.25 miles) to the northeast along Braley Road (MassGIS 2005).

Site History

From 1960 through the late 1990s, the property was operated by Polymerine (formerly known as Polyply) as a manufacturing facility of composite fiberglass boards produced through a process that laminated sheets of fiberglass impregnated with epoxy. Hazardous materials used or generated by manufacturing processes included solvents, resins, waste oil, fuel oil, and PCB-

containing heat transfer oil (Roy F. Weston 1998, 2001). Hazardous waste generated at the former Polymerine site included 2-propanone and acetone, both of which are ignitable wastes. Two violations were cited at the facility but both were subsequently resolved (Tighe & Bond 2008).

In September 1993, the MDEP was notified of a release of oil or hazardous substances at the Polymerine site and assigned the Release Tracking Number (RTN) 4-0001347 (MDEP 2009). Based on a review of available environmental data for the former Polymerine site, assessment activities included the collection of groundwater, soil, sediment and interior building surface samples. The maximum levels of contaminants detected at the site were compared to health-based comparison values to determine if further evaluation was necessary.

Groundwater samples contained acetone, toluene, phenanthrene, PCBs and zinc at concentrations below applicable MDEP Method 1 GW-1 standards (MDEP 2008,; MassGIS 2008; Tighe & Bond 2008, 2009). PCBs were detected in wipe samples collected from interior building surfaces with concentrations exceeding the Toxic Substance Control Act (TSCA) High Occupancy Standard of 10 micrograms per 100 square centimeters (µg/100 cm^2) (USEPA 1991; USEPA 2005; Roy F. Weston 1998; Tighe & Bond 2008). Sediment samples collected from the abutting wetland contained concentrations of PCBs that exceeded soil comparison values (ATSDR 2008a; Tighe & Bond 2009). Soil samples collected on-site contained elevated concentrations of PCBs and the metal copper that exceeded comparison values[2] (ATSDR 2008a; Roy F. Weston 1998; Tighe & Bond 2008). See Table 1 for a summary of the maximum concentrations of contaminants detected in on-site soil and sediment samples that exceeded comparison values.

[2] It has been reported that an on-site soil sample collected in 1993 contained elevated concentrations of total petroleum hydrocarbons (TPH) that exceeded soil comparison values (24,000 ppm). However, documentation of this sample with its location and depth could not be located (Tighe & Bond 2008). In addition, data on the constituent composition of the TPH detected on-site were not available. TPH is a mixture of many different compounds, all of which originate from crude oil, and affect the body in different ways. Although there are no federal regulations or guidelines for TPH as a single entity, the government has developed regulations and guidelines for some of the specific TPH fractions and compounds. Due to the lack of documentation and appropriate health-based comparison values, TPH was not evaluated further.

In 1998, the property owner excavated approximately 220 tons of PCB-contaminated soil from the site in response to a Unilateral Administrative Order (UAO) from the U.S. Environmental Protection Agency (USEPA). The excavated soil was stockpiled on the property until it was disposed of in 2000. From 2000 through 2001, the USEPA conducted removal activities at the site, excavating and disposing of approximately 2,000 tons of PCB-contaminated soil off-site. Excavation of soil with PCB concentrations exceeding the site-specific USEPA action level of 2 ppm was conducted and continued to depths where PCB concentrations were less than 2 ppm or until the water table was encountered. The excavation was generally continued to depths between 12 and 30 inches below ground surface with some areas excavated to depths of between 60 and 72 inches below ground surface. Several areas of soil with PCB concentrations exceeding the USEPA action level located at or below the water table were covered with geotextile fabric and a layer of clean soil. Site restoration consisted of backfilling and grading soil as well as planting vegetation and trees (Roy F. Weston 2001). Post-excavation confirmatory soil samples were collected in 2000, 2001 and 2008. Confirmatory soil samples contained concentrations of PCBs (e.g., maximum of 2,600 ppm) that still exceeded soil comparison values (Tighe & Bond 2009). Since the removal actions were focused on PCB contamination, concentrations of copper remaining in on-site soil were not measured. Presumably, the concentration of copper has been reduced by the soil excavation since the contaminants were co-located.

Site Status

The former Polymerine site is currently classified by the MDEP as a Tier II disposal site under the MCP. The City of New Bedford has assumed the role of Responsible Party (RP) and has agreed to initiate voluntary Comprehensive Response Actions (CRAs) as required by the MCP and TSCA. To date, a Phase I Initial Site Investigation, Tier Classification Report, and a Phase II Comprehensive Site Assessment (CSA) have been submitted to the MDEP (Tighe & Bond 2008, 2009).

Groundwater within the western portion of the former Polymerine site is located within an area designated by MDEP as a potentially productive aquifer. However, groundwater samples collected in this area did not contain contaminants above the applicable MDEP Method 1 GW-1

standards (MDEP 2008; MassGIS 2008; Tighe & Bond 2008, 2009). It is important to note that groundwater at the former Polymerine site is not used for drinking water. The on-site building and the surrounding neighborhoods are served by the City of New Bedford Water Department, which obtains its drinking water from surface water bodies. Testing of the drinking water is conducted on a regular basis, as is required of all municipal drinking water sources. No private drinking water wells are located within 500 feet of the site (Tighe & Bond 2009). As a result, there is no completed groundwater exposure pathway and, hence, data were not evaluated for possible exposures through household water use.

Although the groundwater at the former Polymerine site could be a potential source of indoor air exposures in the on-site building (a private business) since the average depth to groundwater is less than 15 feet below ground surface and the release area is located within 30 feet of an occupied structure, vapor intrusion was eliminated as an exposure pathway for nearby residents in the past, present and future due to the distance to the nearest residences. Therefore, vapor intrusion as a potential exposure pathway at the former Polymerine site was not evaluated further in this health consultation (Tighe & Bond 2009; ITRC 2007; USEPA 2002).

Prior to remediation in 2001, it is possible that a trespasser, such as an older child, may have been exposed through incidental ingestion of or dermal contact with PCBs and copper detected in on-site surface soil and sediment at levels above comparison values. However, it is important to consider that comparison values are based on a residential exposure scenario, and it is unlikely that a trespasser would have had contact with on-site surface soil and sediment for a comparable frequency and duration of time as a resident, i.e., daily over a lifetime. The site is located within an industrial area and, hence, trespassing would occur only occasionally, if at all.

Although unlikely, assuming that an older child trespassed on the site and inadvertently ingested 200 milligrams of surface soil containing the maximum concentration of copper detected on-site (8,300 ppm) for 1 day every week for 22 weeks (May through September, the warmer months of the year) over 5 years, the predicted exposure dose 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 exposure dose for an older child

playing on-site, even under highly conservative exposure assumptions, is below the MRL, noncancer health effects would not be expected. See Appendix A for more information on the exposure dose calculations.

The maximum detected concentration of PCBs for which documentation is available is 17,000 ppm[3]. This concentration is the average of two duplicate samples (19,000 ppm and 15,000 ppm) collected at a sampling station located near the northwestern corner of the on-site building (Paragon 1998). It is very unlikely, however, that a trespasser would have had consistent contact with soil containing the highest concentration of PCBs at this particular location. Rather, it is more likely that a trespasser at the former Polymerine site would contact surface soil with a range of PCB concentrations based on the distribution of contaminated soil within an area encompassing roughly 30,000 square feet located northwest, west and south of the on-site building (Figures 3 and 4). A more reasonable and realistic scenario uses the average concentration of PCBs detected in surface soil in this area to reflect the range of contaminant concentrations that could have been contacted over time. Assuming that an older child who trespassed regularly on the site could have been exposed to the average concentration of PCBs detected in surface soil (403 ppm) in this area through incidental ingestion, the estimated noncancer effects exposure dose (0.0001 mg/kg/day) is greater than the ATSDR Chronic MRL. The chronic MRL is based on adverse noncancer health effects observed in studies of monkeys that were exposed to 0.005 mg/kg/day (ATSDR 2008d). Because the estimated noncancer effects exposure dose for an older child who trespassed in the above scenario is 50 times lower than the lowest exposure dose that resulted in adverse health effects in animal studies, noncancer health effects from past exposure to PCBs at the former Polymerine site are not expected even under the highly unlikely assumption that an individual frequently visited and/or played on the site itself.

In order to evaluate the potential for carcinogenic health effects, exposure doses were estimated and compared to health guideline values for cancer. The USEPA and the International Agency

[3] According to several reports, an environmental site investigation conducted in 1993 detected a maximum concentration of PCBs in on-site soil and sediment of 49,000 ppm. However, no documentation of this sample with its location and depth (surface or subsurface) could be located (Tighe and Bond 2008).

for Research on Cancer (IARC) have classified PCBs as a probable human carcinogen based on sufficient evidence of carcinogenicity in animals and limited evidence in humans (ATSDR 2008d). Because it is difficult to show that a chemical causes cancer in humans, animal studies are used to identify chemicals that have the potential to cause cancer in humans. PCBs do cause cancer in animals. Thus, it is assumed that exposure to PCBs over a period of time might pose a health risk for humans. The degree of risk depends on the intensity and frequency of exposure.

Under similar and highly unlikely assumptions as for the above noncancer health effects, an unusual cancer risk would not be expected. See Appendix A for more information on the exposure dose and cancer risk calculations for exposure via incidental ingestion. Exposure of trespassers to contaminated subsurface soil is not expected due to its depth below ground surface.

Under the same exposure conditions and assumptions described above, dermal exposure to the average concentration of PCBs detected in the contaminated section of the site results in an estimated noncancer effects exposure dose (0.00006 mg/kg/day) that is greater than the ATSDR Chronic MRL. This indicates that it is possible that a trespassing older child could have been exposed via dermal contact to PCBs at a level that could have presented an increased risk of adverse noncancer health effects. However, the estimated dermal exposure dose is more than 80 times lower than the lowest exposure dose that resulted in adverse noncancer health effects in animal studies. Therefore, noncancer health effects from past dermal exposure to PCBs at the former Polymerine site are not expected. With regard to the potential for carcinogenic health effects, dermal exposure to the average concentration of PCBs detected in the contaminated section of the site under the same exposure conditions and assumptions as above is not expected to present an unusual cancer risk. See Appendix B for more information on the exposure dose and cancer risk calculations for exposure via dermal contact.

Although removal activities have been conducted at the former Polymerine site, concentrations of PCBs detected in surface soil still exceed the MDEP Method 1 standard of 2 ppm for category S-1 & GW-1 soil as evidenced by post-excavation confirmatory sampling. As a result, additional remedial actions at the former Polymerine site are required in accordance with the MCP as

specified in the Phase II CSA (Tighe & Bond 2009). Furthermore, present and future ingestion of and dermal contact with contaminants in on-site surface soil by trespassers remain possible exposure pathways. These exceedances are limited to two small, isolated areas located on the north and south sides of the on-site building (Figure 5). The average concentration of PCBs in surface soil samples collected north of the building in an area encompassing roughly 400 square feet is 440 ppm (maximum concentration of 2,600 ppm) whereas the average concentration of PCBs in samples collected south of the building in an area encompassing about 200 square feet is 50 ppm (maximum concentration of 150 ppm) (Tighe & Bond 2009). These concentrations are similar to or less than those used above to estimate possible exposure doses. Hence, based on that evaluation, health concerns would not be expected under current and future use conditions (i.e., as an industrial park). See Appendices A and B for more information on the exposure dose and cancer risk calculations.

B. Former Tallyrand Site

Site Description

The former Tallyrand site is located at 129 John Vertente Boulevard within the New Bedford Business Park in New Bedford. The property is currently owned by the City of New Bedford (New Bedford 2008b; Roy F. Weston 1997). Aerovox Corporation leases a portion of the 79-acre site and uses the 136,500 square foot manufacturing building to produce AC film capacitors, which are used in applications such as electric motors, fluorescent light ballasts, and microwave ovens. The remaining section of the property is currently vacant (Aerovox 2008; New Bedford 2008b).

The property is bordered to the north and west by Hobomock Swamp, to the east by Conrail Railroad tracks and Hobomock Swamp, and to the south by John Vertente Boulevard and Hobomock Swamp (SITEC Environmental 1996; Roy F. Weston 1997). The nearest residence is located approximately 1,400 feet (0.27 miles) to the west along Demoranville Road in Dartmouth (MassGIS 2005).

Site History

The site was originally owned by the Acushnet Saw Mill Co. from 1895 to 1969. Since then, it has changed ownership several times. From 1969 to 1973, the site was owned by the New Bedford Industrial Foundation. From 1973 to 1984, all of the corporations listed as either owners or lessees were involved in the manufacturing of polyvinyl chloride (PVC) pipe. Between 1978 and 1987, the site was owned by Tallyrand Chemicals, Inc. and Imex Polymers, Inc. In 1992, the City of New Bedford acquired the property through foreclosure. The site had been abandoned for several years and repeatedly vandalized. Buildings associated with the former Tallyrand operations have since been demolished and a new building was constructed in 1999 to accommodate Aerovox Corporation (New Bedford 2008b; VHB 1999a; Roy F. Weston 1997).

The following releases of oil and/or hazardous material have occurred at the former Tallyrand site and were reported to the MDEP under the MCP:

- Release of transformer oil at an electrical transformer and pad in 1995 (RTN 4-0011419)
- Release adjacent to the former mixing basin in 1999 (RTN 4-0014594)
- Release beneath the former reactor building in 1999 (RTN 4-0014633)

The release of transformer oil in 1995 occurred at the southern boundary of the former Tallyrand site and was likely a result of vandalism. Consequently, the exact time of its occurrence is uncertain. A six foot chain link security fence was installed to eliminate any further potential exposure via trespassing (SITEC Environmental 1996).

Based on a review of available environmental data that was collected in response to the release of transformer oil, elevated concentrations of TPH and PCBs (primarily Aroclor-1260) that exceeded regulatory standards were detected in on-site soil in 1995 (MDEP 2008; SITEC Environmental 1996; Roy F. Weston 1997). Exposure to contaminated subsurface soil would not be expected due to its depth below ground surface. In addition, PCBs were found to have migrated along the surface of the shallow water table in an east-northeast direction (Roy F. Weston 1997). See Table 2 for a summary of the maximum concentrations of contaminants detected in on-site soil that exceeded comparison values.

In October 1997, the USEPA completed a limited removal action at the site, excavating PCB-contaminated soil and disposing of it in a hazardous waste landfill. Excavation was conducted in areas of soil with total PCB concentrations exceeding the site-specific USEPA action level of 10 ppm. Excavation was continued to depths where PCB concentrations were less than the action level or until the water table was encountered (approximately 4.5 feet below the ground surface). Excavated areas were backfilled with clean fill (Roy F. Weston 1997). As a result of the removal action, concentrations of PCBs in surface soil (0-3 inches) are expected to meet the USEPA action level of 10 ppm. They may, however, exceed the ATSDR CREG of 0.4 ppm (ATSDR 2008a). PCB concentrations of up to 4,500 ppm remain on-site in subsurface soil at depths of 7 feet or greater or below the water table in a small portion of the site (Roy F. Weston 1997). Since the USEPA removal action focused on PCB contamination only, concentrations of TPH remaining in on-site soil are unknown but are expected to have been reduced. In August 2008, the MDEP determined that no further action was required for this release site (MDEP 2009).

Although the two releases that were reported to the MDEP in 1999 were separate and discrete, both involved creolin (a coal tar derivative). Elevated concentrations of semi-volatile organic compounds (SVOCs) and TPH were detected in subsurface soil at the former mixing basin release area and elevated concentrations of TPH were detected in subsurface soil at the former reactor building release area. The maximum concentrations of the SVOC 2-methylnapthalene (3,700 mg/kg) and TPH (20,000 mg/kg) detected at the former mixing basin release site exceeded MDEP Method 1 standards for category S-1 & GW-1 soil. The maximum concentration of TPH (2,100 mg/kg) detected at the former reactor building release site also exceeded MDEP Method 1 standards for category S-1 & GW-1 soil (VHB 1999a). Since all elevated concentrations were detected in subsurface soil (3-5 feet below ground surface), exposure to contaminants from either of these releases is not expected to occur either in the past, present or future due to its depth below the ground surface.

A total of approximately 170 cubic yards of creolin-impacted soil was excavated from the mixing basin release site and approximately 50 cubic yards was excavated at the reactor building release site. All excavated soil was disposed off-site (VHB 1999a,b). As a result of the

excavation, contaminant concentrations in both areas are expected to meet MDEP soil standards. Furthermore, Class A-2 Response Action Outcomes (RAOs) were issued by the MDEP in 2005 for both RTNs (VHB 1999a). This indicates that remedial work was completed at the former mixing basin and former reactor building, a permanent solution has been achieved and, although contamination has not yet been reduced to background levels, a level of "no significant risk" of harm to health, safety, public welfare and the environment has been achieved for these two areas (MDEP 2008).

Site Status

Prior to the completion of remediation in 1997 of contaminated soil from the release of transformer oil (RTN 4-0011419), it is possible that a trespasser at the former Tallyrand site may have been exposed through incidental ingestion of or dermal contact with PCBs (mainly Arcolor-1242 and Aroclor-1260) and TPH detected in on-site soil at levels above comparison values[4]. However, it is important to consider that comparison values are based on a residential exposure scenario, and it is unlikely that a trespasser would have had contact with contaminated soil for a frequency and duration of time comparable to a resident. It is particularly unlikely for several reasons. First, the area that was contaminated consisted of a very small, isolated portion of the property, encompassing approximately 40 feet by 24 feet of the 79-acre property (0.03%) (Figure 6). Second, the former Tallyrand facility is centrally located within the New Bedford Business Park. Third, access to the former Tallyrand site is generally limited to the road as it is bordered by Hobomock Swamp in all directions with the nearest residence approximately one-third of a mile away. Observations made by the MDPH (i.e., CAP staff) during a visit to the former Tallyrand site confirmed these conditions.

Although highly unlikely, the potential exposure was estimated for an older child who may have inadvertently ingested surface soil containing the average concentration of total PCBs detected in on-site soil (0-6 inches) in the area of contamination (887 ppm) for 1 day every month for 5 months (May through September, the warmer months of the year) over 5 years. The estimated noncancer effects exposure dose (0.00005 mg/kg/day) is greater than the ATSDR Chronic MRL

[4] Due to the lack of appropriate health-based comparison values, TPH was not evaluated further.

(0.00002 mg/kg/day). As previously noted, the chronic MRL for PCBs is based on adverse noncancer health effects observed in studies of monkeys that were exposed to 0.005 mg/kg/day (ATSDR 2008d). Because the estimated noncancer effects exposure dose for an older child who trespassed in the above scenario is 100 times lower than the lowest exposure dose that resulted in adverse noncancer health effects in animal studies, noncancer health effects from past exposure to PCBs at the former Tallyrand site are not expected. In addition, using the same exposure assumptions, an unusual cancer risk would not have been expected. See Appendix C for more information on the exposure dose and cancer risk calculations. As previously stated, exposure of trespassers to contaminated subsurface soil would not be expected due to its depth below ground surface.

Under the same exposure conditions and assumptions described above, dermal exposure to the average concentration of total PCBs detected in on-site surface soil in the contaminated area (887 ppm) results in an estimated noncancer effects exposure dose (0.00003 mg/kg/day) that is greater than the ATSDR Chronic MRL. However, the estimated noncancer effects (dermal exposure) dose for an older child who trespassed and contacted the average concentration of total PCBs detected on-site in the above scenario is more than 160 times lower than the lowest exposure dose that resulted in adverse health effects in animal studies. Therefore, noncancer health effects from dermal exposure to surface soil containing PCBs prior to remediation at the former Tallyrand site would not have been expected. With regard to the potential for carcinogenic health effects, dermal exposure to the average concentration of total PCBs detected in on-site surface soil under the same exposure conditions and assumptions as above would not be expected to present an unusual cancer risk. See Appendix D for more information on the exposure dose and cancer risk calculations.

As a result of removal activities, concentrations of PCBs in surface soil are not expected to exceed the USEPA action level of 10 ppm. However, it is possible that PCB concentrations could still exceed health-based comparison values. Under the same exposure conditions and assumptions described above, present and future ingestion of and dermal contact with PCBs in on-site surface soil by trespassers are not expected to result in adverse non-cancer or cancer health effects. See Appendices C and D for more information on the exposure dose and cancer

risk calculations for exposure via ingestion and dermal contact, respectively. In addition, the possibility that trespassers could come into contact with contaminated surface soil has been minimized since access to the former Tallyrand site is now restricted by a chain link security fence and monitored by closed circuit TV. Exposure of trespassers to contaminated subsurface soil remaining at the site would not be expected due to its depth below ground surface.

It should be noted that there is no completed groundwater exposure pathway for any of the three releases that occurred at the former Tallyrand site because groundwater at the site is not used for drinking water. The on-site building and the surrounding neighborhoods are served by the City of New Bedford Water Department (VHB 1999a).

Although groundwater at the former Tallyrand site could be a potential source of vapor intrusion for the on-site building (a private business), this potential exposure pathway was eliminated for nearby residents in the past, present and future due to the distance to the nearest residences (VHB 199a; ITRC 2007; USEPA 2002). Therefore, vapor intrusion as a potential exposure pathway was not evaluated further in this health consultation.

C. Former Polaroid Site

Site Description

The former Polaroid site is located at 100 Duchaine Boulevard within the New Bedford Business Park in New Bedford. In January 2008, the property was acquired by Konarka Technologies Inc. for the manufacture of thin-film photovoltaic material (Cohen 2008). The 128-acre property includes manufacturing buildings, a power plant, a wastewater treatment plant (WWTP), asphalt parking areas, undeveloped wooded and wetland areas, and a pond (GEI 2001; New Bedford 2008c).

The site is bordered to the north by commercial and industrial properties, to the east by a residential community referred to as Pine Hill Acres, to the west by Conrail Railroad tracks, and to the west and south by the Acushnet Cedar Swamp State Reservation. The nearest residence to the property is located approximately 275 feet to the east along Ridgewood Road (MassGIS 2005; Roux Associates 2004).

Site History

Prior to construction of the Polaroid facility in 1969-1970, the land consisted of undeveloped farmland and wetlands (Roux Associates 2004). Polaroid owned the property and operated a high resolution media manufacturing division and a photographic negative manufacturing division until 2006, when MultiLayer Coating Technologies, LLC (formerly Polaroid Contract Coating) was established. During this time, the on-site WWTP served all buildings on the property and treated the wastewater generated facility-wide. The on-site power plant supplied the facility with steam and cooling water (Watermill Group 2006; GEI 2002).

In November 1993, the MDEP was notified of a reportable release or threat of release of fuel oil #6 and TPH from an underground storage tank. The RTN 4-0010113 was assigned. In January 1994, a Class B-1 RAO was issued for this release, indicating that remedial actions were not conducted because a level of "no significant risk" of harm to health, safety, public welfare or the environment already existed. Additionally, no "activity and use limitation" on the property was necessary to ensure the existence or maintenance of this level (MDEP 2009, 2008a). Thus, no further analysis of past, present or future exposures related to this release site is required.

In June 2001, MDEP was notified of a release of sulfuric acid from an above-ground storage tank in the vicinity of the WWTP on the western side of the property. The RTN 4-0016316 was assigned (MDEP 2009). An Immediate Response Action Completion Report, Phase I Initial Site Assessment Report, Phase II Comprehensive Site Assessment and Phase III Comprehensive Remedial Action Alternatives Report, and a Phase IV Remedy Implementation Plan have been completed for this site (Roux Associates 2006). The sulfuric acid impacted soil within an area encompassing approximately 1,600 square feet by reducing the pH to less than 4.0 and to less than 2.0 in some smaller areas. Soil contamination was detected at depths ranging from two to ten feet below ground surface. The pH of groundwater in the immediate down-gradient direction was reduced as a result of leaching of sulfuric acid from affected soil by rainwater in the unpaved portions of the site (Roux Associates 2004). Risk characterization conducted as part of the Phase II and Phase III found no impacts to the surface water and sediment in the wetlands that border the WWTP on all four sides (Roux Associates 2005). Remedial activities, including

excavation and disposal of 347 tons of contaminated soil, were implemented in 2005. Sampling confirmed that the pH of the soil remaining at the site is generally within the range of 4 to 7, which is considered background for the area. In 2006, a Class A-1 RAO was issued for this release, indicating that remedial work was completed, a permanent solution was achieved, the source of contamination was adequately removed allowing for concentrations to approach background, and a level of "no significant risk" of harm to health, safety, public welfare, and the environment exists currently and in the foreseeable future (Roux Associates 2006).

Polaroid and MultiLayer Coating Technologies were both classified as a Large Quantity Generator (LQG) under RCRA (Generator Identification MAD058060476). LQG facilities generate over 1,000 kg of hazardous waste per month (Roux Associates 2004). Hazardous waste generated at the facility included methanol, silver compounds, and nitrate compounds (USEPA 2008). An inspection conducted by the MDEP in 1997 cited a violation whereby storage tanks containing hazardous waste lacked secondary containment (MDEP 1997). However, an inspection conducted more recently in 2002 found no violations relative to the management of hazardous waste (MDEP 2002b).

A Draft Air Quality Operating Permit (Application Number 4V95157) was issued by the MDEP for the Polaroid facility in 2004. The permit identifies equipment and processes that produce air emissions as well as any pollution control devices that are in place. Criteria pollutants for which emission limits and restrictions are set in the permit include nitrogen oxides (NO_x), carbon monoxide (CO), particulate matter (PM), sulfur dioxide (SO_2), sulfur in fuel, and VOCs. Additional requirements in the permit include monitoring, testing, and recordkeeping as well as a special term and condition, which states that the facility is subject to Subpart JJJJ of 40 CFR 63 since it is a major stationary source of Hazardous Air Pollutants (HAPs) (MDEP 2004a). This subpart establishes emission standards specifically for paper and other web coating lines and involves technology-based standards that use maximum achievable control technologies.

A review of data reported in the USEPA's Toxics Release Inventory (TRI) was conducted to gain additional information regarding the types and amounts of pollutants emitted from the facility, particularly HAPs. The TRI is a reporting system that estimates the annual releases of

toxic chemicals to the environment. The system evolved from the Emergency Planning and Community Right-to-Know Act. Businesses are required to report the locations and quantities of chemicals stored on a site to state and local agencies to help communities prepare to respond to potential chemical spills and emergency releases (USEPA 2008). Although TRI annual release estimates cannot be used to specifically evaluate whether individuals living near the former Polaroid facility were actually at risk of exposure to air emissions, the information can be helpful when evaluating the pattern of disease in the nearby area and the possibility that environmental factors may play a role.

Review of available TRI data for the former Polaroid facility for the years 1988-2006 indicates that methanol, methyl ethyl ketone (MEK, also known as 2-butanone) and hydrochloric acid have been emitted to air from the facility. All three are classified as HAPs. None have been classified with respect to carcinogenicity (USEPA 2000a,b; ATSDR 2008c). The major health concerns if long-term exposure to methanol were to occur would include headaches, dizziness, insomnia, nausea, gastric disturbances, conjunctivitis, and blurred vision (USEPA 2000a). Limited information is available on the health effects from long-term exposure to MEK but may include mild respiratory irritation of the nose and throat. It should be noted that MEK has a sharp, but sweet odor that may be smelled even at low concentrations (USEPA 2000b; ATSDR 1992). If long-term exposure to hydrochloric acid were to occur, major health concerns may include gastritis, chronic bronchitis, dermatitis, and photosensitization (USEPA 2000c). It should be noted that when such health effects do occur, it is typically in an occupational setting where the concentration level would be higher and the duration of exposure longer than would be expected for nearby residents.

Stack emissions of methanol were highest in the late 1980s and early 1990s. The maximum stack emission of methanol (44,900 pounds) was reported in 1990. Annual stack emissions of methanol have generally decreased since then, with 12,000 pounds released in 2006. Fugitive emissions (emissions from sources other than stacks or vents, such as equipment leaks and evaporative losses) of methanol have generally increased from a minimum annual emission of 100 pounds from 1989 to 1993 to 1,800 pounds in 2004, with a spike of 4,700 pounds in 2005. MEK was last released from the Polaroid facility in 2003, at which time fugitive emissions were

3,900 pounds and stack emissions were 1,400 pounds. Hydrochloric acid was also reported historically, but not since 1995 (see Table 3).

Since 1990, air emission violations were cited on two occasions at the Polaroid facility. One violation in 1990 was observed based on the density and opacity of the smoke emitted from a stack and the other violation in 2004 was observed based on soot fallout in the Pine Hill Acres neighborhood. Reportedly, the cause of both of these violations was promptly identified by Polaroid and corrective actions were taken (MDEP 1990, 2004b; Polaroid 1990, 2004). Air compliance evaluations that were conducted in 1997, 2002 and 2007 by the MDEP, Bureau of Waste Prevention found the facility to be in compliance based upon observation of visible emissions, on-site inspections, and facility records and logs (MDEP 1997, 2002b, 2007). It should be noted that no ambient air sampling data are available for the neighborhoods in the immediate vicinity of the facility.

Site Status

With regards to the release of sulfuric acid near the WWTP in 2001, no past, present or future exposure pathway is complete due to the depth below ground surface (2-10 feet) at which contamination was detected. Remedial activities included the excavation and disposal of contaminated soil. Exposure to impacted soil remaining at the site is not expected to occur due to its presence at depths greater than seven feet below ground surface. The MDEP considers the remediation of this release to be complete as indicated by the Class A-1 RAO that was issued (Roux Associates 2006).

The groundwater at the former Polaroid facility is not used for drinking water. The on-site building is served by the City of New Bedford Water Department. No private drinking water wells are located within 500 feet of the site (GEI 2002). As a result, there is no completed groundwater exposure pathway in the past, present or future.

Prior to the excavation and disposal of contaminated soil, it is possible that the indoor air of the WWTP building could have been affected by vapor intrusion of hydrogen sulfide or hydrogen gas which could be generated via reaction of sulfuric acid with organic materials and cast iron

piping, respectively (Roux Associates 2004). Therefore, the potential exists that on-site employees may have been exposed in the past to contaminants via inhalation of indoor air that was affected by vapor intrusion. However, exposure of nearby residents to contaminants via vapor intrusion is not expected in the past, present or future.

The results of the air compliance evaluations conducted at the former Polaroid facility indicate that the requirements and limitations set forth in the Draft Air Quality Operating Permit have been met. Although violations were cited on two occasions since 1990, corrective actions were promptly taken by Polaroid.

On the basis of the TRI data reviewed, past opportunities for exposure to ambient air emissions from the former Polaroid facility were a possibility for nearby residents when the facility was in operation. Stack and fugitive emissions of methanol may have resulted in exposure opportunities in ambient air. While MEK is no longer emitted at the facility, it is possible that stack and fugitive emissions may have resulted in potential exposure prior to 2004. Likewise, it is possible that stack emissions of hydrochloric acid may have resulted in potential exposure prior to 1996. Since there are no historical ambient air data available for the former Polaroid facility or the surrounding neighborhoods, it is difficult to evaluate whether facility emissions may have resulted in health impacts. As previously mentioned, it is primarily occupational exposure to these chemicals that have been attributed to adverse health effects. Such workplace settings typically involve concentration levels that would be higher and exposure durations that would be longer than those experienced by nearby residents. However, since MEK is a respiratory irritant, the MDPH examined available hospital discharge data for asthma in the area surrounding the former Polaroid facility to assess whether the number of asthma hospitalizations was occurring as expected based on the statewide experience.

Asthma is a chronic inflammatory pulmonary disorder that is characterized by reversible obstruction of the airways. Causes of asthma are unknown; however, episodes of asthma (asthma attacks) can be triggered by certain environmental pollutants such as air pollution, mold, pets/pet dander, and dust mites. A number of studies have reported links between being exposed to air pollution and asthma. The outdoor air pollutants most commonly linked to asthma attacks

are particulate matter and ozone. Reducing exposure to these pollutants can help prevent symptoms.

The Massachusetts Division of Health Care Finance and Policy (DHCFP) collects individual-level information on all patients who are discharged from hospitals in Massachusetts. The collection of data is mandated by regulation 114.1 CMR 17.00, Requirement for the Submission of Hospital Case Mix and Charge Data. Each hospitalization in Massachusetts is coded using the International Classification of Disease 9th Revision codes (ICD-9). The hospital discharge database was queried using the specific ICD-9 code for asthma, where asthma was the primary cause of hospitalization.

It is important to note that there are some limitations to the data on asthma hospitalizations. First, the data are only provided at the level of city/town or zip code. This makes it impossible to determine if one specific neighborhood has more asthma hospitalizations than another. As a result, an aggregate analysis of hospital discharge data was conducted at the zip code level. Second, the data reflect the numbers of hospitalizations reported, not the number of individuals hospitalized. For example, when the database is used to identify the number of asthma hospitalizations in a particular zip code during a specified time period, it would count one individual hospitalized ten times the same number of times as it would count ten individuals each hospitalized once. Third, the hospital discharge data are limited to inpatient hospitalizations with a minimum stay requirement. The data do not include individuals who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, as well as those treated in outpatient settings. It is important to keep this in mind when interpreting the asthma hospitalization data presented here.

The hospital discharge database was queried for asthma hospitalizations of residents of zip code 02745 in New Bedford, which encompasses approximately 10 square miles (Figure 7). These data were examined to determine how many times individuals were hospitalized with asthma each year during the four-year period of 2000 through 2003. This time period reflects the most

complete data available through 2003, after which point MEK was no longer emitted from the former Polaroid facility.

The statistic reported is the Standardized Hospitalization Ratio (SHR), which was calculated as the ratio of the number of hospitalizations observed in zip code 02745 to the number that would be expected if the population of the zip code had the same age-specific hospitalization rates as the statewide population, multiplied by 100. [Note that the SHR is not meaningful for the entire state; by definition the result is always 100.] An SHR of more than 100 indicates that the number of hospitalizations within the zip code was higher than expected compared to the statewide experience and an SHR of less than 100 indicates that the number of hospitalizations was less than expected compared to Massachusetts. A 95% confidence interval (CI) was calculated to determine if the observed number of hospitalizations is "statistically significantly different" from the expected number, meaning that there is less than a 5% percent chance that the observed difference (either increase or decrease) in the number of hospitalizations is the result of random fluctuation.

Table 4 provides a summary of the hospital discharge data for asthma hospitalizations in zip code 02745 for each year from 2000 to 2003 as well as the entire 4-year period. A total of 154 asthma hospitalizations were reported in zip code 02745 during 2000-2003 compared to approximately 133 expected based on the statewide experience; this difference was of borderline statistical significance (SHR = 115, 95% CI = 98-135). With the exception of one year (2000) during which the number of asthma hospitalizations was statistically significantly lower than expected, the number of asthma hospitalizations in any given year during 2000-2003 was elevated for residents of this zip code compared to the statewide experience. The elevations, however, were not statistically significant except during 2003 when 58 asthma hospitalizations were observed and about 39 were expected (SHR = 150, 95% CI = 114-194). Overall, there does not appear to be a relationship between the level of emissions of MEK from the former Polaroid facility and the number of asthma hospitalizations within zip code 02745 over the four-year period 2000-2003. The level of emissions of MEK generally decreased over this time period and a corresponding decrease in the number of asthma hospitalizations was not observed.

For more information regarding the air emissions at the former Polaroid facility, the Bureau of Waste Prevention at the MDEP Southeast Region may be contacted at 508-946-2700.

V. Methods for Analyzing Cancer Incidence

A. Case Identification/Definition

As part of this investigation, the CAP reviewed cancer incidence data available from the MCR for the following six cancer types in New Bedford CTs 6501.01 and 6501.02: cancers of the bladder, breast, kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate. Area residents requested that these specific types of cancer be evaluated. The 10-year period from 1996-2005 was evaluated and constituted the time period for which the most recent and complete cancer incidence data were available at the time of this report[5]. The MCR is a division within the MDPH Bureau of Health Information, Statistics, Research, and Evaluation (BHISRE). It is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain in situ (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111. s 111b). The MCR also gathers background information (e.g. gender, age, 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. Due to the high volume of data collected and the 6-month period between diagnosis and required reporting, the most current registry data that are complete will be a minimum of 2 years prior to the current date.

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

[5] The data summarized in this report are drawn from data entered into the MCR before March 6, 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.

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

It should be noted that the MCR database may occasionally contain duplicate reports of individuals diagnosed with cancer. Duplicate cases are additional reports of the same primary site cancer diagnosed in an individual by another health-care provider. In New Bedford, no duplicate reports were identified during the years 1996-2005. However, reports of individuals with multiple primary site cancers were included as separate cases in the analysis in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology as an earlier cancer, if diagnosed in the same primary site more than two months after the initial diagnosis (MCR 2003). Therefore, duplicate reports of an individual diagnosed with cancer would have been removed from the analysis whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases.

B. Calculation of a Standardized Incidence Ratio (SIR)

To assess the incidence of cancer in a community or CT, a statistic called the standardized incidence ratio (SIR) is calculated using data from the MCR. 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 CT) to the number of expected diagnoses based on the statewide rate. Comparison of SIRs between communities or CTs 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 1990 and 2000 U.S. census data for the community of interest (U.S. DOC 1990, 2000). 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[6].

A CT is a smaller geographic subdivision of a city or town designated by the U.S. Census Bureau. Because age group and gender-specific population information is necessary to calculate cancer incidence rates, the CT is the smallest geographic area for which cancer rates can be accurately calculated. 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).

SIRs were not calculated for some cancer types due to the small number of observed cases. It is standard MDPH/BHISRE policy not to calculate rates with fewer than five observed diagnoses due to the instability of the rate. However, the expected number of diagnoses was calculated during each time period, and the observed and expected numbers of diagnoses were compared to determine whether excess diagnoses of cancer were occurring.

C. Interpretation of a Standardized Incidence Ratio (SIR)

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

Specifically, an SIR of 100 indicates that the number of cancer diagnoses observed in the population being evaluated is equal to the number of cancer diagnoses expected in the comparison or “normal” population. An SIR greater than 100 indicates that more cancer diagnoses occurred than 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.

[6] Using slightly different population estimates or statistical methodologies, such as grouping ages differently or rounding off numbers at different points during calculations, may produce slightly different results.

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 may 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 (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 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.

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

D. Determination of Geographic Distribution of Cancer Diagnoses

Address at the time of diagnosis was mapped for each individual diagnosed with one of the six cancer types in New Bedford from 1996 to 2005 using a computerized geographic information system (GIS) (ESRI 2009). This allowed assignment of CT location for each diagnosis as well as an evaluation of the spatial distribution of individual diagnoses at a smaller geographic level within a CT (i.e., neighborhoods). The geographic pattern was assessed by qualitatively evaluating the point pattern of diagnoses for each of the six cancer types within CTs 6501.01 and 6501.02. This evaluation included consideration of the population density variability through the use of GIS-generated population density overlays. Due to community concerns related to the former Polymerine, Tallyrand and Polaroid sites, particular attention was paid to the spatial pattern of cancer in the vicinity of these sites (See Figure 2). In instances where the address information from the MCR was incomplete, that is, did not include specific streets or street numbers, efforts were made to research those individuals diagnosed with cancer (e.g., by using telephone books issued within two years of an individual's diagnosis or searching files via the Registry of Motor Vehicles).

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.

E. Evaluation of Cancer Risk Factor Information

In those instances where the incidence rate of a particular cancer type was higher than expected, available information from the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the six cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, tobacco history and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to bladder, kidney/renal pelvis, lung and bronchus, and pancreatic cancers. Other risk factors for various cancer types may include lack of crude fiber in the diet, high fat consumption, excessive alcohol consumption, and reproductive history. Heredity, or family history, is an important risk factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic. Environmental contaminants have also been associated with certain types of cancer. This information was evaluated to compare known or established risk factor patterns, as reported in the medical and epidemiological literature for particular cancer types, to risk factor information for individuals diagnosed in CTs 6501.01 and 6501.02, to assess whether any unexpected patterns exist. However, information about personal risk factors that might include family history, hormonal events, diet, and other factors that may also influence the development of cancer is not collected by the MCR; therefore, it was not possible to consider their contributions to cancer in this investigation.

VI. Results of Cancer Incidence Analysis

The following sections present cancer incidence rates for CTs 6501.01 and 6501.02 in the community of New Bedford during the 10-year time period 1996-2005. To evaluate possible trends over time as compared to the statewide cancer experience, these data were analyzed by two smaller time periods, 1996-2000 and 2001-2005. Tables 5 and 6 summarize cancer incidence data for CT 6501.01 while Tables 7 and 8 summarize cancer incidence data for CT 6501.02. The expected number of diagnoses was calculated during each time period and for each CT, and the observed and expected numbers of diagnoses were compared to determine whether excess numbers of cancer diagnoses were occurring. As previously mentioned, SIRs

were not calculated for some cancer types due to the small number of observed cases (less than five).

A. Cancer Incidence in CT 6501.01

Review of cancer incidence rates in CT 6501.01 showed that cancer incidence occurred as or below expected for two of the six cancer types evaluated (see Tables 5 and 6). In each of the two time periods evaluated, the incidence of bladder and breast cancer in both genders was either less than or about as expected when compared to the statewide cancer experience. For these two cancer types, if the number of observed diagnoses was greater than the number expected, the difference was based on one or two diagnoses and in no instance was the difference statistically significant. Furthermore, the geographic and temporal distribution of diagnoses for both of these cancer types was reviewed. Although some diagnoses did occur among individuals whose residences at the time of diagnosis were located within the Briarwood Development, the spatial distribution for each cancer type generally followed the population density pattern within the CT. In addition, the temporal distribution of diagnoses was not unusual. The incidence rates of the remaining four types of cancer – kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate – showed somewhat greater variability. It is important to note, however, that no elevations in incidence for these cancer types were statistically significant. The incidence of these four cancer types is discussed further below.

1. Kidney/Renal Pelvis Cancer Incidence

In CT 6501.01, the incidence of kidney/renal pelvis cancer in males and females combined was elevated during 1996-2000 (8 diagnoses observed compared to approximately 4 expected) but was about as expected based on the statewide experience during 2001-2005. A separate evaluation by gender revealed that the incidence of kidney/renal pelvis cancer among males was about as expected in both of the two time periods evaluated. Among females, however, the incidence was slightly elevated during 1996-2000 (5 diagnoses observed compared to approximately 2 expected) but was about as expected during 2001-2005. It is important to note that where the numbers of observed diagnoses were greater than expected, the differences were not statistically significant.

Available risk factor information was reviewed for those individuals in CT 6501.01 diagnosed with kidney/renal pelvis cancer between 1996 and 2000. According to the American Cancer Society (ACS), the average age of individuals diagnosed with kidney/renal pelvis cancer is 65 years. With the exception of Wilm's tumor, which is most common in children, kidney/renal pelvis cancer is very uncommon under the age of 45 and its incidence is highest among those over the age of 55 (ACS 2009a). In CT 6501.01, seven of the eight (88%) individuals diagnosed with kidney/renal pelvis cancer between 1996 and 2000 were over the age of 55, with an average age of 71 years at diagnosis.

Some lifestyle-related factors have been identified as risk factors for the development of kidney/renal pelvis cancer. They include smoking and obesity (ACS 2009a). Of the eight individuals diagnosed with kidney/renal pelvis cancer in CT 6501.01 between 1996 and 2000, two reported being current or former smokers at the time of diagnosis. No information on history of tobacco use was reported to the MCR for three individuals. Information on some risk factors such as obesity is not reported to the MCR.

Occupational exposure to certain substances such as asbestos, cadmium, benzene, organic solvents and some herbicides may also increase the risk of developing kidney/renal pelvis cancer (ACS 2009a). Occupational information as reported to the MCR at the time of diagnosis was reviewed for the eight individuals diagnosed with kidney/renal pelvis cancer in CT 6501.01 during 1996-2000 to determine the role that workplace factors may have played in the development of these cancers. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual diagnoses. In addition, these data are often incomplete as occupational information can be reported as unknown, at home, or retired. In CT 6501.01, three of the eight individuals diagnosed with kidney/renal pelvis cancer during 1996-2000 reported working in jobs possibly associated with an increased risk of kidney/renal pelvis cancer. Occupation was reported as unknown or at home for three individuals.

The histologies or subtypes of kidney/renal pelvis cancer in the eight individuals were also reviewed and compared to what would be expected, based on the medical literature and national

cancer statistics. In general, about 90% of all diagnoses of kidney/renal pelvis cancer are renal cell carcinomas (ACS 2009a). In CT 6501.01, four individuals were diagnosed with renal cell carcinomas. Histological information as reported to the MCR at the time of diagnosis was not specified for three of the other individuals.

The geographic and temporal distribution of the reported residences of individuals in CT 6501.01 diagnosed with kidney/renal pelvis cancer during 1996-2000 was reviewed. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, the geographic distribution generally followed the population density of the CT. From a temporal (i.e., time of diagnosis) perspective, all eight diagnoses occurred within a two year period. A review of the geographic distribution of individuals diagnosed with kidney/renal pelvis cancer between 2001 and 2005 also generally followed the population density of the CT. No temporal clustering occurred during this later time period as the dates of diagnosis were spread fairly evenly across the 5-year period.

2. Liver/Intrahepatic Bile Duct Cancer Incidence

Although the differences between the number of observed diagnoses and expected diagnoses of liver/intrahepatic bile duct cancer in CT 6501.01 during 1996-2000 and 2001-2005 were only one or two diagnoses, it was evaluated further because this is a fairly rare cancer type in the United States. Among males, the incidence in CT 6501.01 was slightly elevated during 1996-2000 (2 diagnoses observed compared to 1 expected) and 2001-2005 (3 diagnoses observed compared to about 1 expected). Neither difference was statistically significant. Among females, the incidence of liver/intrahepatic bile duct cancer was as expected during both time periods.

Available risk factor information was reviewed for the seven individuals in CT 6501.01 diagnosed with liver/intrahepatic bile duct cancer between 1996 and 2005. According to the ACS, more than 90% of individuals diagnosed with liver/intrahepatic bile duct cancer are older than 45 years of age, with an average age at diagnosis of 63 years (ACS 2012). In CT 6501.01, all 7 individuals diagnosed with liver/intrahepatic bile duct cancer between 1996 and 2005 were over the age of 45, with an average age of 66 years at diagnosis.

Some hereditary and medical conditions have been identified as risk factors for the development of liver/intrahepatic bile duct cancer. They include chronic infection with the hepatitis B virus or hepatitis C virus, cirrhosis (a disease in which liver cells become damaged and are replaced by scar tissue), and certain inherited metabolic diseases that can lead to cirrhosis (ACS 2012). Information on these risk factors is not reported to the MCR.

Exposure to vinyl chloride, a chemical used in making some kinds of plastics, may raise the risk of developing liver/intrahepatic bile duct cancer. Exposure of workers to vinyl chloride is now strictly regulated in the United States. In CT 6501.01, no individuals diagnosed with liver/intrahepatic bile duct cancer during 1996-2005 reported working in a job possibly associated with an increased risk liver/intrahepatic bile duct cancer. Occupation was reported as unknown for one individual.

The histologies of liver/intrahepatic bile duct cancer in the seven individuals were also reviewed and compared to what would be expected based on the medical literature and national cancer statistics. According to the ACS, hepatocellular carcinoma is the most common type of liver cancer in adults and accounts for about 75% of all diagnoses (ACS 2012). In CT 6501.01, six individuals were diagnosed with hepatocellular carcinoma. Histological information as reported to the MCR at the time of diagnosis was not specified for one individual.

The geographic distribution of place of residence for individuals in CT 6501.01 diagnosed with liver/intrahepatic bile duct cancer during 1996-2005 was reviewed. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, these occurrences were located in areas of higher population density. From a temporal perspective, the dates of diagnosis for all seven individuals were spread fairly evenly over a six-year period.

3. Lung and Bronchus Cancer Incidence

The incidence of lung and bronchus cancer in males and females combined was about as expected in CT 6501.01 during both time periods evaluated. A separate evaluation by gender showed that among females, the incidence of lung and bronchus cancer was either less than or

about as expected during both time periods. Among males, the incidence of lung and bronchus cancer was elevated during 1996-2000 (19 diagnoses observed compared to about 13 expected), though this difference was not statistically significant. During 2001-2005, the incidence of lung and bronchus cancer among males was about as expected.

Available risk factor information was reviewed for males in CT 6501.01 diagnosed with lung and bronchus cancer between 1996 and 2000. According to the ACS, lung and bronchus cancer mainly occurs in older individuals, with roughly two-thirds of those diagnosed older than 65 years of age. In CT 6501.01, 12 of the 19 (63%) males diagnosed with lung and bronchus cancer during this time period were 65 years of age or older at the time of diagnosis. The average age at diagnosis was 67 years, which is consistent with the national average of 71 years and the state average of 69 years.

The histologies of lung and bronchus cancer in the 19 males were reviewed and compared to what would be expected, based on the medical literature and national cancer statistics. According to the ACS, about 85% to 90% of all diagnoses of lung and bronchus cancers are non-small cell lung cancer (NSCLC), with small-cell lung cancer (SCLC) comprising the remaining 10% to 15% (ACS 2008a). In CT 6501.01, nine males were diagnosed with NSCLC and six were diagnosed with SCLC. Histological information as reported to the MCR at the time of diagnosis was not specified for four individuals.

Tobacco use is by far the most important risk factor for lung and bronchus cancer. It is estimated that 85% to 95% of deaths from lung and bronchus cancer are caused by smoking. The longer a person has been smoking and the higher the number of cigarettes smoked per day, the greater the risk of lung and bronchus cancer. No matter the age of an individual or how long someone has used tobacco, quitting may help an individual to live longer. It should be noted that SCLC is almost always caused by smoking and rarely develops in an individual who has never smoked (ACS 2008a,b). For the 10 males diagnosed with lung and bronchus cancer in CT 6501.01 during 1996-2000 with a known tobacco history, eight (80%) were current or former tobacco users. The tobacco history of nine of the 19 males is unknown. Of the six males diagnosed with SCLC, three were current or former smokers and the tobacco history of the remaining three

individuals is unknown. On a statewide level, about 93% of individuals in Massachusetts that were diagnosed with lung and bronchus cancer during 1996-2000 and had a known tobacco history were current or former tobacco users.

Exposures to several substances, particularly radon, have been identified as important risk factors in the development of lung and bronchus cancer. Radon is a naturally occurring radioactive gas produced by the breakdown of uranium in soils and rocks. High indoor levels of radon can occur in homes and buildings, especially in basements. Exposure to radon has been identified as the second leading cause of lung and bronchus cancer, and the leading cause among nonsmokers. According to the USEPA, homes within Bristol County have moderate potential for elevated radon levels (USEPA 2009). However, radon levels cannot be more accurately predicted based on state, local, and neighborhood radon measurements because of natural geologic variability. Even homes which are next to each other can have different radon levels. Testing of individual homes is the only way to find out the radon level in a home.

Workplace exposure to asbestos has also been identified as an established risk factor for lung and bronchus cancer. Exposure to asbestos may occur in mines, mills, textile plants, shipyards, and where insulation is used. Asbestos is not usually considered harmful as long as it is not released into the air by deterioration, demolition, or renovation. Additional chemical compounds that are occupational risk factors include arsenic, beryllium, cadmium, silica, vinyl chloride, nickel compounds, chromium, coal products, mustard gas, chloromethyl ethers, diesel exhaust, and radioactive ores such as uranium (ACS 2008a,b). The risk of developing lung and bronchus cancer from workplace exposure to these chemicals is even higher for smokers. Occupational information as reported to the MCR at the time of diagnosis was reviewed for the 19 males diagnosed with lung and bronchus cancer in CT 6501.01 during 1996-2000 to determine the role that workplace factors may have played in the development of these cancers. As mentioned previously, occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual diagnoses. In addition, these data are often limited as occupational information can be reported as unknown, at home, or retired. In CT 6501.01, six of the 19 individuals diagnosed with lung and bronchus cancer during 1996-2000 reported working in jobs possibly associated

with an increased risk of lung and bronchus cancer. Occupation was reported as unknown or retired for two individuals.

The geographic and temporal distribution of place of residence for individuals in CT 6501.01 diagnosed with lung and bronchus cancer between 1996 and 2000 was reviewed. In general, the spatial distribution of diagnoses followed the population density of the CT. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, these occurrences were located in areas of higher population density. In addition, there was no temporal clustering as the dates of diagnosis were spread fairly evenly across the 5-year period. Furthermore, a review of the geographic distribution of individuals diagnosed with lung and bronchus cancer between 2001 and 2005 similarly followed the population density of the CT.

4. Prostate Cancer Incidence

The incidence of prostate cancer in CT 6501.01 was less than expected during 1996-2000 (22 diagnoses observed compared to 26 expected) but greater than expected during 2001-2005 (31 diagnoses observed compared to approximately 25 expected) based on the statewide cancer experience. However, this elevation was not statistically significant.

Available risk factor information was reviewed for those men in CT 6501.01 diagnosed with prostate cancer between 2001 and 2005. According to the ACS, age is the strongest risk factor for prostate cancer. Prostate cancer is very rare before the age of 40, but the risk of developing it rises rapidly after age 50. Almost 2 out of 3 men diagnosed with prostate cancer are over the age of 65 at diagnosis (ACS 2009b). In CT 6501.01, 16 of the 31 (52%) men diagnosed with prostate cancer between 2001 and 2005 were over the age of 65, with an average age at diagnosis of 67 years.

The histologies of prostate cancer in the 31 individuals were also reviewed and compared to what would be expected, based on the medical literature and national cancer statistics. According to the ACS, over 99% of prostate cancers are adenocarcinomas (ACS 2009b). In CT

6501.01, 30 men were diagnosed with adenocarcinomas. Histological information as reported to the MCR at the time of diagnosis was not specified for one individual.

The geographic and temporal distribution of place of residence for males in CT 6501.01 diagnosed with prostate cancer between 2001 and 2005 was reviewed. The spatial distribution generally followed the population distribution of the CT. Although some diagnoses occurred among individuals whose residences at the time of diagnosis were in relative close proximity to one another, these occurrences were located in areas of higher population density. In addition, there was no temporal clustering as the dates of diagnosis of the 31 individuals were spread fairly evenly across the 5-year period.

B. Cancer Incidence in CT 6501.02

In general, cancer incidence in CT 6501.02 was approximately at or near that which was expected during both time periods evaluated (see Tables 7 and 8). In each of the two time periods, the incidence of the following three cancer types in both genders was either less than expected or about as expected when compared to the statewide cancer experience: bladder, breast, and lung and bronchus. For these three cancer types, if the number of observed diagnoses was greater than the number expected, the difference was based on one or two diagnoses and was not statistically significant. The geographic and temporal distribution of diagnoses for each of these three cancer types was also reviewed. Although diagnoses did occur among individuals whose residences at the time of diagnosis were located within Pine Hill Acres and near the New Bedford Business Park, the spatial distribution for each cancer type generally followed the population density pattern within the CT. In addition, the temporal distribution of diagnoses was not unusual. The incidence rate of the remaining three cancer types – kidney/renal pelvis, liver/intrahepatic bile duct and prostate cancer – showed slightly greater variability. However, no elevations in the incidence of these cancer types were statistically significant, indicating that they could represent natural or random variation. The incidence of kidney/renal pelvis cancer, liver/intrahepatic bile duct cancer and prostate cancer in CT 6501.02 are discussed further below.

1. Kidney/Renal Pelvis

Although the incidence of kidney/renal pelvis cancer in males and females combined was about as expected in CT 6501.02 during both time periods evaluated, a separate evaluation by gender

revealed a slightly different pattern. Among females, the incidence of kidney/renal pelvis cancer was either less than or about as expected during both time periods. Among males, however, the incidence of kidney/renal pelvis cancer was slightly elevated during 1996-2000 (4 diagnoses observed compared to approximately 2 expected), though this difference was not statistically significant. During 2001-2005, the incidence of kidney/renal pelvis cancer among males was about as expected.

Available risk factor information was reviewed for those individuals in CT 6501.02 diagnosed with kidney/renal pelvis cancer between 1996 and 2000. As mentioned previously, the incidence of kidney/renal pelvis cancer is highest among those over the age of 55 and the average age of individuals diagnosed with this cancer type is 65 years (ACS 2009a). In CT 6501.02, two of the four (50%) individuals diagnosed with kidney/renal pelvis cancer between 1996 and 2000 were over the age of 65. The average age at diagnosis for all four individuals was 56 years. Although smoking is an established risk factor for kidney/renal pelvis cancer, history of tobacco use was unknown for three of the four individuals diagnosed in this CT during 1996-2000. Occupational information as reported to the MCR at the time of diagnosis was reported as unknown for one of the four individuals. Of the remaining three individuals diagnosed with kidney/renal pelvis cancer during 1996-2000, one reported working in a job possibly associated with an increased risk of kidney/renal pelvis cancer.

The histologies of kidney/renal pelvis cancer in the four individuals were also reviewed and compared to what would be expected, based on the medical literature and national cancer statistics. In CT 6501.02, three individuals (75%) were diagnosed with renal cell carcinomas. This is consistent with the national trend in which about 90% of all diagnoses of kidney/renal pelvis cancer are renal cell carcinomas (ACS 2009a). Histological information as reported to the MCR at the time of diagnosis was not specified for one individual.

The geographic and temporal distribution of the reported residences of individuals in CT 6501.02 diagnosed with kidney/renal pelvis cancer during 1996-2000 was reviewed. The spatial distribution of diagnoses generally followed the population density pattern of the CT. Although two diagnoses occurred among individuals whose residences at the time of diagnosis were in

relative close proximity to one another, this occurrence was located in an area of higher population density. From a temporal perspective, two diagnoses occurred within a period of one month. The four diagnoses occurred over a four year period. However, it is difficult to ascertain the significance of this, if any, due to the latency period for kidney/renal pelvis cancer and the small number of individuals diagnosed with this type of cancer. A review of the geographic distribution of individuals diagnosed with kidney/renal pelvis cancer during 2001 and 2005 similarly did not show any unusual spatial patterns.

2. Liver/Intrahepatic Bile Duct Cancer Incidence

As mentioned previously, liver/intrahepatic bile duct cancer is fairly rare in the United States. For this reason, the incidence of this cancer type was further evaluated. In CT 6501.02, no diagnoses of liver/intrahepatic bile duct cancer were observed during 1996-2000. During 2001-2005, two diagnoses were observed compared to about one expected. The incidence of liver/intrahepatic bile duct cancer among males was about as expected (1 observed diagnosis compared to about 1 expected) whereas that among females was slightly elevated (1 observed diagnosis compared to 0 expected). These differences were not statistically significant.

Available risk factor information was reviewed for those individuals diagnosed in CT 6501.02 with liver/intrahepatic bile duct cancer between 2001 and 2005. As discussed previously, more than 90% of individuals diagnosed with liver/intrahepatic bile duct cancer in the United States are older than 45 years of age, with an average age at diagnosis of 63 years (ACS 2012). In CT 6501.02, both individuals diagnosed with liver/intrahepatic bile duct cancer during 2001-2005 were over the age of 45, with an average age of 57 years.

Although some hereditary and medical conditions (e.g., chronic infection with the hepatitis B virus or hepatitis C virus, cirrhosis) have been identified as risk factors for the development of liver/intrahepatic bile duct cancer, this information is not reported to the MCR. Furthermore, neither of the two individuals in CT 6501.02 diagnosed with liver/intrahepatic bile duct cancer during 2001-2005 reported working in a job possibly associated with an increased risk of liver/intrahepatic bile duct cancer.

The histologies of liver/intrahepatic bile duct cancer in the two individuals diagnosed in CT 6501.02 during 2001-2005 were hepatocellular carcinomas. This is consistent with the national trend in which about 75% of all diagnoses of liver/intrahepatic bile duct cancer are hepatocellular carcinomas (ACS 2012).

Although one diagnosis was located near the New Bedford Business Park, it was within an area of higher population density within the CT. In general, no unusual geographic or temporal patterns were observed.

3. Prostate Cancer Incidence

In CT 6501.02, the incidence of prostate cancer was less than expected during 1996-2000 (13 diagnoses observed compared to about 16 expected) but slightly greater than expected during 2001-2005 (19 diagnoses observed compared to about 16 expected) based on the statewide cancer experience. It is important to note, however, that while the number of observed diagnoses was greater than the number of expected diagnoses, the difference was not statistically significant.

Available risk factor information was reviewed for those men in CT 6501.02 diagnosed with prostate cancer between 2001 and 2005. As mentioned previously, age is the strongest risk factor for prostate cancer with almost 2 out of 3 diagnoses occurring in men over the age of 65 (ACS 2009b). In CT 6501.02, 9 of the 19 (47%) men diagnosed with prostate cancer between 2001 and 2005 were over the age of 65, with an average age at diagnosis of 67 years.

The histologies of prostate cancer in the 19 individuals were also reviewed and compared to what would be expected, based on the medical literature and national cancer statistics. All 19 (100%) of the men in CT 6501.02 were diagnosed with adenocarcinomas, which is consistent with the national trend in which over 99% of prostate cancers are adenocarcinomas.

The geographic and temporal distribution of place of residence for males in CT 6501.02 diagnosed with prostate cancer between 2001 and 2005 was reviewed. The spatial distribution of diagnoses generally followed the population density of the CT. Although many diagnoses

occurred among individuals whose residences at the time of diagnosis were located within Pine Hill Acres, this is an area with relatively high population density. In addition, there was no temporal clustering as the dates of diagnosis of the 19 individuals were spread fairly evenly across the 5-year period.

VII. Discussion

MDPH/BEH reviewed available environmental data related to specific facilities within the New Bedford Business Park to assess possible exposure opportunities to constituents related to these properties. In addition, asthma hospitalization and cancer incidence data were reviewed to help address community concerns about possible impacts of these facilities on the pattern of disease in nearby neighborhoods.

The New Bedford Business Park was initially developed in 1960 and currently consists of more than 40 businesses. In the past, a number of properties within the business park have had a release of oil or other hazardous material that was reported to MDEP under the statewide hazardous waste site cleanup program. Most of these releases were localized, addressed with an immediate response action, and considered by MDEP to pose no significant risk of health or environmental impacts. This health consultation focused on facilities within the business park that had a release of oil or hazardous material that was under regulation or remediation at the time that this investigation was initiated, as well as the former Polaroid facility, which was of particular community concern.

All of the sites within the New Bedford Business Park in this investigation (former BorgWarner, Polymerine, Tallyrand and Polaroid) had releases to the environment that resulted in groundwater contamination. Importantly, all residential areas near the business park are either served by municipal drinking water supplies and, hence, do not use local groundwater for household water, or are located upgradient from the business park. In addition, nearby residents would not be affected by groundwater contamination through possible vapor intrusion into houses due to the distance to the nearest residences. Thus, we would not expect adverse health impacts associated with groundwater contamination in the past, present, or future.

Three sites (the former BorgWarner, Polymerine, and Tallyrand facilities) all had soil contamination that included PCBs and/or petroleum compounds. In all cases, extensive soil removal was conducted. Remediation efforts are ongoing at the former BorgWarner and Polymerine sites. Although it was possible for an individual to contact soils containing some of these compounds in the past, it is unlikely that anyone came into contact with contaminated soils on a regular basis currently or in the past given the nature of the site as an industrial business park. However, even under the highly unlikely possibility that an individual frequently visited and/or played at the former Polymerine or Tallyrand sites in the past or currently, health impacts would not be expected from contact with contaminated soils. The business park is expected to continue as such for the foreseeable future, and hence, health impacts are not expected under future scenarios.

The former Polaroid facility is not under active investigation or remediation. However, nearby residents had specific concerns about historical exposure opportunities from this facility, particularly from air emissions. Based on available data, this facility emitted methanol, MEK and hydrochloric acid in the past and was generally in compliance with air quality permit specifications with the exception of two violations, after which corrective actions were promptly taken. Although these compounds can produce noticeable odors and may cause a range of health concerns such as headaches and respiratory irritation from long term exposure, they have not been classified with respect to carcinogenicity. No ambient air quality concentration data were available for this facility, from either onsite or offsite locations. Thus, it is difficult to quantitatively evaluate exposure opportunities that may have occurred in the past. However, since MEK is a respiratory irritant, the MDPH examined available hospital discharge data for asthma hospitalizations of residents of zip code 02745 during 2000-2003. This four-year time period reflects the most complete data available through 2003, after which point MEK was no longer emitted from the former Polaroid facility.

The number of asthma hospitalizations in zip code 02745 generally increased during 2000-2003. The total number of hospitalizations during this four-year time period was greater than expected based on the statewide experience and the difference was of borderline statistical significance. The number of asthma hospitalizations in zip code 02745 was statistically significantly lower

than expected in 2000 whereas the number was statistically significantly elevated in 2003. Overall, there does not appear to be a relationship between the level of emissions of MEK from the former Polaroid facility and the number of asthma hospitalizations within zip code 02745 during 2000-2003. The level of emissions of MEK generally decreased over this time period and a corresponding decrease in the number of asthma hospitalizations was not observed.

To address community concerns about cancer incidence, the CAP reviewed cancer incidence data available from the MCR for six cancer types (bladder, breast, kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate cancer) in the two CTs (6501.01 and 6501.02) containing the neighborhoods of concern during the 10-year time period, 1996-2005. The six cancer types evaluated were chosen based on residents' concerns over suspected elevations of these cancer types.

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 town. 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.

In general, there does not seem to be an unusual pattern of cancer incidence in either CT 6501.01 or 6501.02. In both CTs, the incidence of bladder and breast cancer was either less than or about as expected based on the statewide cancer experience during 1996-2000 and 2001-2005. For these two cancer types, if the number of observed diagnoses was greater than the number expected, the difference was based on one or two diagnoses and was not statistically significant. Although there were elevations in cancer of the kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate during certain time periods, in no case was the difference between the numbers of observed and expected diagnoses statistically significant, meaning that they could be due to chance and represent natural variability in rates.

Available risk factor information on tobacco use and occupation for those diagnosed with kidney/renal pelvis in both CTs and those diagnosed with lung and bronchus cancer in CT 6501.01 were compared to known or established trends to assess whether any unexpected patterns occurred. Review of these data suggests that tobacco use likely played some role in the development of these cancers among some individuals. Occupational exposures may have also been important in the development of these cancers among some individuals. However, because of the large number of individuals for whom tobacco history and/or occupation was unknown, it is difficult to fully assess the extent to which these factors influenced the overall cancer pattern.

Because liver/intrahepatic bile duct cancer is a fairly rare cancer type in the United States, available risk factor information on age and occupation was reviewed for those individuals diagnosed in CT 6501.01 and 6501.02 during 1996-2005. The ages at diagnosis for these individuals were consistent with national statistics. In addition, the available data suggest that occupational exposures are unlikely to have played a role in the development of these cancers amongst these individuals. According to the ACS, the incidence of liver/intrahepatic bile duct cancer in the United States has been rising slowly for several decades.

Available risk factor information was also reviewed for those men diagnosed with prostate cancer in both CTs, with an emphasis on age as this is the strongest risk factor for this cancer type. It should be noted that prostate cancer is the most common cancer (other than skin cancers)

in American men. The ACS estimates that about 1 man in 6 will be diagnosed with prostate cancer during his lifetime.

The analysis of the geographic distribution of address at the time of diagnosis for individuals diagnosed with the six cancer types evaluated in the two CTs did not reveal any atypical spatial patterns that would suggest a common factor (environmental or non-environmental) played a primary role in the incidence of cancer in either CT during the 10-year time period, 1996-2005. Although diagnoses did occur among individuals whose residences at the time of diagnosis were located within the two neighborhoods of interest, the spatial distribution for each cancer type generally followed the population density pattern within the CT.

VIII. Limitations

This health consultation is an investigation that analyzed descriptive health outcome data for asthma hospitalizations and cancer incidence to determine whether the pattern or occurrence of disease in CTs 6501.01 and 6501.02 is unusual. The purpose of this investigation was to evaluate the pattern of cancer in a geographical context in relation to available information about risk factors, including environmental factors, related to six specific cancer types 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. However, inherent limitations in the available data make it impossible to determine the precise causal relationships or synergistic roles that may have contributed to the development of individual cancers in this community. Also, this type of analysis cannot determine what may have caused cancer in any one particular individual. Cancers in general have a variety of risk factors known or suggested to be related to the cause of the disease that could not be evaluated in this investigation. It is believed that many cancers are related largely to lifestyle factors such as tobacco use, diet, and alcohol consumption. Other factors associated with cancer include socioeconomic status, heredity/genetics, race, and geography. It is beyond the scope of this investigation to determine the causal relationship of these factors and the development of cancer in these two CTs.

IX. Conclusions

Based on the MDPH's evaluation of the available environmental data, the exposure pathway analysis, asthma hospitalization data, and risk factor information related to the six cancer types evaluated (bladder, breast, kidney/renal pelvis, liver/intrahepatic bile duct, lung and bronchus, and prostate cancer), MDPH concludes that:

- **Drinking tap water in the past, present or future** is not expected to harm people's health. The reason for this is because groundwater at the former BorgWarner, Polymerine, Tallyrand, and Polaroid sites was not used for drinking water historically and is not used as a source presently. The surrounding neighborhoods are generally served by the City of New Bedford Water Department, with the exception of possible private wells on Braley Road that are located upgradient. As a result, there are no completed groundwater exposure pathways in the past, present, or future. In addition, there are no completed vapor intrusion exposure pathways resulting from releases at these four sites for nearby residents in the past, present or future.
- **Incidentally eating or touching soil or sediment while at the former Polymerine or Tallyrand sites in the past** is not expected to have harmed people's health. The reason for this is because, based on the available information and conservative assumptions about the frequency and duration of potential past exposures of trespassers, levels of chemical contaminants that could get into an adult or older child's body are below levels that would harm their health.
- **Incidentally eating or touching soil or sediment at the former Polymerine or Tallyrand sites presently or in the future** is not expected to harm people's health. Although removal activities were conducted at the former Polymerine site, PCBs were still detected in remaining on-site surface soils at levels above health-based comparison values. Likewise, although remediation at the former Tallyrand site included the removal of contaminated soil, PCBs remaining in on-site surface soil are not expected at concentrations above the USEPA action level but could still exceed health-based comparison values. However, based on conservative assumptions about the frequency and duration of potential exposures in the present or future, levels of chemical contaminants that could get into an adult or older child's body while trespassing at either the former Polymerine or Tallyrand sites are below levels that would harm their health.

- **Incidentally eating or touching soil at the former Polaroid facility in the past, present or future** is not expected to harm people's health. The reason for this is because no exposures to contaminated soil from the release of sulfuric acid at the former Polaroid facility are expected to occur due to the depth below ground surface.
- **Exposure of nearby residents to chemicals in air emissions from the former Polaroid facility** was possible when the facility was in operation. Since there are no historical ambient air data available for the facility or the surrounding neighborhoods, it is not possible to quantitatively evaluate whether facility emissions may have resulted in chemical concentrations in ambient air greater than health-based comparison values. Since MEK is a respiratory irritant, the MDPH examined available hospital discharge data for asthma hospitalizations of residents of zip code 02745 during the four-year time period of 2000-2003. Overall, there does not appear to be a relationship between the level of emissions of MEK from the former Polaroid facility and the number of asthma hospitalizations within the zip code. The use of MEK at the former facility was discontinued after 2003.
- **Within CT 6501.01, the incidence of bladder and breast cancer** in both genders during 1996-2000 and 2001-2005 was either less than or about as expected based on the statewide cancer experience.
- **Within CT 6501.01, the incidence of kidney/renal pelvis, lung and bronchus, and prostate cancer** was elevated during one of the two time periods evaluated and varied by gender. However, no elevations were statistically significant.
- **Within CT 6501.02, the incidence of bladder, breast, and lung and bronchus cancer** was either less than or about as expected among males and females during 1996-2000 and 2001-2005.
- **Within CT 6501.02, the incidence of kidney/renal pelvis and prostate cancer** was either elevated or slightly elevated among males during one of the two time periods evaluated. No elevations, however, were statistically significant.
- **Within CT 6501.01 and CT 6501.02, the incidence of liver/intrahepatic bile duct cancer**, which is fairly rare in the United States, was slightly greater than expected and varied by gender. However, no elevations were statistically significant.

- **Review of risk factor information** suggests that tobacco use likely played some role in the development of kidney/renal pelvis and lung and bronchus cancer among some individuals. Occupational exposures may have also been important in the development of these cancer types among some individuals. It is difficult to fully assess the extent to which these factors influenced overall cancer patterns due to the number of individuals for whom tobacco history and/or occupation was unknown.
- **Analysis of the geographic distribution of place of residence** for individuals diagnosed with the six cancer types evaluated in the two CTs revealed spatial patterns that generally followed the population density pattern. Although several diagnoses did occur among individuals whose residences at the time of diagnosis were located within the two neighborhoods of interest, these were areas of relative higher population density.

X. Recommendations

The MDPH recommends no further investigation of cancer incidence in CTs 6501.01 and 6501.02 at this time.

For more information about quitting smoking, contact the Massachusetts Tobacco Cessation and Prevention Program at 1-800-Quit-Now or 1-800-784-8669 or visit the website http://makesmokinghistory.org/.

The only way to know if your home has a radon problem is to do a radon test. The MDPH recommends that all residences in Massachusetts be tested for radon. For further questions about radon, you may contact the MDPH/BEH's Radiation Control Program at 1-800-723-6695 for advice on home testing.

XI. References

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Figures

Figure 1
Location of Environmental Concern
New Bedford, Massachusetts

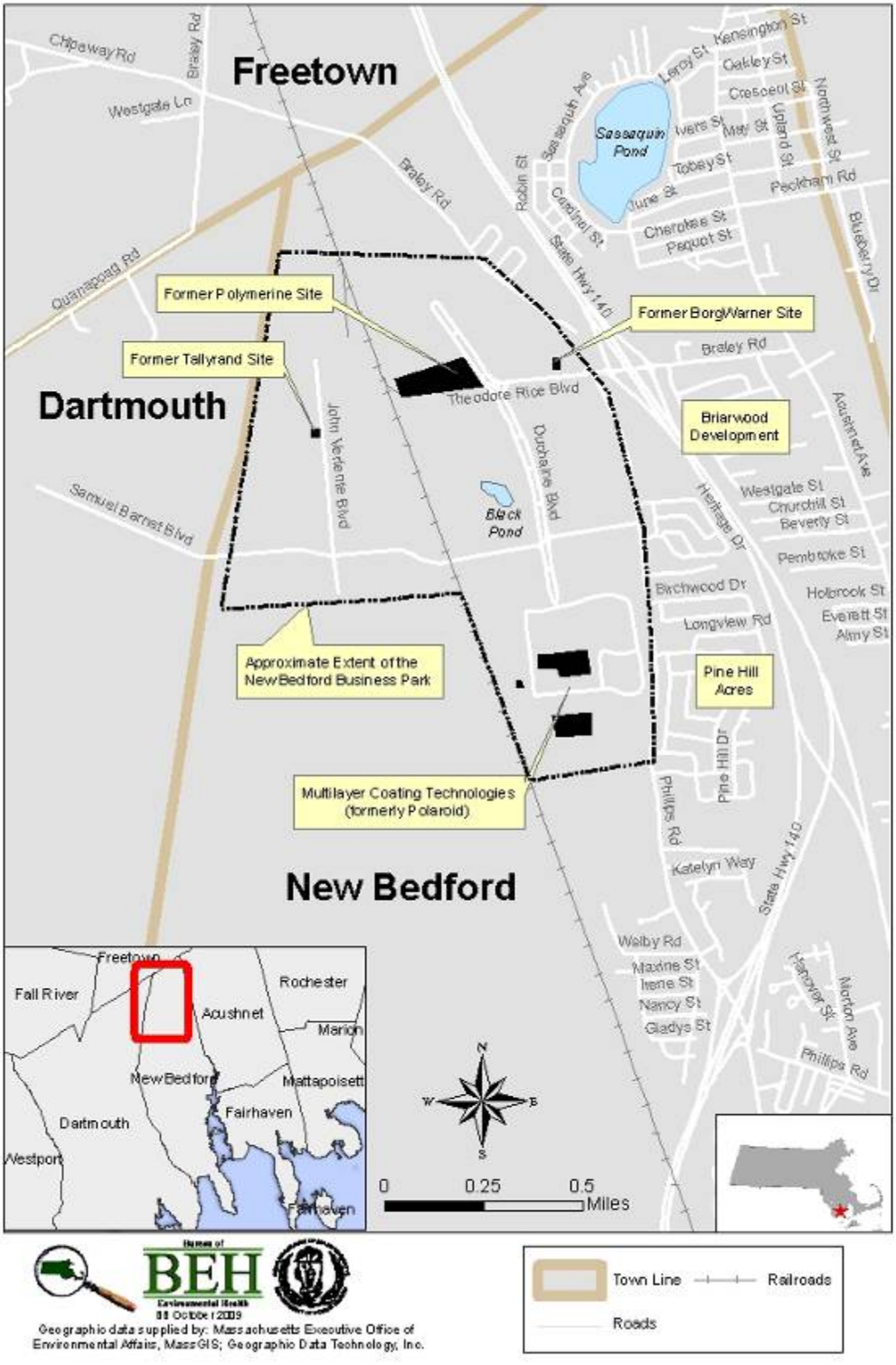


Figure 2
Area of Analysis
New Bedford, Massachusetts

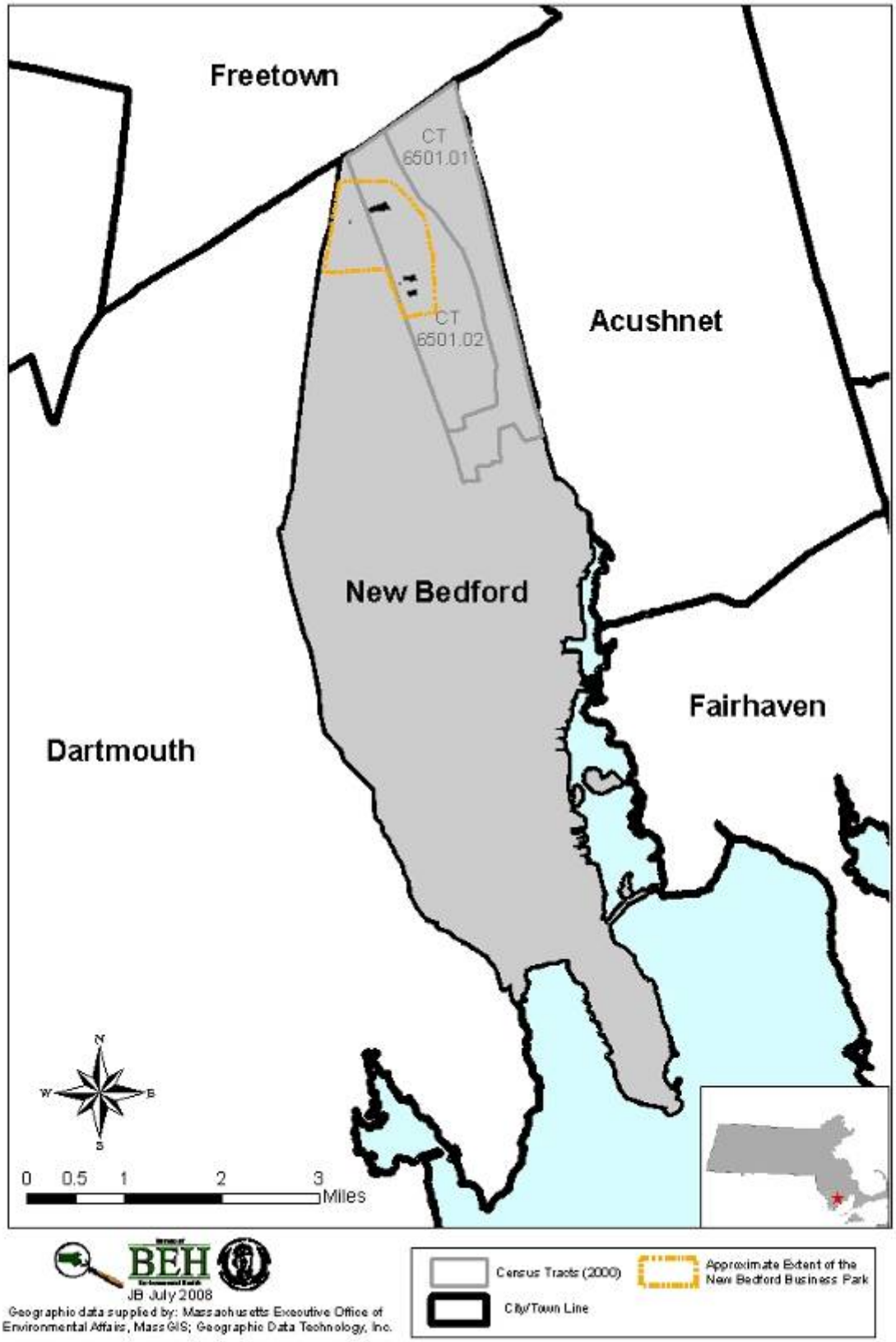


Figure 3
Surface Soil Samples Prior to 1998 Excavation at the Former Polymerine Site
New Bedford, Massachusetts

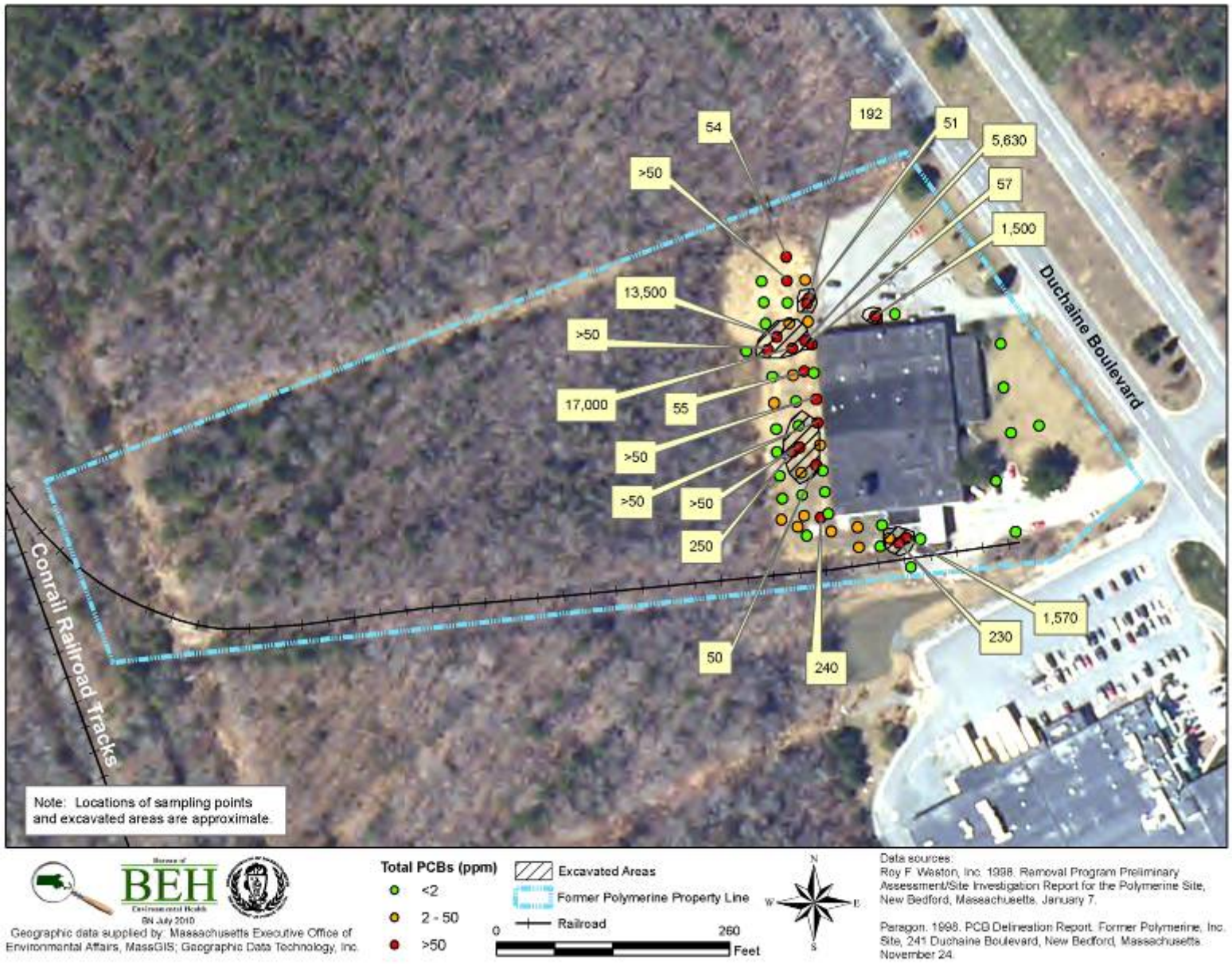


Figure 4
Surface Soil Samples Prior to 2000-2001 Excavation at the Former Polymerine Site
New Bedford, Massachusetts

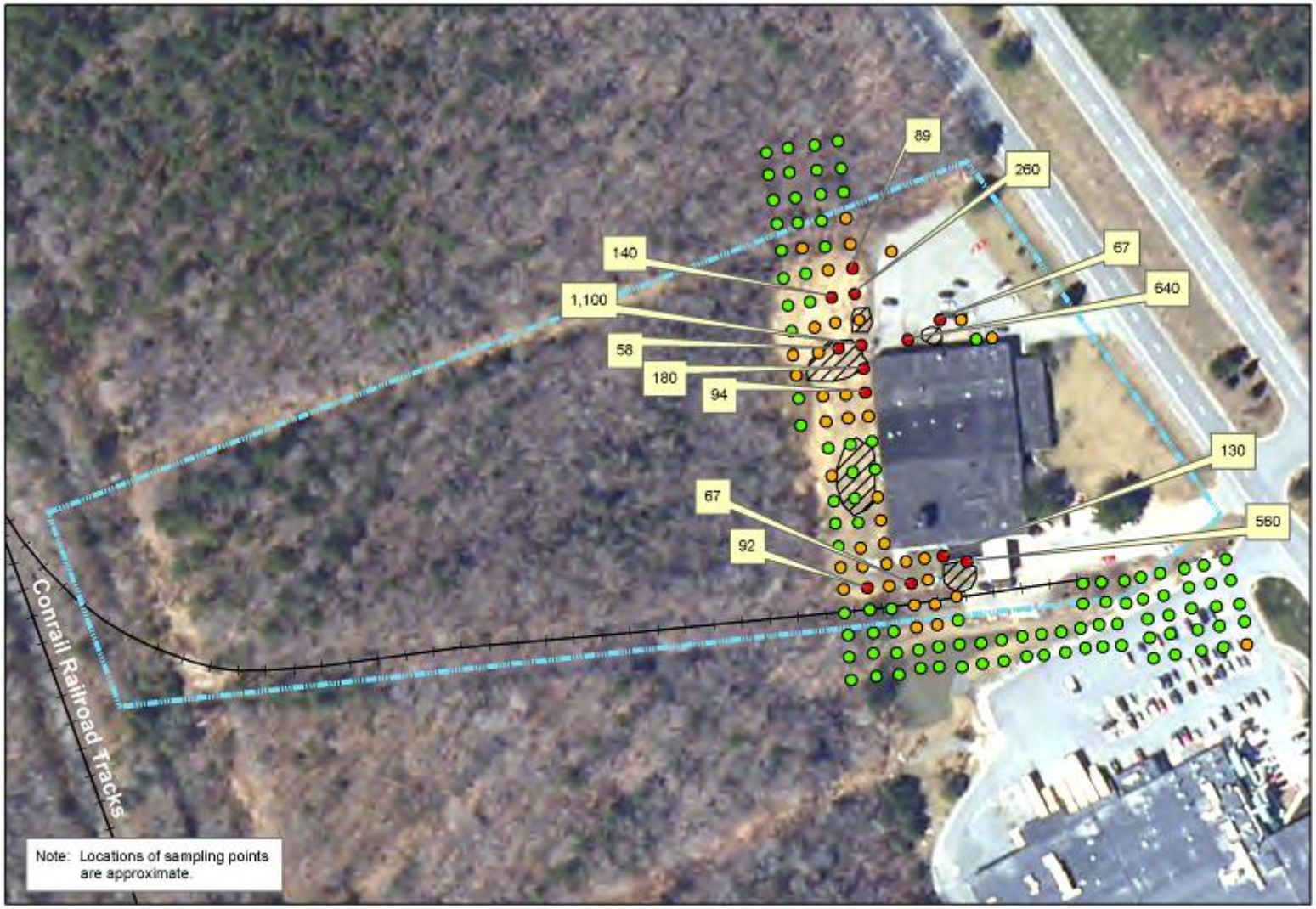
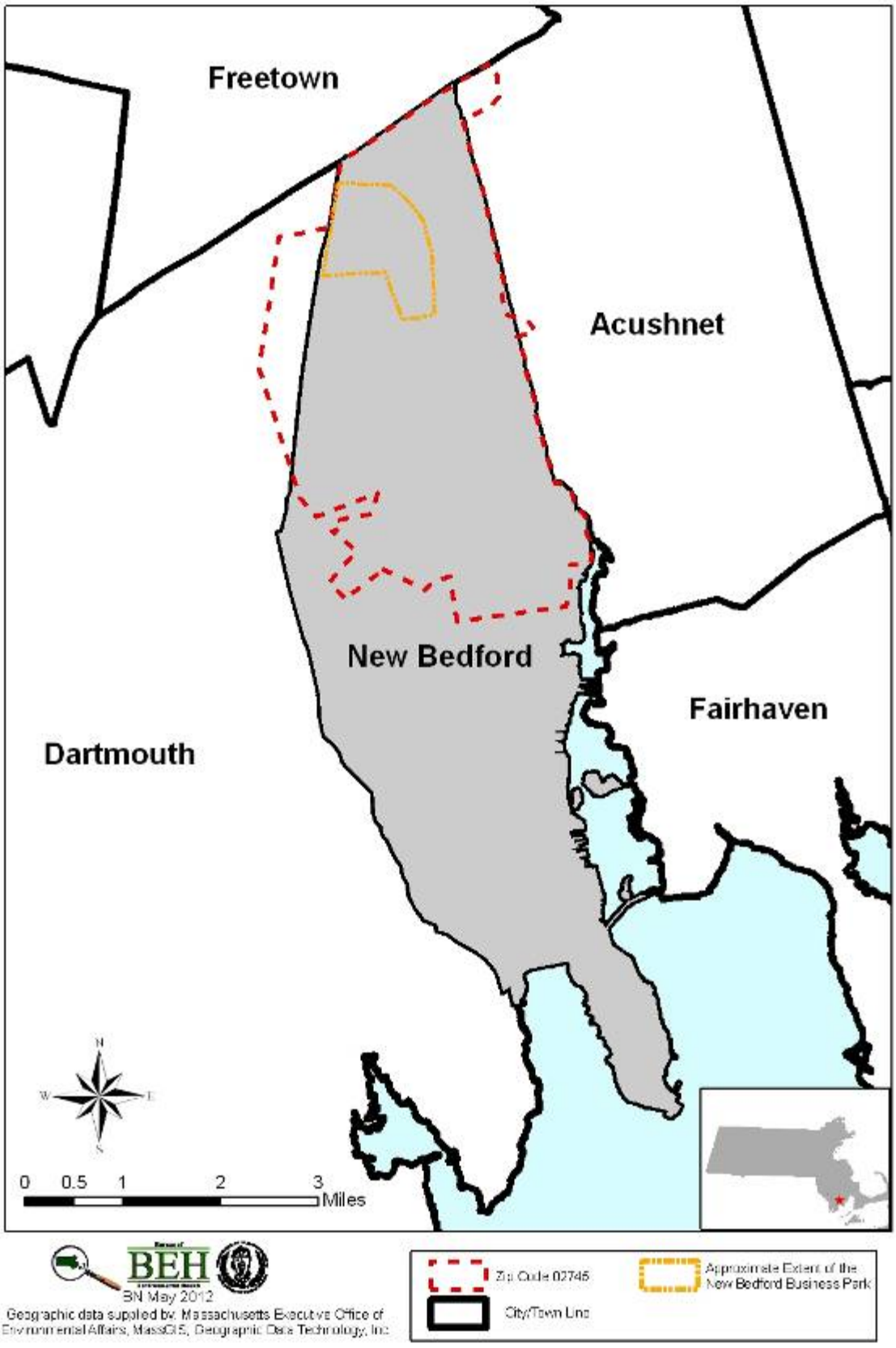


Figure 5
Post-Excavation Soil Surface Samples Collected in 2008-2009 at the Former Polymerine Site
New Bedford, Massachusetts

Figure 6
Former Tallyrand Site Map and Release Area
New Bedford, Massachusetts

Figure 7
Area of Analysis for Asthma Hospitalization Data
Zip Code 02745, New Bedford, Massachusetts



Tables

Table 1
Maximum Concentrations of Contaminants Detected in On-Site Sediment and Soil Samples at the Former Polymerine Site that Exceed Comparison Values

Contaminant	Medium	Date of sample	Descriptive location of sample Sample depth (feet)	Maximum concentration (ppm)	Soil Background (ppm)	Soil comparison value (ppm)
Copper	Soil	10/6/1998	Unknown (surficial)	8,300	40 (natural soil) 200 (fill material)	Intermediate EMEG (child) = 500 Intermediate EMEG (adult) = 7,000
Total Polychlorinated Biphenyls	Soil	10/6/1998	West of the on-site buliding 13S/30W (1-3 inches)	17,000 [1]	--	CREG = 0.4 MDEP S-1 & GW-1 standard = 2
Total Polychlorinated Biphenyls	Sediment	3/27/2009	Northwest of the on-site building Station C-42 (0 - 6 inches)	1.2	--	CREG[2] = 0.4 MDEP S-1 & GW-1 standard = 2

Notes:

-- = No value available.

[1] Maximum concentration is the average of two duplicate samples (19,000 ppm, 15,000 ppm) taken at this location.

[2] Because ATSDR comparison values do not exist for sediment, soil comparison values were used for screening purposes.

Data sources:

Massachusetts Department of Environmental Protection, Office of Research and Standards. 2002. Technical Update: Background Levels of Polycyclic Aromatic Hydrocarbons and
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Tighe & Bond. 2009. Phase II Comprehensive Site Assessment, Former Polymerine, 241 Duchaine Boulevard, New Bedford, MA, RTN 4-1347. May.

Comparison values (source organization, reference):

CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR, ATSDR 2008a)
Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures between 14 days and 1 year) (ATSDR, ATSDR 2008a)
Intermediate EMEG (child) = Environmental Media Evaluation Guide for children (i.e., for exposures between 14 days and 1 year and considers vulnerabilities of children when it comes to environmental exposures) (ATSDR, ATSDR 2008a)

MDEP S-1 & GW-1 = MCP Method 1 soil category S-1 standards applicable to soil where the combination of soil & groundwater categories are S-1 & GW-1 (MDEP, MDEP 2008)

Table 2
Maximum Concentrations of Contaminants Detected in On-Site Soil Samples at the Former Tallyrand Site that Exceed Comparison Values

Contaminant	Date of sample	Descriptive location of sample Sample depth (feet)	Maximum concentration (ppm)	Soil Background (ppm)	Soil comparison value (ppm)
Total Polychlorinated Biphenyls (PCBs)	10/21/1997	1-FS-2.0-009 (2 feet)	13,500	--	CREG = 0.4 MassDEP S-1 & GW-1 standard = 2
Total Petroleum Hydrocarbons	12/8/1995	Site SS2E (Surficial)	4,200	--	MassDEP S-1 & GW-1 standard = 1,000

Notes:
-- = No value available.

Data sources:
SITEC Environmental, Inc. 1996. Immediate Response Action Plan, RTN 4-11419. January 26.

Roy F. Weston, Inc. 1997. After Action Report for the New Bedford Industrial Park Removal Site, New Bedford, Massachusetts, 20 through 28 October 1997. December.

Comparison values (source organization, reference):
CREG = Cancer Risk Evaluation Guide for 1 x 10^{-6} excess cancer risk (ATSDR, ATSDR 2008a)
MassDEP S-1 & GW-1 = MCP Method 1 soil category S-1 standards applicable to soil where the combination of soil & groundwater categories are S-1 & GW-1 (MDEP, MDEP 2008)

Table 3
Toxics Release Inventory Data for the Former Polaroid Facility (1988-2006)

Chemical Name	Media	Year (Pounds)																		
		2006	2005	2004	2003	2002	2001	2000	1999	1998	1997	1996	1995	1994	1993	1992	1991	1990	1989	1988
Copper compounds	WATER*	NR†	NR	NR	NR	NR	NR	NR	NR	NR	2	2	2	0	0	0	NR	NR	NR	NR
Hydrochloric acid§	AIR STACK‡	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	1	1	1	1	1	1	1	0
Methanol	AIR FUG††	1500	4700	1800	1300	1900	400	700	600	410	440	500	380	132	100	100	100	100	100	250
Methanol	AIR STACK	12000	12000	18000	21000	15000	18000	29000	28000	28000	31200	32000	33000	39265	33000	36000	35000	44900	42700	42000
Methanol	DISP NON METALS‡‡	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	110	NR
Methyl ethyl ketone	AIR FUG	NR	NR	NR	3900	3200	5200	5500	5300	5400	5500	6300	3700	3587	1500	NR	NR	NR	NR	NR
Methyl ethyl ketone	AIR STACK	NR	NR	NR	1400	1000	2800	1700	2000	3100	3900	6600	5600	1450	80	NR	NR	NR	NR	NR
Silver compounds	DISP NON METALS	50	60	100	120	120	80	120	120	120	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR

* WATER = Discharges to receiving streams or water bodies
† NR = not reported
‡ AIR STACK = Stack or point air emissions
†† AIR FUG = Fugitive or non-point air emissions
‡‡ DISP NON METALS = Summation of a group of the methods that can be used to dispose of a metal or non-metal chemical off-site
§ 1995 and after "ACID AEROSOLS" only

Data source: Toxics Release Inventory (TRI) USEPA, 2008.

Table 4
Standard Hospitalization Ratio for Asthma
Zip Code 02745, New Bedford, Massachusetts
2000-2003

Time Period	Obs	Exp	SHR	95% CI
2000	17	31.2	54	* 32 — 87
2001	39	32.0	122	87 — 166
2002	40	31.3	128	91 — 174
2003	58	38.7	150	* 114 — 194
2000 - 2003	154	133.4	115	98 — 135

Notes: Asthma is the primary cause of hospitalizaiton.
SHRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

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

Data Source: MA Division of Health Care Finance and Policy (DHCFP).

Table 5
Cancer Incidence, CT 6501.01
New Bedford, Massachusetts
1996-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	6.1	98	36 -- 214	5	4.2	119	38 -- 278	1	1.9	NC	NC -- NC
Breast	19	27.5	69	42 -- 108	0	0.2	NC	NC -- NC	19	27.3	70	42 -- 109
Kidney and Renal Pelvis	8	4.1	197	85 -- 388	3	2.4	NC	NC -- NC	5	1.7	296	95 -- 691
Liver and Intrahepatic Bile Duct	3	1.6	NC	NC -- NC	2	1.0	NC	NC -- NC	1	0.5	NC	NC -- NC
Lung and Bronchus	27	25.3	107	70 -- 155	19	13.0	146	88 -- 229	8	12.3	65	28 -- 128
Prostate	22	26.1	84	53 -- 128	22	26.1	84	53 -- 128	0	NC	NC	NC -- NC

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

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

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

Table 6
Cancer Incidence, CT 6501.01
New Bedford, Massachusetts
2001-2005

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	4.7	128	47 -- 279	3	3.1	NC	NC -- NC	3	1.6	NC	NC -- NC
Breast	28	26.2	107	71 -- 155	0	0.2	NC	NC -- NC	28	26.0	108	72 -- 156
Kidney and Renal Pelvis	6	5.0	119	44 -- 260	4	2.9	NC	NC -- NC	2	2.1	NC	NC -- NC
Liver and Intrahepatic Bile Duct	4	2.0	NC	NC -- NC	3	1.4	NC	NC -- NC	1	0.6	NC	NC -- NC
Lung and Bronchus	29	26.5	109	73 -- 157	14	12.6	111	61 -- 187	15	13.9	108	60 -- 178
Prostate	31	24.9	125	85 -- 177	31	24.9	125	85 -- 177	0	NC	NC	NC -- NC

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

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

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

Table 7
Cancer Incidence, CT 6501.02
New Bedford, Massachusetts
1996-2000

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	3.2	NC	NC -- NC	2	2.4	NC	NC -- NC	2	0.8	NC	NC -- NC
Breast	14	16.0	88	48 -- 147	0	0.1	NC	NC -- NC	14	15.9	88	48 -- 148
Kidney and Renal Pelvis	4	2.5	NC	NC -- NC	4	1.5	NC	NC -- NC	0	0.9	NC	NC -- NC
Liver and Intrahepatic Bile Duct	0	0.9	NC	NC -- NC	0	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung and Bronchus	12	14.1	85	44 -- 148	10	7.9	127	61 -- 234	2	6.3	NC	NC -- NC
Prostate	13	16.3	80	42 -- 136	13	16.3	80	42 -- 136	0	NC	NC	NC -- NC

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

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

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

Table 8
Cancer Incidence, CT 6501.02
New Bedford, Massachusetts
2001-2005

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	2	2.4	NC	NC -- NC	1	1.7	NC	NC -- NC	1	0.7	NC	NC -- NC
Breast	15	15.9	94	53 -- 155	0	0.1	NC	NC -- NC	15	15.8	95	53 -- 156
Kidney and Renal Pelvis	4	3.1	NC	NC -- NC	2	1.9	NC	NC -- NC	2	1.2	NC	NC -- NC
Liver and Intrahepatic Bile Duct	2	1.2	NC	NC -- NC	1	0.9	NC	NC -- NC	1	0.3	NC	NC -- NC
Lung and Bronchus	14	14.9	94	51 -- 158	7	7.7	90	36 -- 186	7	7.1	98	39 -- 202
Prostate	19	16.3	116	70 -- 182	19	16.3	116	70 -- 182	0	NC	NC	NC -- NC

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

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

Data Source: Massachusetts Cancer Registry, 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 ON-SITE SURFACE SOIL AT THE FORMER POLYMERINE SITE

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Former Polymerine Site, New Bedford, 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 (Ingestion):

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

Cancer Effects Exposure Factor:

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

Cancer Effects Exposure Dose (Ingestion):

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

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)
$[C]_{soil}$	= 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 receptor evaluated was an older child who trespasses on the site.
2) The soil concentration was assumed as either the maximum or average concentration of copper and PCBs detected in on-site surface soil.
3) The amount of soil ingested was assumed to be 200 milligrams per day for the older child.
4) The exposure factor was determined assuming the older child receptor was exposed to site soil 1 day per week, for 22 weeks per year (May through September) over a 5 year period.
5) The average body weight of the older child receptor was assumed to be 45 kilograms.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Former Polymerine Site, New Bedford, Massachusetts

1. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Maximum Concentration of Copper by an Older Child Prior to Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\ \text{years}}{5\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{8{,}300\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.06 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.002\ \text{mg/kg/day}$$

NOTES:

1. The ATSDR Intermediate MRL for copper is 0.01 mg/kg/day.
2. The EPA has not classified copper with respect to its cancer causing potential and has not developed an EPA Oral Cancer Slope Factor for copper. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for copper.

2. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Average Concentration of PCBs by an Older Child Prior to Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\ \text{years}}{5\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{403\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.06 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.0001\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.004$$

$$\text{Cancer Effects Exposure Dose} = \frac{403\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.004 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.000007\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000007 \times 2 = 0.00001$$

NOTES:

1. The ATSDR Chronic MRL for Aroclor 1254 is 0.00002 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for PCBs is 2.0 mg/kg/day^{-1}.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Former Polymerine Site, New Bedford, Massachusetts

3. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Average Concentration of PCBs in the Area North of the On-Site Building by an Older Child After Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\text{ years}}{5\text{ years} \times 365\text{ days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{440\text{ mg/kg} \times 200\text{ mg/day} \times 0.06 \times 10^{-6}\text{ kg/mg}}{45\text{ kg}} = 0.0001\text{ mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\text{ years}}{70\text{ years} \times 365\text{ days}} = 0.004$$

$$\text{Cancer Effects Exposure Dose} = \frac{440\text{ mg/kg} \times 200\text{ mg/day} \times 0.004 \times 10^{-6}\text{ kg/mg}}{45\text{ kg}} = 0.000008\text{ mg/kg/day}$$

$$\text{Cancer Risk} = 0.000008 \times 2 = 0.00002$$

4. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Average Concentration of PCBs in the Area South of the On-Site Building by an Older Child After Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\text{ years}}{5\text{ years} \times 365\text{ days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{50\text{ mg/kg} \times 200\text{ mg/day} \times 0.06 \times 10^{-6}\text{ kg/mg}}{45\text{ kg}} = 0.00001\text{ mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week x 22 weeks/year} \times 5\text{ years}}{70\text{ years} \times 365\text{ days}} = 0.004$$

$$\text{Cancer Effects Exposure Dose} = \frac{50\text{ mg/kg} \times 200\text{ mg/day} \times 0.004 \times 10^{-6}\text{ kg/mg}}{45\text{ kg}} = 0.0000009\text{ mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000009 \times 2 = 0.000002$$

NOTES:

1. The ATSDR Chronic MRL for Aroclor 1254 is 0.00002 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for PCBs is 2.0 mg/kg/day^{-1}.

APPENDIX B: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA DERMAL CONTACT WITH ON-SITE SURFACE SOIL AT THE FORMER POLYMERINE SITE

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with On-Site Surface Soil Former Polymerine Site, New Bedford, 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 (Dermal Contact):

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

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)
$[C]_{soil}$	= 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 receptor evaluated was an older child who trespasses on the site.
2. The soil concentration was assumed as the average concentration of PCBs detected in the contaminated section of the site to reflect the range of contaminant concentrations that would likely have been contacted over time.
3. The exposure factor was determined assuming the older child receptor was exposed to site soil 1 day per week, for 22 weeks per year (May through September) over a 5 year period.
4. The average body weight of the older child receptor was assumed to be 45 kilograms.

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with On-Site Surface Soil Former Polymerine Site, New Bedford, Massachusetts

1. Exposure Dose and Cancer Risk Calculations for Dermal Contact with On-Site Surface Soil Containing the Average Concentration of PCBs by an Older Child Prior to Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weaks/year x 5 years}}{5\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{403\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.06}{45\ \text{kg}} = 0.00006\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weeks/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.004$$

$$\text{Cancer Health Effects Exposure Dose} = \frac{403\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\ \text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.004}{45\ \text{kg}} = 0.000004\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000004 \times 2 = 0.000008$$

2. Exposure Dose and Cancer Risk Calculations for Dermal Contact with On-Site Surface Soil Containing the Average Concentration of PCBs in the Area North of the On-Site Building by an Older Child After Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weaks/year x 5 years}}{5\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{440\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.06}{45\ \text{kg}} = 0.00007\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weeks/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.004$$

$$\text{Cancer Health Effects Exposure Dose} = \frac{440\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\ \text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.004}{45\ \text{kg}} = 0.000005\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000005 \times 2 = 0.00001$$

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with On-Site Surface Soil Former Polymerine Site, New Bedford, Massachusetts

3. Exposure Dose and Cancer Risk Calculations for Dermal Contact with On-Site Surface Soil Containing the Average Concentration of PCBs in the Area South of the On-Site Building by an Older Child After Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weaks/year x 5 years}}{5\ \text{years} \times 365\ \text{days}} = 0.06$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{50\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.06}{45\ \text{kg}} = 0.000008\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/week} \times 22\ \text{weeks/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.004$$

$$\text{Cancer Health Effects Exposure Dose} = \frac{50\ \text{mg/kg} \times 0.2\ \text{mg/cm}^2 \times 4100\ \text{cm}^2 \times 0.14 \times 10^{-6}\ \text{kg/mg} \times 0.004}{45\ \text{kg}} = 0.0000005\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000005 \times 2 = 0.000001$$

NOTES:

1. The ATSDR Chronic MRL for Aroclor 1254 is 0.00002 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for PCBs is 2.0 mg/kg/day^{-1}.

APPENDIX C: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA INGESTION OF ON-SITE SURFACE SOIL AT THE FORMER TALLYRAND SITE

APPENDIX C

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Former Tallyrand Site, New Bedford, 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 (Ingestion):

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

Cancer Effects Exposure Factor:

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

Cancer Effects Exposure Dose (Ingestion):

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

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)
$[C]_{soil}$	= Analyte Concentration in Surface 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 receptor evaluated was an older child who trespasses on the site.
2) The soil concentration was assumed as the average concentration of PCBs detected in surface soil in the contaminated section of the site.
3) The amount of soil ingested was assumed to be 200 milligrams per day for the older child.
4) The exposure factor was determined assuming the older child receptor was exposed to on-site soil 1 day per month, for 5 months per year (May through September) over a 5 year period.
5) The average body weight of the older child receptor was assumed to be 45 kilograms.

APPENDIX C

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Former Tallyrand Site, New Bedford, Massachusetts

1. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Average Concentration of PCBs by an Older Child Prior to Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/month x 5 months/year} \times 5\ \text{years}}{5\ \text{years} \times 365\ \text{days}} = 0.014$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{887\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.014 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.00005\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/month x 5 months/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.001$$

$$\text{Cancer Effects Exposure Dose} = \frac{887\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.001 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.000004\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000004 \times 2 = 0.000008$$

2. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing the Maximum Concentration of PCBs by an Older Child After Remediation:

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/month x 5 months/year} \times 5\ \text{years}}{5\ \text{years} \times 365\ \text{days}} = 0.014$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{10\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.014 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.0000006\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/month x 5 months/year} \times 5\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.001$$

$$\text{Cancer Effects Exposure Dose} = \frac{10\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.001 \times 10^{-6}\ \text{kg/mg}}{45\ \text{kg}} = 0.00000004\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000004 \times 2 = 0.00000008$$

NOTES:

1. The ATSDR Chronic MRL for Aroclor 1254 is 0.00002 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for PCBs is 2.0 mg/kg/day^{-1}.

APPENDIX D: EXPOSURE DOSE AND CANCER RISK CALCULATIONS FOR EXPOSURE VIA DERMAL CONTACT WITH ON-SITE SURFACE SOIL AT THE FORMER TALLYRAND SITE

APPENDIX D

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with On-Site Surface Soil Former Tallyrand Site, New Bedford, 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 (Dermal Contact):

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

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)
$[C]_{soil}$	= 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 receptor evaluated was an older child who trespasses on the site.
2. The soil concentration was assumed as the average concentration of PCBs detected in surface soil in the contaminated section of the site.
3. The exposure factor was determined assuming the older child receptor was exposed to on-site soil 1 day per month, for 22 months per year (May through September) over a 5 year period.
4. The average body weight of the older child receptor was assumed to be 45 kilograms.

APPENDIX D

Exposure Dose and Cancer Risk Calculations for Exposure via Dermal Contact with On-Site Surface Soil Former Tallyrand Site, New Bedford, Massachusetts

1. **Exposure Dose and Cancer Risk Calculations for Dermal Contact with On-Site Surface Soil Containing the Average Concentration of PCBs by an Older Child Prior to Remediation:**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/month} \times 5\,\text{months/year x 5 years}}{5\,\text{years} \times 365\,\text{days}} = 0.014$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{887\,\text{mg/kg} \times 0.2\,\text{mg/cm}^2 \times 4100\text{cm}^2 \times 0.14 \times 10^{-6}\,\text{kg/mg} \times 0.014}{45\,\text{kg}} = 0.00003\,\text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/month} \times 5\,\text{months/year} \times 5\,\text{years}}{70\,\text{years} \times 365\,\text{days}} = 0.001$$

$$\text{Cancer Health Effects Exposure Dose} = \frac{887\,\text{mg/kg} \times 0.2\,\text{mg/cm}^2 \times 4100\,\text{cm}^2 \times 0.14 \times 10^{-6}\,\text{kg/mg} \times 0.001}{45\,\text{kg}} = 0.000002\,\text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000002 \times 2 = 0.000004$$

2. **Exposure Dose and Cancer Risk Calculations for Dermal Contact with On-Site Surface Soil Containing the Average Concentration of PCBs by an Older Child After Remediation:**

$$\text{Noncancer Health Effects Exposure Factor} = \frac{1\,\text{day/month} \times 5\,\text{months/year x 5 years}}{5\,\text{years} \times 365\,\text{days}} = 0.014$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{10\,\text{mg/kg} \times 0.2\,\text{mg/cm}^2 \times 4100\text{cm}^2 \times 0.14 \times 10^{-6}\,\text{kg/mg} \times 0.014}{45\,\text{kg}} = 0.0000004\,\text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{1\,\text{day/month} \times 5\,\text{months/year} \times 5\,\text{years}}{70\,\text{years} \times 365\,\text{days}} = 0.001$$

$$\text{Cancer Health Effects Exposure Dose} = \frac{10\,\text{mg/kg} \times 0.2\,\text{mg/cm}^2 \times 4100\,\text{cm}^2 \times 0.14 \times 10^{-6}\,\text{kg/mg} \times 0.001}{45\,\text{kg}} = 0.00000003\,\text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000003 \times 2 = 0.00000006$$

NOTES:

1. The ATSDR Chronic MRL for Aroclor 1254 is 0.00002 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for PCBs is 2.0 mg/kg/day^{-1}.