Massachusetts
Department Of
Public Health

Health Consultation: Evaluation of Environmental Concerns and Lung and Bronchus Cancer Incidence, 1999–2005, in Rockland Census Tract 5022

Rockland, Plymouth County, Massachusetts

Suburban Auto of Rockland
MDEP RTN 4-0006043

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I. SUMMARY

Introduction: This health consultation was conducted because residents of Rockland, Massachusetts, were concerned about potential health risks related to soil and air contamination at the former Suburban Auto of Rockland facility prior to its remediation and suspected increases of cancer in nearby neighborhoods. Remediation efforts were completed in 2005, at which time a condominium complex was developed on the site.

Overview: MDPH has reached several important conclusions about the former Suburban Auto site in Rockland.

Conclusion 1: MDPH concluded that breathing dust or volatiles originating from the former Suburban Auto site in the past, present or future is not expected to result in health effects for nearby residents or children attending the adjacent daycare.

Basis for Decision: Due to the lack of historical ambient air monitoring data, the MDPH used U.S. Environmental Protection Agency (USEPA) soil screening levels (SSLs) for inhalation to evaluate potential past exposures of nearby residents to contaminants from breathing dust or volatiles originating from the former Suburban Auto site. With the exception of lead, levels of chemical contaminants detected in soil at the site are generally at or below USEPA SSLs for inhalation or typical background values indicating that concentrations are not expected to be of health concern for nearby residents.

Because the maximum concentration of lead was above the SSL and children living nearby or attending the adjacent daycare center may have been exposed via offsite deposition of lead in soil, the MDPH evaluated data on blood lead levels among children who resided within a one-quarter mile radius of the site between July 1992 and January 2006. The percentage of children living within one-quarter mile of the former Suburban Auto site with confirmed blood lead test results that were equal to or greater than 10 ug/dL (2.4%, n=84) was similar to that observed community-wide (2.5%, n=2,796). No unusual concentration of children with elevated blood lead levels was noted in the vicinity of the site. Therefore, it is unlikely that children who attended the daycare would have experienced elevated blood lead levels as a result of potential exposure to lead dust originating from the former Suburban Auto site.

Present and future exposures to contaminants at the former Suburban Auto site via inhalation of dust or volatiles are not expected because the contaminated soil was removed.

Conclusion 2: MDPH concluded that incidentally eating or touching soil, automotive shredder residue (ASR), or sediment while at the former Suburban Auto site in the past is not expected to result in health effects.

Basis for Decision: Past activities at the former Suburban Auto site resulted in chemical contaminants in the soil and sediment. Individuals, including employees, nearby residents, and trespassers, could have come into contact with chemical contaminants when on the site. Based on the available information and conservative assumptions about the frequency and duration of potential exposures, levels of chemical contaminants that could get into a child's or an adult's body are below levels that would result in adverse non-cancer or cancer health effects.

Conclusion 3: MDPH concluded that incidentally eating or touching soil, ASR, or sediment at the former Suburban Auto site presently or in the future is not expected to result in health effects.

Basis for Decision: Remediation at the former Suburban Auto site included the removal of contaminated soil, ASR, and sediment. Confirmatory sampling verified that contaminant concentrations were reduced to levels below the applicable standards. Therefore, present and future incidental ingestion of or dermal contact with contaminants in soil, ASR, or sediment by nearby residents or trespassers have been eliminated as exposure pathways.

Conclusion 4: MDPH concluded that health effects are not expected to result from drinking tap water or from volatilization of chemicals into buildings from underlying groundwater at the former Suburban Auto site in the past, present, or future.

Basis for Decision: Groundwater at the former Suburban Auto site was not used as a source of drinking water historically and is not used as a source presently. An on-site house used by a caretaker in the past and the condominiums located on the site presently are supplied with municipal drinking water. In addition, there are no municipal wells or known private drinking water wells in the vicinity or down gradient of the site. Therefore, ingestion of or dermal contact with contaminants in groundwater was eliminated as an exposure pathway in the past, present and future. Furthermore, exposure to contaminants via vapor intrusion of chemicals into buildings from underlying groundwater at the former Suburban Auto site was eliminated as a potential pathway since no compound was found to exceed the applicable state standard.

Conclusion 5: MDPH concluded that incidentally drinking or touching surface water at the former Suburban Auto site in the past, present, or future is not expected to result in health effects.

Basis for Decision: A small stormwater detention pond located on the former Suburban Auto site discharges to a tributary, which flows north to Whitman's Pond in Weymouth. During low precipitation, water from Whitman's Pond is pumped and treated before being supplied as drinking water for the town of Weymouth. Based on the available information, levels of chemical contaminants in surface water from the stormwater detention pond, stream and wetlands located on the former Suburban Auto site are below levels that would result in health effects.

Conclusion 6: MDPH concluded that although the incidence of lung and bronchus cancer is statistically significantly elevated within the census tract (CT) that contains the former Suburban Auto site during the 7-year time period 1999-2005, no unusual trends emerged when the overall age and gender patterns were examined in more detail.

Basis for Decision: To determine whether the incidence of lung and bronchus cancer in the CT containing the former Suburban Auto site (CT 5022) 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. Between 1999 and 2005, 57 diagnoses were reported in CT 5022 when approximately 43 would be expected. This elevation was statistically significant. Among males, the elevation was not statistically significant (25 observed diagnosed compared to about 21 expected). Among females, the elevation was of borderline statistical significance (32 observed diagnoses compared to 22 expected).

Smoking is, by far, the most important risk factor for lung and bronchus cancer. Among the 47 individuals with a known tobacco history, 45 (93%) were current or former smokers at the time of their diagnosis.

Exposure to radon (a naturally occurring radioactive gas produced by the breakdown of uranium in soils and rocks) has been identified as the second leading cause of lung and bronchus cancer, and the leading cause among nonsmokers. The USEPA has designated Plymouth County, where Rockland is located, as an area with moderate potential for indoor radon levels in homes to exceed the USEPA's recommended remediation level. Due to natural geologic variability, radon levels in a home cannot be predicted. Testing is the only way to determine the indoor radon level in a home.

Age at diagnosis, histology (cell type), and the temporal pattern of diagnoses were evaluated for those individuals diagnosed with lung and bronchus cancer in CT 5022 during 1999-2005. No unusual patterns emerged. In addition, MDPH evaluated the geographic distribution of residence at the time of diagnosis. The distribution was generally consistent with the population density. No unusual spatial pattern or

concentration of diagnoses was noted in the vicinity of the former Suburban Auto site or elsewhere in the CT.

Next Steps:

❖ The MDPH will continue to monitor the incidence of lung and bronchus cancer in the community of Rockland through city/town cancer reports published by the Massachusetts Cancer Registry.

❖ The MDPH recommends that Massachusetts residents test their houses for radon. Priority areas to place radon monitors are in the lower levels of a home, such as the basement. 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'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 Control Program at 1-800-Try-To-Stop or 1-800-879-8678.

For More Information: If you have concerns about your health, you should contact your health care provider. You may also call the MDPH at 617-624-5757 and ask for information on the former Suburban Auto site.

II. INTRODUCTION

At the request of the North Rockland Neighborhood Association, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health (BEH), conducted an evaluation of potential health impacts associated with the former Suburban Auto of Rockland facility (Suburban Auto)[1]. Suburban Auto operated until about 2003. The former Suburban Auto site is a closed release site that was regulated by the Massachusetts Department of Environmental Protection (MDEP). MDEP enforces regulations governing the investigation and cleanup of oil and hazardous material under Chapter 21E of Massachusetts General Laws, also known as the Massachusetts Contingency Plan (MCP) (M.G.L. c21E, 310 CRM 40.0000). After all remediation efforts were completed in 2005, a condominium development was built at the site. Community concerns prior to remediation of the site focused on potential health risks associated with possible environmental exposures including lung cancer in the neighborhood surrounding the former Suburban Auto site.

In two previous reports, the MDPH reviewed the incidence of several cancer types for Rockland as a whole and for Rockland's three census tracts (CTs). In *Assessment of Cancer Incidence in Rockland, Massachusetts, 1982-1994*, the MDPH found that while lung cancer was statistically significantly elevated among females in one CT of Rockland (CT 5021.01, located in central Rockland) during 1987-1994, the percent of those diagnosed who were current or former smokers was higher than that observed for the state as a whole (MDPH 2000a). In a larger study of four communities entitled *Assessment of Cancer Incidence in Weymouth, Abington, Hingham, and Rockland, Massachusetts 1982-1998*, the MDPH found that lung cancer was statistically significantly elevated among both males and females for the community of Rockland as a whole during 1995-1998 (MDPH 2002). In both assessments, all other cancer types that were evaluated occurred approximately as expected with no statistically significant elevations. This current investigation provides an updated review of the pattern of lung and bronchus cancer from 1999–2005 for Rockland CT 5022, the northernmost census tract where the former Suburban Auto site and nearby residential areas are located. Available information about risk factors, including environmental factors, related to the development of lung and bronchus cancer was also

[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

evaluated. Additionally, this investigation provides a review of potential exposure pathways to contaminants detected in environmental media at the former Suburban Auto site.

III. BACKGROUND

The town of Rockland is located 15 miles southeast of Boston, Massachusetts. The 14-acre site is located at 163 Forest Street and 200 VFW Drive in northern Rockland. Figure 1 depicts the site as it was configured when Suburban Auto of Rockland, Inc. operated on the property from 1997 until about 2003. Past operations by Suburban Auto of Rockland, Inc. and previous companies were carried out on Lot 86 and Lot 87 (C&C 1999). A gravel pit on adjacent Lot 79, which had a different owner, was once filled with automotive shredder residue (ASR) that was produced from shredding scrapped automobiles. Although Lot 79 was not used by Suburban Auto of Rockland, Inc., the three properties were surrounded by a continuous fence; therefore, Lots 79, 86 and 87 were evaluated collectively by the MDPH and will be referred to as "the former Suburban Auto site" in this health consultation.

A mix of residential and commercial properties border the former Suburban Auto site (Figure 1). An estimated 1,315 people live within a ½-mile radius (C&C 1999). Across Forest Street to the north are residences and wooded areas. Adjacent to the property on the east are residential properties, a daycare center, a cable television receiving station, and an auto body repair business. To the south, the site is abutted by an automotive repair business, skating rink, ice cream stand, and restaurant. Wooded wetlands exist on the western edge of the property and extend into the adjacent property where there is also a tributary to the Old Swamp River. Farther west, residential properties are located on Lincoln Road. The South Weymouth Naval Air Station, which encompasses approximately 1,442 acres and was closed in 1997, is located within 0.5 mile of the former Suburban Auto site to the north and west (USEPA 2010a).

From the late 1940s to the mid-1950s, an automobile repair business operated at the former Suburban Auto site. In the early 1970s, a pit was filled with ASR on Lot 79 (J. Hobill, MDEP, personal communication, 2006; Rizzo Associates 2005). From 1977 to 1997, the site was used as an automotive materials disposal area with metals recycling operations. Motor oil, gasoline, transmission fluids, anti-freeze, and lead acid storage batteries were likely stored on the property during that time (C&C 1999). In 1989, a fire involving automobile wreckage and approximately

1,000 tires occurred at the site (Goldsmith 1989). Following the fire, the site was entered into the MDEP spill location database due to a sheen that was observed on water leaving the site. In 1992, it was reported to MDEP that runoff from heavy rainfall was leaving the site with a sheen, and the presence of stockpiled soils on the site was also noted. In 1993, significant revisions to the MCP were promulgated and MDEP assigned the Release Tracking Number (RTN) 4-0006043 to the site in 1994 (C&C 1999; MDEP 2009a). Suburban Auto of Rockland, Inc. operated a metals recycling facility on the site from 1997 to about 2003, during which one of its employees acted as a caretaker of the property, living in a one-story house in the center of the site (C&C 1999, 2002; J. Hobill, MDEP, personal communication, 2001). Scrap metal products brought to the site were cut, sorted, and moved to metal stockpiles primarily located on the southern half of the site. Eventually, the recycled metals were transported off the property for resale. When Suburban Auto was open for business, the property was generally accessible via a set of gates on both the northern and southern boundaries (MDPH 2000b).

In 1997, 1,385 tons of stockpiled soil were removed and transported to a landfill. In 2004 and 2005, a total of 4,000 cubic yards of contaminated soil and sediment was excavated and removed from an area encompassing approximately 60,000 square feet in the southern portion of Lot 87 and the detention pond. Soil was removed to a depth of two to four feet below grade, with additional excavation in some smaller areas. Confirmatory sampling and additional excavation were performed until the MCP Method 1 standards for soil category S-1 (M.G.L. c21E, 310 CRM 40.0000) were met (Coneco 2005). In 2005, approximately 8,400 cubic yards of ASR were excavated and removed from an area encompassing 26,000 square feet in the center of Lot 79 and from underneath an adjacent offsite soil berm on the property to the east (Rizzo Associates 2005). A 12-foot high dust control barrier was erected about 5 feet from the daycare center fence and removal activities were conducted in January and February in order to minimize potential exposure of children to soil and ASR dust. Following excavation, results of confirmatory sampling verified that removal of ASR and contiguous soils resulted in the reduction of contaminant concentrations to levels below the MCP Method 1 standards for soil category S-1. Upon authorization by the MDEP, the excavated area was then filled with clean bank run gravel (Rizzo Associates 2005). After all remediation efforts were completed in 2005, the MDEP issued a Class A-2 Response Action Outcome (RAO) (MDEP 2009a). This indicates that remedial work was completed, a permanent solution has been achieved, and although oil and

hazardous material has not been reduced to background levels, no "activity and use limitations" are required to maintain a level of "no significant risk" of harm to health, safety, public welfare and the environment (MDEP 2007). Later in 2005, a condominium development called Boxberry Square was built at the former Suburban Auto site.

IV. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS AND HEALTH CONCERNS

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

In order to evaluate concerns about potential environmental exposures to contaminants from the former Suburban Auto site, the MDPH contacted the MDEP to obtain and review available environmental information. Sampling data were available for on-site soil, ASR, groundwater, surface water and sediment.

The maximum concentrations of contaminants detected in the various types of environmental media were identified and compared to health-based comparison values (ATSDR 2005, 2008a,c; USEPA 2010b; MDEP 2007, 2008). Comparison values are developed based on health

guidelines and assumed situations that represent conservative estimates of human exposure. Contaminant concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, contaminant concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a compound to impact one's health, it must not only be present in the environmental media, but one must also come in contact with it. Therefore, if a contaminant concentration is greater than the comparison value, the potential for exposure should be further evaluated (ATSDR 2005). Concentration levels that are considered typical or "background" were also used to analyze the environmental data from the former Suburban Auto site (ATSDR 1995, 2002, 2008; USGS 1984; MDEP 2002).

A. Exposure to Air

Residents were primarily concerned about possible past exposure to site contaminants via inhalation of dust when Suburban Auto of Rockland, Inc. occupied the site. It is possible that dust from contaminated soil may have been stirred up by past operations at the site when it was used for an automobile repairs business and later as an automotive materials disposal area with metals recycling. This dust could have migrated to nearby residences and the adjacent daycare facility.

Due to the lack of historical ambient air monitoring data, the MDPH used USEPA soil screening levels (SSLs) for inhalation to evaluate concerns about potential past exposures of nearby residents to contaminants from breathing volatiles and fugitive dusts originating from the former Suburban Auto site. SSLs are screening levels designed to consider potential health effects from inhaling, ingesting, or coming into contact with contaminated soils. The models and assumptions used to develop the SSLs for the inhalation pathway take into account soil-to-air volatilization factors, soil saturation limits, particulate emission factors and a dispersion model. SSLs for inhalation were compared to the maximum concentration of constituents detected in surface soil. Because volatile compounds can migrate from deeper soils to the surface, VOC concentrations in subsurface soil were also compared to SSLs (USEPA 1996, 2010b). A summary of the maximum concentrations detected at the former Suburban Auto site that exceed SSLs is presented in Table 1.

Contaminants detected in soil at a concentration less than the SSL are not likely to pose a health concern. However, contaminants detected in soil at concentrations above the SSL do not necessarily indicate that a health concern is present. In order for a compound to impact one's health, it must not only be present in the environmental media, but one must also come in contact with it at a sufficient dose and for a sufficient duration. It is important to note that the SSLs are conservative values and are based on an exposure assumption of a residential setting whereby contact with the contaminated environmental media is expected to occur on a regular, daily basis over a lifetime (USEPA 1996, ATSDR 2005).

Surface soil samples collected at the former Suburban Auto site in 1999 and 2001 contained levels of lead (maximum = 2,900 ppm) that exceed the USEPA SSLs for inhalation and typical background levels. No other constituent in surface soil exceeded its respective SSL. No SSLs are available for three PAHs [acenaphthylene, benzo(g,h,i)perylene, phenanthrene] detected on-site in surface soil and, hence, these were not further evaluated.

In humans, the main target for lead toxicity is the nervous system. Lead exposure is of most concern for young children because children exposed to lead, primarily due to the presence of lead paint in houses built before 1978, may experience neurological damage (including learning disabilities) and behavioral changes. Because the maximum concentration of lead was above the SSL and children living or visiting nearby may have been exposed via off-site deposition of lead in soil, the MDPH evaluated readily available data on blood lead levels among children living near the former Suburban Auto site. Data were obtained from the BEH Childhood Lead Poisoning Prevention Program (CLPPP). CLPPP was established for the prevention, screening, diagnosis, and treatment of lead poisoning in children residing in Massachusetts. The Massachusetts Lead Law requires that all children be tested for blood lead levels once between the ages of 9 months and 12 months, and again at the ages of 2 and 3 years (CLPPP 2010).

CLPPP blood lead level testing data for Massachusetts children include address information on the child's place of residence. Information about other places where a child spends time, such as a daycare, was not available; therefore, the MDPH was unable to evaluate blood lead levels in children who attended the daycare in the past when lead-contaminated surface soil existed at the former Suburban Auto site. Instead, the MDPH evaluated blood lead levels for children who

resided within a one-quarter mile radius of the site. Daily living near a site likely presents more exposure opportunities than attending a daycare facility but not living nearby.

The time period July 1992 - January 2006 represents the period for which CLPPP blood lead level testing data were readily available prior to the construction of condominiums at the site. The percentage of children (under 36 months) living within one-quarter mile of the site with confirmed blood lead test results equal to or greater than 10 µg/dL, which the CDC defines as a level of concern, was similar to that observed community-wide (ATSDR 2007). Specifically, 2.5% (n = 2,796) of all children in Rockland who had test results reported to CLPPP during the time period analyzed had confirmed blood lead levels equal to or greater than 10 µg/dL. During the same time period, 2.4% (n = 84) of children residing within a one-quarter mile radius of the former Suburban Auto site who had test results reported to CLPPP had confirmed blood lead levels equal to or greater than 10 µg/dL. These percentages are both lower than that of children state-wide where 4.6% of children who had test results reported to CLPPP during this time period had confirmed blood lead levels equal to or greater than 10 µg/dL.

Furthermore, there was no geographic pattern of higher blood lead levels closer to the site during this time period that would indicate that exposure to lead from the site resulted in adverse health effects. Also, since there was no unusual pattern among children living nearby, it is unlikely that children who attended the daycare would have experienced elevated blood lead levels as they would spend less time and have lower opportunity for exposure to lead from the site.

To minimize potential exposure of children at the nearby daycare center to fugitive dust from remediation activities, excavation and removal of soil and ASR at the former Suburban Auto site in 2005 was conducted during the winter months of January to March when children were likely to be indoors. In addition, continuous air monitoring for particulate matter was conducted both on-site and at a downwind location at the fence-line adjacent to the daycare center. No exceedances of the applicable dust action levels were observed at any point during the excavation and removal activities (Rizzo Associates 2005).

Present and future exposures to contaminants at the former Suburban Auto site via inhalation of dust or volatiles are not expected because the contaminated soil was removed.

Residents also expressed concern about possible exposure to emissions from a fire that occurred at the site in 1989 and involved wrecked automobiles and approximately 1,000 tires (C&C 1999; Goldsmith 1989). Exposure to contaminants in dust and smoke could have been possible in the past but air monitoring data were not available to evaluate the potential for adverse health effects that could result from possible exposure. It is unlikely, however, that a one-time exposure to possible contaminants in the smoke from the fire would substantially increase cancer risk.

Because the extent of exposure opportunities to other ambient air emissions from the site is not known, the MDPH examined the geographic pattern of lung and bronchus cancer among individuals living in the neighborhood to assess whether any unusual patterns might be evident in relation to the site. This evaluation of cancer incidence is discussed in Section V of this report.

B. Exposure to Soil

Surface soil samples collected at the former Suburban Auto site in 1999 and 2001 contained levels of cadmium, lead, mercury, polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs) [acenaphthylene, benzo(a)pyrene, dibenzo(a,h)anthracene and phenanthrene] that exceed both soil comparison values and typical background levels (C&C 1999, 2002). Subsurface soil samples collected in 1999, 2001, 2004 and 2005 revealed levels of antimony, cadmium, chromium, lead, PCBs, PAHs [benzo(a)pyrene and dibenzo(a,h)anthracene] that also exceed both soil comparison values and typical background levels (C&C 1999, 2002; Coneco 2005). A summary of the maximum concentration of contaminants detected in on-site surface soil samples that exceed comparison values is presented in Table 2 and those of subsurface soil samples are provided in Table 3. The highest levels of these contaminants were detected in isolated samples, indicating that the contamination was generally limited to the southern portion of the site and was not widespread across the entire site.

Prior to remediation of the former Suburban Auto site, it is possible that employees, including the caretaker could have been exposed via incidental ingestion of or dermal contact with contaminated surface soil. The caretaker was an adult individual who lived in a one-story house located in the center of the site. It is assumed that the on-site caretaker would have had greater opportunities for exposure to constituents in surface soil than other employees. Under the conservative assumption that the caretaker incidentally ingested surface soil with the maximum

concentration of PCBs detected at the site for 7 days/week for 50 weeks over 30 years, the estimated exposure would not result in an unusual cancer risk or adverse noncancer health effects. The same exposure conditions for cadmium and mercury would also be unlikely to result in adverse noncancer health effects. If the caretaker had incidentally ingested surface soil containing the maximum concentration of PAHs [acenaphthylene, benzo(a)pyrene, dibenzo(a,h)anthracene, phenanthrene] detected on-site under the same exposure conditions described above, adverse noncancer health effects would not be expected. Toxicity equivalence in terms of benzo(a)pyrene was calculated for the two carcinogenic PAHs [dibenzo(a,h)anthracene, benzo(a)pyrene] that exceed both background values and soil comparison values by multiplying the concentration of each by its corresponding toxicity equivalency factor in terms of benzo(a)pyrene (MDEP 1995). The maximum benzo(a)pyrene equivalency for surface soil was 57.5 milligrams per kilogram (mg/kg). If the caretaker had incidentally ingested surface soil containing the maximum concentration of carcinogenic PAHs detected on-site under the same exposure conditions described above, the caretaker could have been exposed to PAHs at a level that could have presented low increased cancer risk. These exposure assumptions are conservative and represent a worst-case scenario. Under a more reasonable scenario that uses the average benzo(a)pyrene equivalency (19.9 mg/kg) to reflect the range of contaminant concentrations that would likely have been ingested over time, an unusual cancer risk would not be expected. See Appendix A for more information on the exposure dose and cancer risk calculations. Exposure to contaminated subsurface soil is not expected due to its depth below ground surface.

Although evidence of trespassing was not observed and it is unlikely that trespassing would have occurred while employees were present, it is possible that older children might have accessed the site either through the gates or breaks in the fence prior to remediation activities. As a result, trespassers may have been exposed via incidental ingestion of or dermal contact with contaminants detected in on-site surface soil 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 for a comparable frequency (e.g., 365 days per year) and duration of time (e.g., 30 years). Assuming that an older child who trespassed incidentally ingested the maximum contaminant concentration of PCBs or PAHs detected in on-site surface soil for 2 days/week for 26 weeks over 10 years, no unusual cancer risk or adverse noncancer health effects would be expected. It would also be unlikely that

such exposure to the maximum contaminant concentration of cadmium or mercury would result in adverse noncancer health effects. See Appendix A for more information on the exposure dose and cancer risk calculations. As mentioned previously, exposure to contaminated subsurface soil is not expected due to its depth below ground surface.

In order to evaluate potential health concerns related to exposure opportunities to lead in surface soil at the former Suburban Auto site, the MDPH used the U.S. EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children. This model is widely used throughout the country to predict blood lead levels based on lead intake via various sources (e.g., soil, food, water). Environmental data specific to a given scenario are input into the model in order to predict blood lead levels for children aged 6 months to 7 years. The IEUBK model generally uses typical or average concentrations for various source media, assumes daily exposures, and predicts blood lead concentrations based on chronic exposures (e.g., 1 year or more).

At the former Suburban Auto site, the average lead concentration detected in surface soil was 836 ppm. To be conservative, the MDPH ran the IEUBK model with the assumption that one-quarter of a child's typical daily incidental soil ingestion occurred while at the former Suburban Auto site, even though it is unlikely that children aged 6 months to 7 years would have trespassed on a daily basis at the site. Under this assumption, the predicted mean blood lead concentration of children aged 6 months to 7 years who hypothetically played at the former Suburban Auto site was 4.1 micrograms per deciliter (µg/dL). The IEUBK model predicted that 3% of this hypothetical population of children would have blood lead levels greater than 10 µg/dL, which the Centers for Disease Control and Prevention (CDC) define as a level of concern (ATSDR 2007). The prediction of a 3% risk of blood lead levels above 10 µg/dL is below the U.S. EPA Office of Solid Waste and Emergency Response's specified level of protectiveness of no more than a 5% risk of an elevated blood lead level for a given scenario (USEPA 2002). Thus, it appears unlikely that young children playing at the former Suburban Auto site would have had blood lead levels above the CDC level of concern given the exposure assumptions specified above.

As previously mentioned, contaminated soil at the former Suburban Auto site was removed during MDEP-approved cleanup activities in 2004 and 2005. Soil was removed to a depth of

two to four feet below grade in an area encompassing approximately 60,000 square feet in the southern portion of Lot 87. Confirmatory sampling and additional excavation were performed until the MCP Method 1 standards for soil category S-1 were met. Additional excavation was required in some areas, with excavation to bedrock in one location (Coneco 2005). As a result, present and future ingestion of or dermal contact with contaminants in on-site soil by nearby residents or trespassers have been eliminated as exposure pathways.

C. Exposure to Automotive Shredder Residue (ASR)

The ASR located on-site, which consisted of crushed automobile parts, metal, tire and rubber, was generally covered by surface soil but was exposed on the surface in some areas (C&C 1999; J. Hobill, MDEP, personal communication, 2001). Samples of ASR collected in 1999 and 2001 contained levels of cadmium, lead, PCBs, and semi-volatile organic compounds (SVOCs) [di(2-ethylhexyl)phthalate] that exceed both soil comparison values and typical background levels (RSB 1999; Kaegael Environmental 2001). A summary of the maximum concentration of contaminants detected in on-site ASR that exceed soil comparison values is provided in Table 4.

Since ASR was exposed in some areas of the site prior to remediation, it is possible that the caretaker, employees, or trespassing older children may have been exposed via incidental ingestion or dermal contact. Under the same exposure conditions assumed previously, no unusual cancer risk or noncancer health effects would be expected from exposure of the caretaker, an employee or a trespassing older child via incidental ingestion of or dermal contact with the maximum concentration of cadmium, lead, PCBs or SVOCs detected on-site in ASR. See Appendix B for more information on the exposure dose and cancer risk calculations. It should be noted that the estimated exposure dose for the caretaker from incidentally ingesting ASR with the maximum concentration of PCBs (0.000027 mg/kg/day) is greater than the ATSDR Chronic Minimal Risk Level (MRL) (0.00002 mg/kg/day). The MRL is an estimate of daily exposure to a contaminant below which adverse noncancer health outcomes are unlikely to occur. The level of exposure for the caretaker would be 200 times lower than the Lowest Observed Adverse Effect Level (LOAEL) used to derive the MRL (0.005 mg/kg/day) (ATSDR 2000). Therefore, noncancer health effects in the caretaker would not necessarily be expected as a result of exposure to PCBs via incidental ingestion of ASR.

As previously mentioned, approximately 8,400 cubic yards of ASR were excavated and removed from an area encompassing 26,000 square feet in the center of Lot 79 and from underneath an adjacent offsite soil berm on the property to the east during MDEP-approved cleanup activities in 2005. Following excavation, results of confirmatory sampling verified that removal of ASR and contiguous soils resulted in the reduction of contaminant concentrations to levels below the MCP Method 1 standards for soil category S-1. Upon authorization by MDEP, the excavated area was then filled with clean bank run gravel (Rizzo Associates 2005). As a result, present and future incidental ingestion of or dermal contact with contaminants in ASR by nearby residents or trespassers have been eliminated as exposure pathways.

D. Exposure to Groundwater

Groundwater samples were collected from monitoring wells in 1999, 2001, 2004 and 2005 (C&C 1999, 2002; Coneco 2005; Rizzo Associates 2005). Because ATSDR comparison values do not exist for groundwater, drinking water comparison values were used for screening purposes. Tetrachloroethylene (PCE), trichloroethylene (TCE) and vinyl chloride were detected at concentrations in at least one sample that exceed the comparison values. The maximum concentrations of these compounds that were detected are provided in Table 5.

The groundwater wells sampled at the site were for monitoring purposes only; no one drank from these wells. The on-site house was supplied with municipal drinking water. According to the Rockland Board of Health, there are no municipal wells or known private drinking water wells in the vicinity or down gradient of the site (C&C 1999). Drinking water for the Boxberry Square condominiums is provided by Rockland municipal water (Town of Rockland, Water Department, personal communication, 2008). Because groundwater in this area is not used as a source of drinking water, ingestion of or dermal contact with contaminated groundwater was eliminated as an exposure pathway for on-site employees and nearby residents in the past, present and future.

Vapor intrusion, which involves the volatilization of chemicals from groundwater through soil and into the indoor air of a building located above the groundwater, was also eliminated as an exposure pathway for on-site employees and nearby residents of the former Suburban Auto site in the past, present and future. This is based on findings of the Phase II site assessment that a condition of "no significant risk" exists for groundwater at the former Suburban Auto site.

Groundwater monitoring at the site indicated that no individual compound, including volatile organic compounds (VOCs) and metals, exceed an applicable MDEP Method 1 standard for groundwater categories GW-1, GW-2 or GW-3 in any monitoring well (C&C 2002). Groundwater is subject to GW-2 standards [310 CMR 40.0932] if it is located within 30 feet of an existing or planned building or structure that is or will be occupied, and the average annual depth to ground water in that area is 15 feet or less. Such groundwater is considered to be a potential source of vapors of oil and/or hazardous material to indoor air (MDEP 2007).

E. Exposure to Surface Water

A small stormwater detention pond located on the western part of the former Suburban Auto site discharges to a tributary of the Old Swamp River during heavy precipitation. This tributary flows north to Whitman's Pond in Weymouth, Massachusetts (C&C 1999). During low precipitation, water from Whitman's Pond is pumped into Great Pond and treated before being supplied as drinking water for the town of Weymouth (Weymouth Water Division 2004).

Surface water samples were collected from the stormwater detention pond, stream and wetlands in 1999 and 2001 (C&C 1999, 2002; Rizzo Associates 2005). Because ATSDR comparison values do not exist for surface water, drinking water comparison values were used for screening purposes. This is a conservative approach as exposures to contaminants in surface water that is not used for drinking water are expected to be less than exposures to those in drinking water. No constituent was found to exceed drinking water comparison values, and hence no adverse health effects due to this potential exposure pathway would be expected.

F. Exposure to Sediment

Sediment samples were collected in 1999, 2001 and 2004 from the stormwater detention pond, stream and wetlands (C&C 1999, 2002; Coneco 2005). Because ATSDR comparison values do not exist for sediment, soil comparison values were used for screening purposes. Cadmium, lead, PCBs, and PAHs [benzo(a)pyrene] were detected at concentrations that exceed both the soil comparison values and typical background levels. The maximum concentrations of contaminants that exceed soil comparison values are presented in Table 6.

Prior to remediation, it is possible that the caretaker, employers or trespassing older children may have been exposed though incidental ingestion of and dermal contact with contaminants in sediment. However, the maximum concentrations of cadmium, lead, PCBs, and PAHs [benzo(a)pyrene] detected in sediment were below that of on-site surface soil. Since it was previously determined that an unusual cancer risk or adverse noncancer health effects were unlikely to occur due to potential past exposure to the same contaminants in surface soil, adverse health effects would also be unlikely to occur due to potential exposure to lower levels of the contaminants in sediment for which the extent of potential exposure would likely be less.

As previously mentioned, some sediment in the stormwater detention pond was removed during MDEP-approved cleanup activities in 2004 and 2005. Confirmatory sampling and additional excavation were performed until the MCP Method 1 standards for soil category S-1 (M.G.L. c21E, 310 CRM 40.0000) were met (Coneco 2005). As a result, present and future ingestion of or dermal contact with contaminants in on-site sediment by nearby residents or trespassers have been eliminated as exposure pathways.

V. ANALYSIS OF CANCER INCIDENCE

A census tract (CT) is a smaller geographic subdivision of a city or town that is designated by the U.S. Census Bureau. Because age group and gender-specific population information are necessary to calculate cancer incidence rates, the CT is the smallest geographic area for which cancer rates can be accurately calculated. The town of Rockland is divided into three CTs. The former Suburban Auto site is located in CT 5022, which has an approximate area of 4.65 square miles and a population of 5,892 (U.S. Census Bureau 2000). The location and boundaries of CT 5022 in relation to the former Suburban Auto site are shown in Figure 2. In order to address community concerns and provide an update to two previous reports that recommended continuing to monitor the incidence of lung and bronchus cancer in Rockland, incidence rates for lung and bronchus cancer were calculated for CT 5022 for the years 1999–2005. This is the time

period for which the most recent and complete cancer incidence data were available from the Massachusetts Cancer Registry (MCR) at the initiation of this analysis[2].

A. Methods for Analyzing Cancer Incidence

1. Case Identification/Definition

As part of this investigation, the CAP reviewed incidence data available from the MCR for lung and bronchus cancer in Rockland CT 5022. This cancer type was selected for evaluation based on the conclusions and recommendations of earlier assessments completed by the MDPH (MDPH 2000a, 2002). The 7-year period from 1999-2005 constituted the most recent and complete cancer incidence data that were available at the time of this report. The MCR is a division within the MDPH Bureau of Health Information, Statistics, Research, and Evaluation. It is a population-based surveillance system that has been monitoring cancer incidence in the Commonwealth since 1982. All new diagnoses of invasive cancer, as well as certain in situ (localized) cancers, among Massachusetts residents are required by law to be reported to the MCR within 6 months of the date of diagnosis (M.G.L. c.111. s 111b). The MCR also gathers 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). Cancers that occur as the result of the metastasis, or the spread of a primary site cancer to another location in the body, are not considered as separate cancers and, therefore, were not included in this analysis.

[2] The data summarized in this report are drawn from data entered into the MCR before December 22, 2008. 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.

It should be noted that the MCR research file may contain duplicate reports of individuals diagnosed with cancer. Duplicate cases are additional reports of the same primary site cancer case. In Rockland, one duplicate report was identified during the years 1999–2005 and excluded from the analysis. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology 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 were removed from the analysis whereas individuals who were diagnosed with more than one primary site cancer were included as separate cases.

2. Calculation and Interpretation of a Standardized Incidence Ratio (SIR)

To assess the incidence of lung and bronchus cancer in Rockland CT 5022, a statistic called the standardized incidence ratio (SIR) was 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 were applied to the population distribution of the CT to calculate the number of expected cancer diagnoses. The SIR is a comparison of the number of diagnoses in the CT to the number of expected diagnoses based on the statewide rate.

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.

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 have a 95% probability of including the true SIR for the population. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or "normal" population. "Significantly different" means there is less than a 5% 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 was not assessed when fewer than five diagnoses were observed.

3. Evaluation of Cancer Risk Factor Information

As previously mentioned, cancer is not just one disease but a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different risk factors. One or even several factors acting over time can be related to the development of cancer. Information related to risk factors for cancer development is collected for each individual at the time of cancer diagnosis and reported to the MCR. This information includes age at diagnosis, stage of disease, tobacco history and occupation. The available risk factor information from the MCR was reviewed for residents of Rockland CT 5022 diagnosed with lung and bronchus cancer during 1999-2005 and compared to known or established incidence patterns for these cancer types. However, information about personal risk factors such as family history, medical history, 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 development in this investigation.

4. Determination of Geographic Distribution of Cancer Diagnoses

Address at the time of diagnosis was mapped for each individual diagnosed with lung and bronchus cancer in Rockland from 1999 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 in CT 5022.

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.

B. Results of Cancer Incidence Analysis

1. Census Tract (CT) 5022

Table 6 summarizes the incidence of lung and bronchus cancer for one census tract (CT 5022) in the community of Rockland during the seven-year time period of 1999-2005. Overall, an increased incidence of lung and bronchus cancer was observed for residents of this CT. There were 57 diagnoses of lung and bronchus cancer in CT 5022 when approximately 43 would have been expected to occur. This elevation in lung and bronchus cancer incidence was statistically significant (SIR=133, 95% CI=101-173). A separate evaluation by gender revealed that both males and females experienced an elevation in lung and bronchus cancer incidence; however, the statistical significance of the overall elevation is largely due to the excess observed among females. The elevation in lung and bronchus cancer incidence among males was not statistically significant (25 observed versus 20.7 expected, SIR=121, 95% CI=78-178) whereas that of females was of borderline statistical significance (32 observed versus 22.0 expected, SIR=146, 95% CI=100-206). There was no apparent temporal clustering of diagnoses and the number of diagnoses fluctuated from year to year, ranging from five to thirteen diagnoses in any year (Figure 3).

2. Review of Cancer Risk Factor Information

Information available from the MCR related to age and gender patterns, as well as other factors related to the development of lung and bronchus cancer such as tobacco history and occupation, was reviewed. The strongest established risk factor for lung cancer is tobacco use, followed by occupational and environmental exposures. More complete risk factor information for lung and bronchus cancer is included in Appendix C.

a) Age and Gender Distribution

According to the American Cancer Society, about 2 out of 3 people diagnosed with lung and bronchus cancer are older than 65 years of age and fewer than 3% of all diagnoses occur in

individuals under the age of 45 (ACS 2008a,b). In CT 5022, approximately 81% (n=46) of individuals diagnosed with lung and bronchus cancer in the time period of 1999-2005 were 65 years of age or older at the time of diagnosis. The average age at diagnosis was 71 years old with a range of 41 to 90 years of age. The average age at diagnosis for both the nation and the state of Massachusetts is also 71 years.

Slightly more females (56%, n=32) than males (44%, n=25) were diagnosed in CT 5022 with lung and bronchus cancer during this time period. A review of individuals diagnosed with lung and bronchus cancer in Massachusetts in the time period 1999-2005 revealed that diagnoses were more evenly distributed between males (51%) and females (49%) (MCR 2008).

b) Tobacco Use

Tobacco use is by far the most important risk factor for lung and bronchus cancer. It is estimated that 85% to 90% 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 cancer. If an individual stops smoking before a cancer develops, the damaged lung tissue gradually repairs itself. No matter the age of an individual or how long someone has used tobacco, quitting may help an individual to live longer (ACS 2008a,b).

Of the 47 individuals diagnosed with lung and bronchus cancer in CT 5022 during the time period 1999-2005 with a known tobacco history, about 96% (n=45) were current or former tobacco users. On a statewide level, about 93% of individuals in Massachusetts that were diagnosed with lung and bronchus cancer during the same time period and had a known tobacco history were current or former tobacco users.

c) Histology (Cell Type)

Lung and bronchus cancers are often divided into two main types: small cell lung cancer and non-small cell lung cancer. Non-small cell lung cancer is further sub-divided into three types: adenocarcinoma, squamous cell carcinoma, and large cell undifferentiated carcinoma. The different types of lung and bronchus cancer occur with different frequencies in the population. The American Cancer Society estimates that approximately 40% of all lung and bronchus

cancers are adenocarcinomas, 25% to 30% are squamous cell carcinomas, 10% to 15% are large cell undifferentiated carcinomas and 10% to 15% are small cell lung cancer (ACS 2008a,b).

An expected histologic distribution pattern was observed in CT 5022 where lung and bronchus cancer was statistically significantly elevated among males and females combined between 1999 and 2005. In CT 5022, adenocarcinoma was the most common histologic type of lung and bronchus cancer reported to the MCR (about 42% of those lung and bronchus cancers with specific histology classification), followed by squamous cell carcinoma (about 19%), large cell carcinoma (about 17%) and small cell carcinoma (about 14%).

d) Occupational and Environmental Exposures

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 Plymouth County have moderate potential for elevated radon levels (USEPA 2009). However, radon levels cannot be 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 is the only way to determine 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 compounds is even higher for smokers.

Occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with lung and bronchus cancer in CT 5022 in the time period 1999-2005 to determine the role that occupational factors may have played in the development of these cancers in Rockland. 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 5022, about 7% (n=4) of individuals diagnosed with lung or bronchus cancer during 1999-2005 reported working in jobs possibly associated with an increased risk of lung and bronchus cancer. Occupation was reported as unknown, retired or at home for about half (51%, n=29) of the individuals.

3. Geographic Distribution

The geographic distribution of the 57 lung and bronchus cancer diagnoses in CT 5022 during 1999-2005 was generally consistent with the population distribution in the CT. Seven of the individuals lived within a ½-mile radius of the former Suburban Auto site at the time of their diagnosis. No unusual spatial pattern or concentration of diagnoses was noted in the vicinity of the site or elsewhere in the CT. MDPH also evaluated the geographic distribution of residence at diagnosis for those 12 individuals with lung and bronchus cancer who either had never used tobacco or had an unknown tobacco history and found no unusual spatial patterns. The point pattern of diagnoses for this group closely followed the population density of the community.

VI. DISCUSSION

At the request of the North Rockland Neighborhood Association, the MDPH conducted an evaluation of possible environmental exposures in relation to the former Suburban Auto site in Rockland. Community concerns focused on the incidence of cancer in the neighborhood surrounding the former Suburban Auto site and potential exposures to contaminants detected on the site as well as fugitive emissions of site related contaminants in nearby areas. Since the 1940s, there has been an automobile repair business, an automotive materials disposal area, and metals recycling operations at the site (C&C 1999). After remediation efforts were completed in 2005, a condominium development called Boxberry Square was built at the site.

As part of this health consultation, the MDPH evaluated lung and bronchus cancer incidence data for CT 5022 in the community of Rockland and reviewed available environmental information for the former Suburban Auto site to determine possible pathways of exposure for the former caretaker, former employees, potential trespassers, and nearby residents. In addition, the pattern of lung and bronchus cancer was evaluated in neighborhoods within CT 5022 to identify any unusual concentrations of diagnoses.

There are some potential exposure pathways that may have existed in the past relative to the former Suburban Auto site. Past exposure to cadmium, lead, and PCBs in on-site surface soil, ASR, and sediment could have been possible for the former caretaker, former employees, and trespassers. In addition, these individuals could have also been exposed to PAHs in on-site surface soil and sediment, mercury in surface soil and SVOCs in ASR. Furthermore, former employees could have been exposed to contaminants such as antimony, cadmium, chromium, lead, PAHs and PCBs in subsurface soil. However, upon considering conservative exposure assumptions for the former caretaker, a former employee, and a child trespasser, no adverse health effects or unusual cancer risk due to contamination in on-site soil, ASR, and sediment were expected. It is unlikely that any of these individuals would have had contact with the contaminated media for sufficient frequency and duration to result in health effects.

Remediation at the former Suburban Auto site included the removal of a total of 4,000 cubic yards of contaminated soil and sediment. Soil was excavated to a depth of two to four feet below grade and removed from an area encompassing approximately 60,000 square feet in the southern portion of Lot 87. Additional excavation to deeper depths was necessary in some smaller areas (Conceco 2005). Sediment was excavated and removed from the stormwater detention pond. Remediation at the former Suburban Auto site also included the excavation and removal of 8,400 cubic yards of ASR from an area encompassing 26,000 square feet in the center of Lot 79 and from underneath an adjacent offsite soil berm on the property to the east. The excavated area was filled with clean bank run gravel (Rizzo Associates 2005). Confirmatory sampling verified that removal of contaminated soil, sediment and ASR resulted in the reduction of contaminant concentrations to levels below the MCP Method 1 standards for soil category S-1 (Coneco 2005, Rizzo Associates 2005). Therefore, present and future exposures to contaminants by nearby residents or trespassers have been eliminated as pathways.

Past, present, and future pathways of exposure to contaminants in groundwater and surface water at the former Suburban Auto site were eliminated. Although PCE, TCE and vinyl chloride were detected in groundwater at levels exceeding comparison values for drinking water, exposure is unlikely as the groundwater was not and is not used as a source of drinking water. No contaminant detected in surface water exceeds drinking water comparison values; therefore, adverse health effects would not be expected if exposure occurred. Furthermore, past, present, and future pathways of exposure to contaminants via vapor intrusion of chemicals into buildings from underlying groundwater at the former Suburban Auto site were eliminated as no compound was found to exceed the applicable MCP Method 1 standards for groundwater category GW-2.

Past exposures from breathing dust and volatiles originating from contaminated soil at the former Suburban Auto site could have been possible for nearby residents and children attending the adjacent daycare. With the exception of lead, levels of contaminants detected in soil at the former Suburban Auto site are generally at or below USEPA SSLs or typical background values indicating that concentrations are not expected to be of health concern for nearby residents. Based on data obtained from the BEH CLPPP, it is unlikely that children who attended the daycare would have experienced elevated blood lead levels as a result of potential exposure to lead dust originating from the former Suburban Auto site. Between July 1992 and January 2006, the percentage of children living within one-quarter mile radius of the site with blood lead test results equal to or greater than 10 ug/dL (2.4%, n=84) was similar to that observed community-wide (2.5%, n=2,796). In addition, no unusual concentration of children with elevated blood lead levels was noted in the vicinity of the former Suburban Auto site. Present and future exposures to contaminants at the former Suburban Auto site via inhalation of dust or volatiles are not expected because the contaminated soil was removed.

The former caretaker, former employees, and nearby residents may have also been exposed to contaminants via inhalation of smoke from a fire involving tires and wrecked automobiles that occurred at the former Suburban Auto site in 1989. Because air sampling was not performed during the fire, the potential for these individuals to be exposed to possible contaminants in the smoke could not be evaluated. However, it is unlikely that a single exposure to possible contaminants in smoke would result in long-term health effects including unusual cancer risks.

As part of this investigation, the CAP reviewed incidence data available from the MCR for lung and bronchus cancer in CT 5022 during the 7-year time period, 1999-2005. This cancer type was selected for evaluation based on community concerns as well as the conclusions and recommendations of earlier assessments completed by the MDPH (MDPH 2000a, 2002). In particular, it was recommended that the incidence of lung and bronchus cancer in Rockland continue to be monitored because it was statistically significantly elevated during the periods 1982-1994 and 1995-1998. In addition, the geographic pattern of lung and bronchus cancer among individuals living near the former Suburban Auto site was examined to assess whether any unusual patterns might be evident in relation to the site.

As with earlier time periods, CT 5022 experienced a statistically significant elevation in the incidence of lung and bronchus cancer during 1999-2005. Both males and females experienced an elevation in lung and bronchus cancer incidence; however, the elevation among males was not statistically significant and that of females was of borderline statistical significance. When the overall age and gender patterns of lung and bronchus cancer incidence were examined in more detail, no unusual trends emerged. Approximately 81% of individuals were 65 years of age or older at the time of diagnosis and the average age at diagnosis was 71 years old.

Available risk factor information on tobacco use and occupation for those diagnosed with lung and bronchus cancer during 1999-2005 was compared to known or established trends to assess whether any unexpected patterns emerged in Rockland CT 5022. Review of these data suggests that tobacco use likely played a role in the development of lung and bronchus cancer among some individuals. Forty-five of 47 individuals (96%) for whom tobacco history information was available were either current or former smokers (history of tobacco use was not available for 10 individuals). Also, occupational exposures may have been important in the development of lung and bronchus cancer among some individuals. However, because tobacco history and/or occupational information were not available for all individuals, it is difficult to fully assess the extent to which these factors influenced overall cancer patterns in CT 5022.

In addition, analysis of the geographic distribution of place of residence for individuals diagnosed with lung and bronchus cancer in CT 5022 did not reveal any atypical spatial patterns. That is, no apparent concentrations of individuals diagnosed with lung and bronchus cancer were

observed in the vicinity of the former Suburban Auto site that might suggest an association with a common environmental factor.

VII. LIMITATIONS

This health consultation is an investigation that analyzes descriptive health outcome data for cancer to determine whether the pattern or occurrence of lung and bronchus cancer in CT 5022 is unusual. The purpose of this investigation is to evaluate the pattern of lung and bronchus cancer in a geographical context in relation to available information about factors, including environmental factors, related to this cancer type to determine whether further investigation seems warranted. Information from descriptive analyses, which may suggest a common etiology (or cause) is possible, can serve to identify areas where further analyses may be needed. Inherent limitations in the available data and this type of analysis make it impossible to determine the precise casual relationships or synergistic roles that may have contributed to the development of individual cancers in this community. 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 etiology 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 are 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 lung and bronchus cancer in CT 5022.

VIII. CONCLUSIONS

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

- **Breathing dust or volatiles originating from contaminated soil at the former Suburban Auto site in the past** is not expected to have harmed the health of nearby residents or children attending the daycare center. The reason for this is because, based on the available information, levels of contaminants, with the exception of lead, are generally at or below USEPA SSLs for inhalation or typical background values indicating

that concentrations are below levels of health concern. Based on data obtained from the BEH CLPPP, it is unlikely that children who attended the daycare center would have experienced elevated blood lead levels as a result of potential exposure to lead dust originating from contaminated soil at the site given that no unusual elevations in blood lead levels were observed among children who lived in the adjacent neighborhood.

- **Breathing dust or volatiles originating from contaminated soil at the former Suburban Auto site presently or in the future** is not expected to harm the health of nearby residents or children attending the daycare center. The reason for this is because the contaminated soil was removed during remediation.

- **Incidentally eating or touching soil, ASR, or sediment while at the former Suburban Auto site 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 exposures, levels of chemical contaminants that could get into a child's or an adult's body are below levels that would harm their health.

- **Incidentally eating or touching soil, ASR, or sediment at the former Suburban Auto site presently or in the future** is not expected to harm people's health. The reason for this is because remediation at the site included the removal of contaminated soil, ASR, and sediment reducing contaminant concentrations to levels below the applicable standards.

- **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 site was not used as a source of drinking water historically and is not used as a source presently. In addition, volatilization of chemicals into buildings from underlying groundwater at the former Suburban Auto site in the past, present or future is not expected to harm people's health. The reason for this is because levels of contaminants did not exceed applicable standards.

- **Incidentally drinking or touching surface water at the former Suburban Auto site in the past, present or future** is not expected to harm people's health. The reason for this

is because, based on the available information, levels of chemical contaminants in surface water are below levels of health concern.

- **Within Rockland CT 5022, the incidence of lung and bronchus cancer** occurred above the expected rate during the 7-year time period, 1999-2005. This elevation was statistically significant. However, when the overall age and gender patterns of lung and bronchus cancer incidence were examined in more detail, no unusual trends emerged. The elevation among males was not statistically significant and that among females was borderline statistically significant. The ages at diagnosis for individuals in CT 5022 were consistent with state and national trends. Review of risk factor information suggests that tobacco use likely played some role in the development of lung and bronchus cancer among some individuals. Occupational exposures may have also been important in the development of lung and bronchus cancer among some individuals. However, because of the large number of individuals for whom occupation was unknown, it is difficult to fully assess the extent to which this factor influenced overall cancer patterns in CT 5022. The geographic distribution of residence at diagnosis for those who had an unknown occupation did not appear unusual. Analysis of the geographic distribution of place of residence for individuals diagnosed with lung and bronchus cancer in CT 5022 did not reveal any atypical spatial patterns. No apparent concentrations of individuals diagnosed with lung and bronchus cancer were observed in the vicinity of the former Suburban Auto site.

IX. RECOMMENDATIONS

The MDPH recommends no further investigation of lung and bronchus cancer incidence in CT 5022 at this time, but will continue to monitor the incidence of lung and bronchus cancer in the town of Rockland through city/town cancer incidence reports published by the MCR.

The only way to know if your home has a radon problem is to do a radon test. The MDPH recommends that Massachusetts residents test their houses for radon. For further questions about radon, you may contact MDPH's Radiation Control Program toll free at (800) 723-6695 for advice on home testing.

For more information about quitting smoking, contact the Massachusetts Tobacco Control Program at 1-800-Try-To-Stop or 1-800-879-8678.

X. PUBLIC HEALTH ACTION PLAN

The purpose of the Public Health Action Plan is to ensure that this health consultation not only identifies potential public health hazards, but also provides a plan of action designed to mitigate and prevent adverse health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of MDPH to follow up on this plan to ensure that it is implemented. The public health action to be implemented by MDPH is as follows: Cancer incidence in the town of Rockland will continue to be monitored by the Community Assessment Program of the MDPH, BEH through city/town reports published by the MCR.

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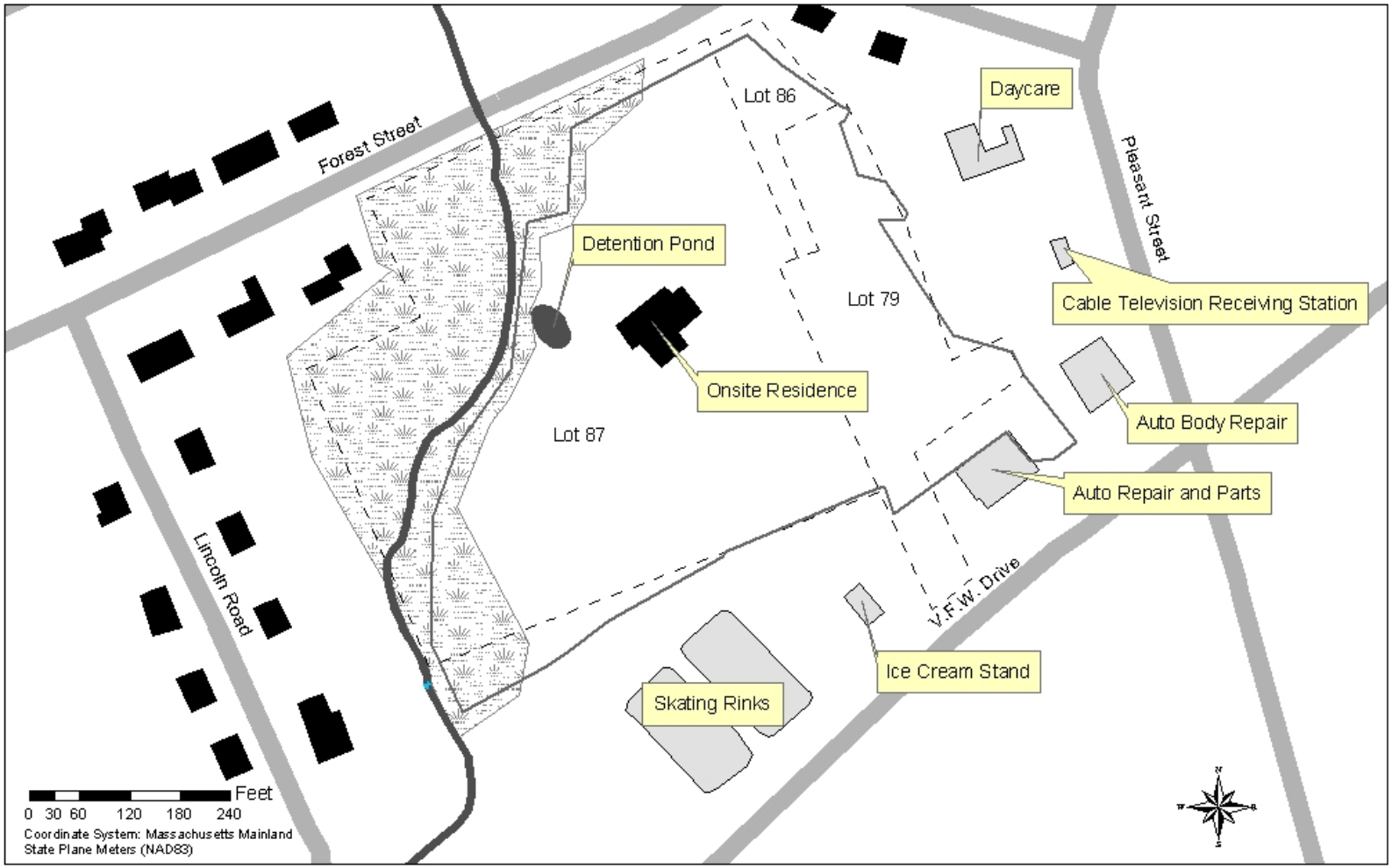
FIGURES

Figure 1
Former Suburban Auto Site
Rockland, Massachusetts



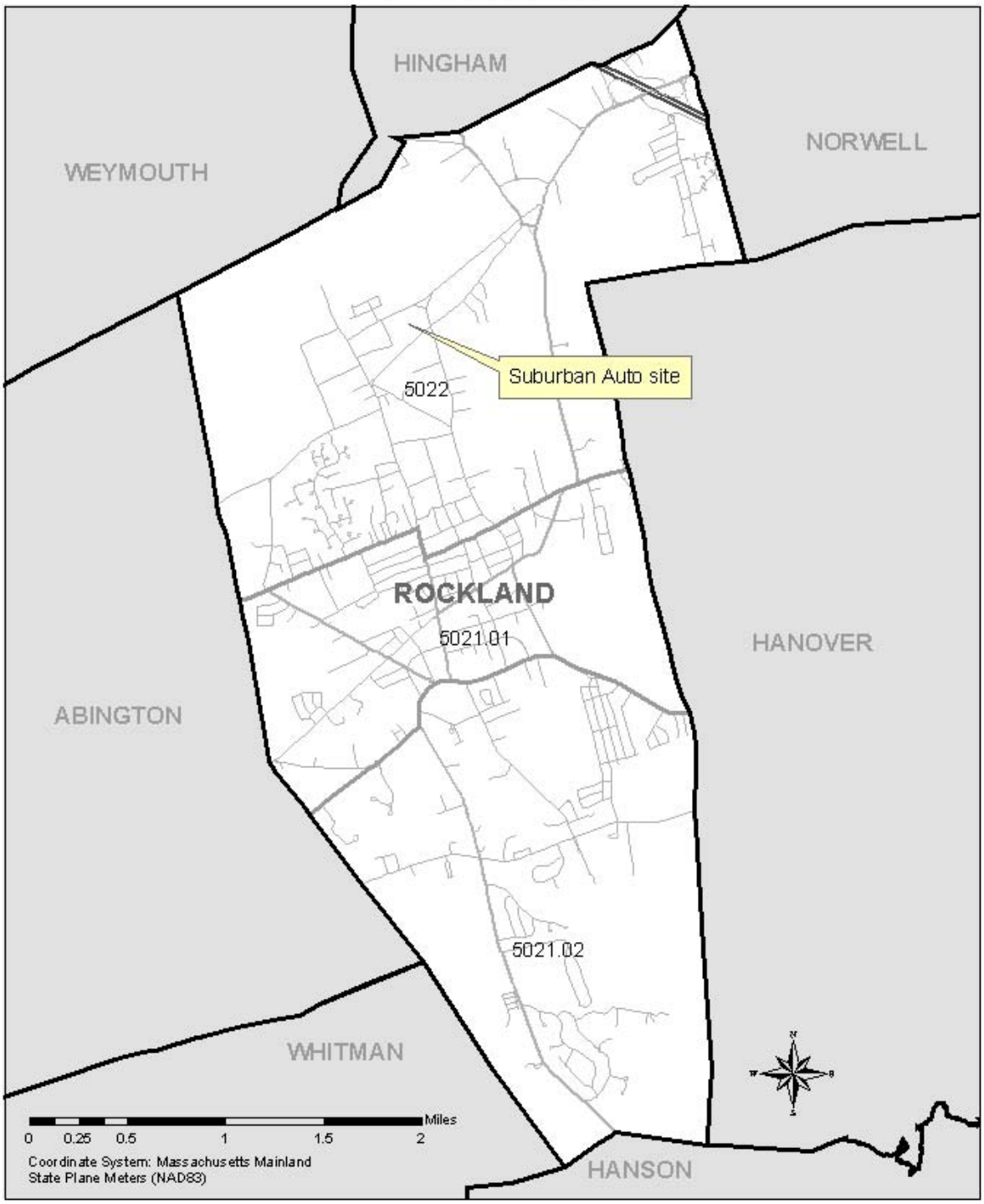
Note: Map based on original site sampling maps by Coler & Colantonio, Inc. 1999, 2002.

Map Created by:
Community Assessment Program
MDPH Bureau of Environmental Health
BB 10/30/2008

Geographic data supplied by:
Massachusetts Executive Office of Environmental Affairs, MassGIS.

Roads
Stream
Fence Perimeter
Commercial Buildings
Residences
Wooded Wetlands
Property Line

Figure 2
Census Tracts
Rockland, Massachusetts

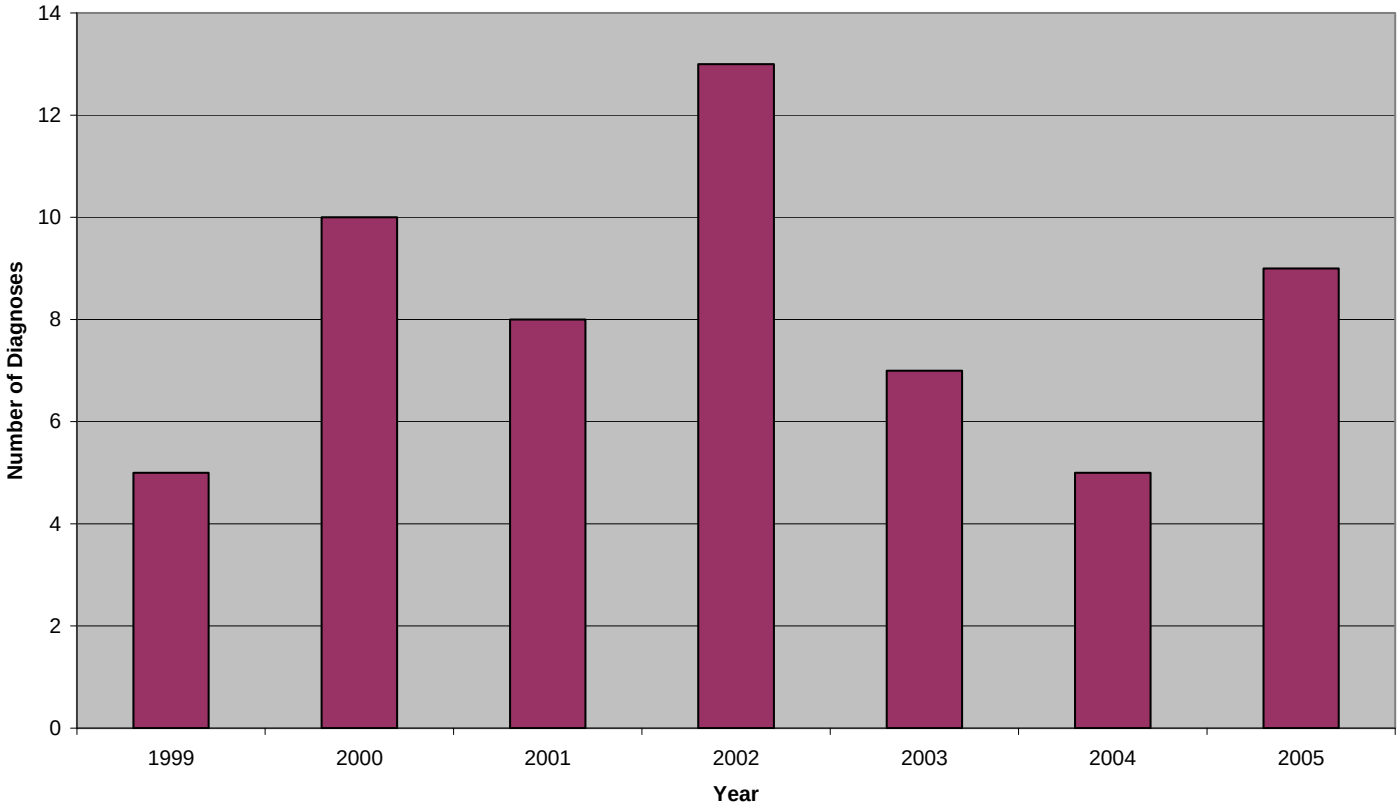


Map Created by:
Community Assessment Program
MDPH Bureau of Environmental Health
BB 10/30/2008

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

Roads
Census Tracts
Towns

Figure 3
Temporal Distribution of Lung and Bronchus Cancer, 1999-2005
CT 5022, Rockland, Massachusetts



TABLES

Table 1
Maximum concentrations of non-volatile contaminants in surface soil and volatile contaminants in subsurface soil that exceed USEPA SSLs for inhalation
Former Suburban Auto Site, Rockland, Massachusetts

CHEMICAL	CHEMICAL FAMILY	FREQUENCY OF DETECTION*	SAMPLE DEPTH	MAXIMUM DETECTED CONCENTRATION (ppm)	BACKGROUND SOIL LEVEL (ppm)	USEPA SOIL SCREENING LEVEL (SSL) FOR INHALATION (mg/kg)
Acenaphthylene	PAH	2 / 9	Surface	2.4	0.005 (agricultural soil)† 0.5 (natural soil)††	Not Available
Benzo(g,h,i)perylene	PAH	8 / 9	Surface	5.6	0.01 - 0.07 (rural soil)† 0.09 - 47 (urban soil)† 1 (natural soil)††	Not Available
Lead	Metal	14 / 14	Surface	2,900	<10-300** 100 (natural soil)††	400 (Noncancer)‡‡
n-Butylbenzene	VOC	1 / 2	Subsurface	0.03	Not applicable	Not available
Phenanthrene	PAH	9 / 9	Surface	31	0.03 (rural soil)† 0.048 - 0.14 (agricultural soil)† 3 (natural soil)††	Not Available

Data Sources:

Coler & Colantonio. 1999. Phase I Initial Site Investigation and Tier Classification Opinion, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN: 4-6043. Norwell, Massachusetts. September.

Coler & Colantonio. 2002. Phase II Comprehensive Site Assessment, Suburban Auto of Rockland Site, 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Norwell, Massachusetts. March.

Coneco Engineers & Scientists. 2005. Release Abatement Measure Status Report, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Bridgewater, Massachusetts. January.

Rizzo Associates. 2005. Supplemental excavation completion report and restoration completion report, 200 VFW Drive (Lot 79), Rockland, Massachusetts. Framingham, Massachusetts. September 8.

Notes:

* "Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.

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

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

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

‡‡ Inhalation value not available. Value provided is Total SSL that incorporates ingestion, dermal and inhalation pathways (USEPA 2010b)

PAH = Polycyclic aromatic hydrocarbon

VOC = Volatile organic compound

Table 2
Maximum concentration of contaminants detected in 1999 and 2001 surface soil samples (0-6 inches) that exceed comparison values
Former Suburban Auto Site
Rockland, Massachusetts

Contaminant	Frequency of Detection	Maximum Detected Concentration (ppm)	Sample Name	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Acenaphthylene	2 / 9	2.4	B-5 Surface	MDEP S-1 = 1	0.005 (agricultural soil)[†] 0.5 (natural soil)[††]
Arsenic	6 / 6	19	B-5 Surface	CREG = 0.5 Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200	<0.1 - 73* 20 (natural soil)[††]
Barium	6 / 6	1,000	B-5 Surface	Int. EMEG (pica child) = 400 Chronic EMEG (child) = 10,000 Chronic EMEG (adult) = 100,000	10 - 1,500* 50 (natural soil)[††]
Benzo(a)anthracene	9 / 9	12	B-5 Surface	EPA RSL (residential) = 0.15 CREG** = 1	0.005 - 0.02 (rural soil)[†] 0.169 - 59 (urban soil)[†] 2 (natural soil)[††]
Benzo(a)pyrene	9 / 9	11	B-5 Surface	CREG = 0.1	0.002 - 1.3 (rural soil)[†] 0.165 - 0.22 (urban soil)[†] 2 (natural soil)[††]
Benzo(b)fluoranthene	9 / 9	10	B-5 Surface	EPA RSL (residential) = 0.15 CREG** = 1	0.02 - 0.03 (rural soil)[†] 15 - 62 (urban soil)[†] 2 (natural soil)[††]
Benzo(k)fluoranthene	9 / 9	8.9	B-5 Surface	EPA RSL (residential) = 1.5 CREG** = 1	0.01 - 0.11 (rural soil)[†] 0.3 - 26 (urban soil)[†] 1 (natural soil)[††]
Cadmium	9 / 10	40	S-3 Composite	Chronic EMEG (child) = 5 Chronic EMEG (adult) = 70	0.01 - 2.7[‡] 2 (natural soil)[††]
Dibenzo(a,h)anthracene	6 / 9	9.3	B-5 Surface	EPA RSL (residential) = 0.015 CREG** = 0.02	0.5 (natural soil)[††]
Indeno(1,2,3-cd)pyrene	8 / 9	9.3	B-5 Surface	EPA RSL (residential) = 0.15 CREG** = 1	0.01 - 0.015 (rural soil)[†] 8.0 - 61 (urban soil)[†] 1 (natural soil)[††]
Lead	14 / 14	2,900	B-5 Surface	EPA RSL (residential) = 400	<10 - 300* 100 (natural soil)[††]
Mercury	6 / 6	5.9	S-3 Composite	EPA RSL (residential) = 5.6	0.01 - 3.4* 0.3 (natural soil)[††]
Phenanthrene	9 / 9	31	B-5 Surface	MDEP S-1 = 10	0.03 (rural soil)[†] 0.048 - 0.14 (agricultural soil)[†] 3 (natural soil)[††]
Polychlorinated biphenyls (PCBs)	8 / 8	8.8	S-3 Composite	CREG = 0.4 Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable

"Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.

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

[†]Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polyaromatic hydrocarbons. Atlanta: U.S. Department of Health and

[‡]ATSDR. 2008. Toxicological profile for cadmium. Draft for public comment. Atlanta: U.S. Department of Health and Human Services.

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

ppm = parts per million

< = less than

Comparison Values:

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

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

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

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

Intermediate EMEG (adult/child) = Environmental Media Evaluation Guide for adults (i.e., for adult or childhood exposures between 14 days and 1 year) (ATSDR 2005, 20010c)

MDEP S-1 = Massachusetts Contingency Plan Method 1 soil category S-1 standards [310 CMR 40.0975(6)(a)] (MDEP 2007)

Data Sources:

Coler & Colantonio. 1999. Phase I Initial Site Investigation & Tier Classification Opinion, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN: 4-6043. Norwell, Massachusetts. September.

Coler & Colantonio. 2002. Phase II Comprehensive Site Assessment, Suburban Auto of Rockland Site, 163 Forest Street, Rockland, Massachusetts, RTN 4-6043.

Table 3
Maximum concentration of contaminants detected in subsurface soil samples (1999-2005) that exceed comparison values
Former Suburban Auto Site
Rockland, Massachusetts

Contaminant	Frequency of Detection	Maximum Detected Concentration (ppm)	Sample Name	Sample Depth (feet)	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Antimony	2 / 9	50.7	TP-17	1 - 3	RMEG (child) = 20 RMEG (adult) = 300	<1 - 8.8* 1 (natural soil)††
Aroclor - 1016 / 1242	3 / 21	0.47	E-2 Bottom	Not specified	CREG = 0.4 (for PCBs) Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable
Arcolor - 1248	2 / 24	3.04	CS-120	1	CREG = 0.4 (for PCBs) Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable
Aroclor - 1254	11 / 38	0.62	A-1 Side	Not specified	CREG = 0.4 (for PCBs) Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable
Aroclor - 1260	15 / 38	0.77	E-2 Bottom	Not specified	CREG = 0.4 (for PCBs) Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable
Barium	14 / 14	428	E-2 Bottom	Not specified	Chronic EMEG (child) = 10,000 Chronic EMEG (adult) = 100,000 Int. EMEG (pica child) = 400	10 - 1,500* 50 (natural soil)††
Benzo(a)anthracene	21 / 45	7.9	CS-126	1	EPA RSL (residential) = 0.15 CREG** = 1	0.005 - 0.02 (rural soil)† 0.169 - 59 (urban soil)† 2 (natural soil)††
Benzo(a)pyrene	22 / 59	9.5	CS-126	1	CREG = 0.1	0.002 - 1.3 (rural soil)† 0.165 - 0.22 (urban soil)† 2 (natural soil)††
Benzo(b)fluoranthene	22 / 59	2.8	CS-131	1	EPA RSL (residential) = 0.15 CREG** = 1	0.02 - 0.03 (rural soil)† 15 - 62 (urban soil)† 2 (natural soil)††
Benzo(k)fluoranthene	18 / 45	4.4	CS-126	1	EPA RSL (residential) = 1.5 CREG** = 1	0.01 - 0.11 (rural soil)† 0.3 - 26 (urban soil)† 1 (natural soil)††
Cadmium	6 / 23	19.6	E-2 Bottom	Not specified	Chronic EMEG (child) = 5 Chronic EMEG (adult) = 70	0.01 - 2.7‡ 2 (natural soil)††
Chromium (total)	19 / 23	1,600	TP-17	1 - 3	*Hexavalent* RMEG (child) = 200 RMEG (adult) = 2,000 *Trivalent* RMEG (child) = 80,000 RMEG (adult) = 1,000,000	1 - 1,000* 30 (natural)††
Copper	9 / 9	552	SB-03	0 - 1	Int. EMEG (pica child) = 20 Int. EMEG (child) = 500 Int. EMEG (adult) = 7,000	<1 - 700* 40 (natural soil)††
Dibenzo(a,h)anthracene	2 / 45	0.54	CS-126	1	EPA RSL (residential) = 0.015 CREG** = 0.02	0.5 (natural soil)††
Indeno(1,2,3-cd)pyrene	12 / 45	4.9	CS-126	1	EPA RSL (residential) = 0.15 CREG** = 1	0.01 - 0.015 (rural soil)† 8.0 - 61 (urban soil)† 1 (natural soil)††
Lead	59 / 64	16,000	CS-126	1	EPA RSL (residential) = 400	<10 - 300* 100 (natural soil)††
n-Butylbenzene	1 / 2	0.03	TP-13	2 - 6	Not applicable***	Not applicable

"Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.
*Observed range for the Eastern United States (east of 96th meridian). USGS. 1984. Shacklette HT, Boerngen JG. Element Concentrations in Soils and Other Surficial Materials of the conterminous United States. U.S. Geological Survey Professional Paper 1270.
†Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polyaromatic hydrocarbons. Atlanta: U.S. Department of Health and Human Services.
‡ATSDR. 2008d. Toxicological profile for cadmium. Draft for public comment. Atlanta: U.S. Department of Health and Human Services.
††MDEP. 2002. Background levels of polycyclic aromatic hydrocarbons and metals in soil. Office of Research and Standards.
ppm = parts per million
< = less than

Comparison Values:
RMEG (adult/child) = Reference Dose Media Evaluation Guide (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure)
CREG = Cancer Risk Evaluation Guide for 1 x 10-6 excess cancer risk (ATSDR 2005, 2010c)
CREG** = Estimated CREG using toxicity equivalency factors relative to benzo(a)pyrene developed by USEPA.
Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2005, 2010c)
Intermediate EMEG (adult/child) = Environmental Media Evaluation Guide for adults (i.e., for adult or childhood exposures between 14 days and 1 year) (ATSDR 2005, 2010c)
EPA RSL = EPA Region 3 Regional Screening Level for soil (USEPA 2010b)
***No comparison values could be located for n-butylbenzene. As a result, no further analysis was conducted.

Data Source:
Coler & Colantonio. 1999. Phase I Initial Site Investigation and Tier Classification Opinion, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN: 4-6043. Norwell, Massachusetts. September.

Coler & Colantonio. 2002. Phase II Comprehensive Site Assessment, Suburban Auto of Rockland Site, 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Norwell, Massachusetts. March.
Coneco Engineers & Scientists. 2005. Release Abatement Measure Status Report, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Bridgewater, Massachusetts. January.
Rizzo Associates. 2005. Supplemental excavation completion report and restoration completion report, 200 VFW Drive (Lot 79), Rockland, Massachusetts. Framingham, Massachusetts. September 8.

Table 4
Concentration of contaminants detected in automotive shredder residue samples (1999-2001) that exceed comparison values
Former Suburban Auto Site
Rockland, Massachusetts

Contaminant	Frequency of Detection	Maximum Detected Concentration (ppm)	Sample Name	Sample Depth (feet)	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Arsenic	3/3	25	TP-6	5	CREG = 0.5 Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200	<0.1 - 73* 20 (natural soil)‡‡
Benzo(a)anthracene	1/1	1.4	TP-6	5	EPA RSL (residential) = 0.15 CREG** = 1	0.005 - 0.02 (rural soil)† 0.169 - 59 (urban soil)† 2 (natural soil)‡‡
Benzo(a)pyrene	1/1	1.1	TP-6	5	CREG = 0.1	0.002 - 1.3 (rural soil)† 0.165 - 0.22 (urban soil)† 2 (natural soil)‡‡
Cadmium	3/3	48.3	KE1	6	Chronic EMEG (child) = 5 Chronic EMEG (adult) = 70	0.01 - 2.7‡ 2 (natural soil)‡‡
Di(2-ethylhexyl)phthalate	1/1	600	TP-6	5	CREG = 50 Chronic EMEG (child) = 3,000 Chronic EMEG (adult) = 40,000	Not available††
Lead	3/3	2,740	KE1	6	EPA RSL (residential) = 400	<10 - 300* 100 (natural soil)‡‡
Polychlorinated biphenyls (PCBs)	3/3	20	KE1	6	CREG = 0.4 Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable

"Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.

*Observed range for the Eastern United States (east of 96th meridian). USGS. 1984. Shacklette HT, Boerngen JG. Element concentrations in soils and other surficial materials of the conterminous United States. U.S. Geological Survey Professional Paper 1270. Washington: United States Government Printing Office.

†Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polyaromatic hydrocarbons. Atlanta: U.S. Department of Health and Human Services.

††ATSDR. 2002. Toxicological profile for di(2-ethylhexyl)phthalate (DEHP). Atlanta: U.S. Department of Health and Human Services.

‡ATSDR. 2008. Toxicological profile for cadmium. Atlanta: U.S. Department of Health and Human Services.

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

ppm = parts per million

< = less than

Comparison Values:

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

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

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

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

Data Source:

Kaegael Environmental. 2001. Work authorization for LSP services, 200 VFW Drive, Rockland, Massachusetts. Fall River, Massachusetts. May.

Rackemann, Sawyer & Brewster. 1999. Letter to Jonathan E. Hobill, MDEP Regional Engineer, from Sanford M. Matathia concerning contamination at the Suburban Auto of Rockland, Inc. site. Boston, Massachusetts. July.

Table 5
Maximum concentration of contaminants detected in onsite groundwater samples (1999-2004) that exceed comparison values
Former Suburban Auto Site
Rockland, Massachusetts

Contaminant	Frequency of Detection	Maximum Detected Concentration (ppb)	Sample Name	Date of Sample	Drinking Water Comparison Value (ppb)
Tetrachloroethylene (PCE)	5 / 28	97	MW-8	6/16/1999	RMEG (child) = 100 RMEG (adult) = 400 LTHA = 10 MCL = 5 EPA RSL (tap water) = 0.11 MDEP MMCL = 5
Trichloroethylene (TCE)	4 / 28	7.8	MW-8	5/20/2004	EPA RSL (tap water) = 2 MCL = 5 MDEP MMCL = 5
Vinyl Chloride	1 / 27	5.8	MW-8	5/20/2004	CREG = 0.02 Chronic EMEG (child) = 30 Chronic EMEG (adult) = 100 MCL = 2 MDEP MMCL = 2 EPA RSL (tap water) = 0.016

"Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.

ppb = parts per billion
MW = monitoring well

Comparison Values:

CREG = Cancer Risk Evaluation Guide for 1 x 10^{-6} excess cancer risk (ATSDR 2010a)
Chronic EMEG (adult/child) = Environmental Media Evaluation Guide (i.e., for adult or childhood exposures greater than 1 year) (ATSDR 2010a)
EPA RSL = EPA Region 3 Regional Screening Level for tap water (USEPA 2010b)
LTHA = EPA Lifetime health advisory for drinking water (ATSDR 2010a)
MCL = EPA Maximum Contaminant Level for drinking water (ATSDR 2010a)
MDEP MMCL = Massachusetts Department of Environmental Protection Massachusetts Maxiumum Contaminant Level for drinking water (MDEP 2009b)
RMEG (adult/child) = Reference Dose Media Evaluation Guides (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure) (ATSDR 2010a)

Data Sources:

Coler & Colantonio. 1999. Phase I initial site investigation and tier classification opinion, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN: 4-6043. Norwell, Massachusetts. September.

Coler & Colantonio. 2002. Phase II comprehensive site assessment, Suburban Auto of Rockland Site, 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Norwell, Massachusetts. March.

Coneco Engineers & Scientists. 2005. Release abatement measure status report, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Bridgewater, Massachusetts. January.

Table 6
Maximum concentration of contaminants detected in sediment samples (1999-2004) that exceed comparison values
Former Suburban Auto Site
Rockland, Massachusetts

Contaminant	Frequency of Detection	Maximum Detected Concentration (ppm)	Sample Name	Soil Comparison Value (ppm)	Background Soil Level (ppm)
Arsenic	1 / 6	<20*	Detention pond sediment	CREG = 0.5 Chronic EMEG (child) = 20 Chronic EMEG (adult) = 200	<0.1 - 73† 20 (natural soil)††
Benzo(a)anthracene	7 / 19	1.3	Detention pond sediment	EPA RSL (residential) = 0.15 CREG** = 1	0.005 - 0.02 (rural soil)‡ 0.169 - 59 (urban soil)‡ 2 (natural soil)††
Benzo(a)pyrene	8 / 19	1.4	Detention pond sediment	CREG = 0.1	0.002 - 1.3 (rural soil)‡ 0.165 - 0.22 (urban soil)‡ 2 (natural soil)††
Benzo(b)fluoranthene	8 / 19	3.7	Detention pond sediment	EPA RSL (residential) = 0.15 CREG** = 1	0.02 - 0.03 (rural soil)‡ 15 - 62 (urban soil)‡ 2 (natural soil)††
Benzo(k)fluoranthene	6 / 19	1.2	Detention pond sediment	EPA RSL (residential) = 1.5 CREG** = 1	0.01 - 0.11 (rural soil)‡ 0.3 - 26 (urban soil)‡ 1 (natural soil)††
Cadmium	6 / 6	13	Detention pond sediment	Chronic EMEG (child) = 5 Chronic EMEG (adult) = 70	0.01 - 2.7‡‡ 2 (natural soil)††
Indeno(1,2,3-cd)pyrene	5 / 19	1.1	Sed Basin-1	EPA RSL (residential) = 0.15 CREG** = 1	0.01 - 0.015 (rural soil)‡ 8.0 - 61 (urban soil)‡ 1 (natural soil)††
Lead	17 / 19	1,330	Sed Basin-1	EPA RSL (residential) = 400	<10 - 300† 100 (natural soil)††
Polychlorinated Biphenyls (PCBs)	6 / 9	1.9	Sed Basin-1	CREG = 0.4 Chronic EMEG (child) = 1 (for Aroclor 1254) Chronic EMEG (adult) = 10 (for Aroclor 1254)	Not applicable

"Frequency of Detection" refers to the ratio of the number of samples in which a particular chemical was detected versus the number of samples taken and analyzed for this chemical.

*Exact value not quantified in laboratory analytical results

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

‡Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polyaromatic hydrocarbons. Atlanta: U.S. Department of Health and Human Services.

‡‡ATSDR. 2008. Toxicological profile for cadmium. Draft for public comment. Atlanta: U.S. Department of Health and Human Services.

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

ppm = parts per million

< = less than

Comparison Values:

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

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

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

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

Data Sources:

Coler & Colantonio. 1999. Phase I Initial Site Investigation & Tier Classification Opinion, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Norwell, Massachusetts. September.

Coler & Colantonio. 2002. Phase II Comprehensive Site Assessment, Suburban Auto of Rockland Site, 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Norwell, Massachusetts. March.

Coneco Engineers & Scientists. 2005. Release Abatement Measure Status Report, Suburban Auto of Rockland, Inc., 163 Forest Street, Rockland, Massachusetts, RTN 4-6043. Bridgewater, Massachusetts. January.

TABLE 7
Lung & Bronchus Cancer Incidence
Rockland, Massachusetts
1999-2005

Census Tract	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
5022	57	42.7	133 *	101 -- 173	25	20.7	121	78 -- 178	32	22.0	146	100 -- 206

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

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

Exposure Dose and Cancer Risk Calculation Formulas:

Noncancer Health Effects Exposure Factor:

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

Noncancer Health Effects Exposure Dose (Ingestion):

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

Cancer Effects Exposure Factor:

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

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)
$[C]_{soil}$	= Maximum Analyte Concentration in Soil (mg/kg)
IR	= Soil Ingestion Rate (mg/day)
CF	= Conversion Factor (kg/mg)
BW	= Body Weight (kg)
C_EF	= Cancer Exposure Factor (unitless)
C_D	= Cancer Exposure Dose (mg/kg/day)
CR	= Cancer Risk (unitless)
CSF	= Cancer Slope Factor ($mg/kg/day^{-1}$)

Assumptions:

1) The receptors evaluated were an adult caretaker who lives on-site and an older child who trespasses.
2) The maximum concentration of cadmium, lead, mercury, PCBs and PAHs detected in on-site soil was assumed as the soil concentration.
3) The amount of soil ingested was assumed to be 100 milligrams per day for the adult receptor and 200 milligrams per day for the older child.
4) The exposure factor was determined assuming the adult receptor was exposed to site soil 7 days per week, for 50 weeks per year over a 30 year time period and the older child receptor was exposed to site soil 2 days per week, for 26 weeks per year over a 10 year period.
5) The average body weight of both receptors was assumed to be 70 kilograms.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

1. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing Cadmium:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{40\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000055\ \text{mg/kg/day}$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{40\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000016\ \text{mg/kg/day}$$

NOTES:

1. The ATSDR Chronic MRL for cadmium is 0.0001 mg/kg/day.
2. The EPA has not classified cadmium with respect to its cancer causing potential and has not developed an EPA Oral Cancer Slope Factor for cadmium. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for cadmium.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

2. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing Lead:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{2900\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96\ \text{x}\ 10^{-6}}{70\ \text{kg}} = 0.004\ \text{mg/kg/day}$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{2900\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14\ \text{x}\ 10^{-6}}{70\ \text{kg}} = 0.0012\ \text{mg/kg/day}$$

NOTES:

1. There is no ATSDR MRL or EPA RfD available for lead. The calculated exposure dose for lead was input into the US Environmental Protection Agency's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows® (IEUBKwin32, Lead Model Version 1.0, Build 264) to estimate blood lead (PbB) levels in children exposed to lead-contaminated media. The IEUBK model results indicated that exposure to the average concentration of lead in on-site surface soil (836 mg/kg) would not result in a predicted mean blood lead concentration above 10 µg/dL, which the CDC defines as a level of concern.
2. The USEPA has categorized lead as a probable human carcinogen; however, they have concluded that existing scientific information cannot determine whether or not exposure to lead can cause cancer in humans; thus, no USEPA Oral Cancer Slope Factor has been developed for lead. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for lead.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

3. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing Mercury:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\,\text{days/year} \times 30\,\text{years}}{30\,\text{years} \times 365\,\text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{5.9\,\text{mg/kg} \times 100\,\text{mg/day} \times 0.96 \times 10^{-6}\,\text{kg/mg}}{70\,\text{kg}} = 0.0000081\,\text{mg/kg/day}$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\,\text{days/year} \times 10\,\text{years}}{10\,\text{years} \times 365\,\text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{5.9\,\text{mg/kg} \times 200\,\text{mg/day} \times 0.14 \times 10^{-6}\,\text{kg/mg}}{70\,\text{kg}} = 0.0000024\,\text{mg/kg/day}$$

NOTES:

1. The ATSDR Intermediate MRL for inorganic mercury is 0.002 mg/kg/day.
2. The EPA has not classified mercury with respect to its cancer causing potential and has not developed an EPA Oral Cancer Slope Factor for mercury. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for mercury.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

4. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing PAHs:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose for Acenapthylene} = \frac{2.4\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000033\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Benzo(a)pyrene} = \frac{11.0\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000015\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Dibenzo(a, h)anthracene} = \frac{9.3\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000013\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Phenanthrene} = \frac{31.0\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000043\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Cancer Effects Exposure Dose} = \frac{57.5\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.41 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000034\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000034 \times 7.3 = 0.00025$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose for Acenapthylene} = \frac{2.4\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00000098\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Benzo(a)pyrene} = \frac{11.0\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000045\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Dibenzo(a, h)anthracene} = \frac{9.3\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000038\ \text{mg/kg/day}$$

$$\text{Noncancer Health Effects Exposure Dose for Phenanthrene} = \frac{31.0\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000013\ \text{mg/kg/day}$$

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

$$\text{Cancer Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.02$$

$$\text{Cancer Effects Exposure Dose} = \frac{57.5\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.02 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000033\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000033 \times 7.3 = 0.000024$$

NOTES:

1. The EPA RfD for pyrene (0.03 mg/kg/day) was used to evaluate noncancer health effects from PAHs detected on-site.
2. The sum of the toxicity equivalence (TEQ) in terms of benzo(a)pyrene was used to evaluate cancer risk from carcinogenic PAHs detected on-site.
3. The EPA Oral Cancer Slope Factor for benzo(a)pyrene is 7.3 $mg/kg/day^{-1}$.

APPENDIX A

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site Surface Soil Suburban Auto, Rockland, Massachusetts

5. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site Surface Soil Containing PCBs:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{8.8\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000012\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Cancer Effects Exposure Dose} = \frac{8.8\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.41 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000052\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.0000052 \times 2 = 0.00001$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{8.8\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000036\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.02$$

$$\text{Cancer Effects Exposure Dose} = \frac{8.8\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.02 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00000051\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000051 \times 2 = 0.000001$$

NOTES:

1. The ATSDR 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 INGESTION OF ON-SITE AUTOMOTIVE SHREDDER RESIDUE

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site ASR Suburban Auto, Rockland, Massachusetts

1. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site ASR Containing Cadmium:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{48.3\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000066\ \text{mg/kg/day}$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{48.3\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00002\ \text{mg/kg/day}$$

NOTES:

1. The ATSDR Chronic MRL for cadmium is 0.0001 mg/kg/day.
2. The EPA has not classified cadmium with respect to its cancer causing potential and has not developed an EPA Oral Cancer Slope Factor for cadmium. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for cadmium.

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site ASR Suburban Auto, Rockland, Massachusetts

2. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site ASR Containing Di(2-ethylhexyl)phthalate:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{600\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00082\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Cancer Effects Exposure Dose} = \frac{600\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.41 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00035\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00035 \times 0.014 = 0.0000049$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{600\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.00024\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.02$$

$$\text{Cancer Effects Exposure Dose} = \frac{600\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.02 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000035\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000035 \times 0.014 = 0.00000049$$

NOTES:

1. The ATSDR MRL for di(2-ethylhexyl)phthalate is 0.06 mg/kg/day.
2. The EPA Oral Cancer Slope Factor for di(2-ethylhexyl)phthalate is 0.014 mg/kg/day^{-1}.

APPENDIX B

Exposure Dose and Cancer Risk Calculations for Exposure via Ingestion of On-Site ASR Suburban Auto, Rockland, Massachusetts

3. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site ASR Containing PCBs:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{20\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000027\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.41$$

$$\text{Cancer Effects Exposure Dose} = \frac{20\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.41 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.000012\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.000012 \times 2 = 0.000023$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{20\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000081\ \text{mg/kg/day}$$

$$\text{Cancer Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{70\ \text{years} \times 365\ \text{days}} = 0.02$$

$$\text{Cancer Effects Exposure Dose} = \frac{20\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.02 \times 10^{-6}\ \text{kg/mg}}{70\ \text{kg}} = 0.0000012\ \text{mg/kg/day}$$

$$\text{Cancer Risk} = 0.00000012 \times 2 = 0.0000023$$

NOTES:

1. The ATSDR 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 Ingestion of On-Site ASR
Suburban Auto, Rockland, Massachusetts

4. Exposure Dose and Cancer Risk Calculations for Ingestion of On-Site ASR Containing Lead:

a. Adult

$$\text{Noncancer Health Effects Exposure Factor} = \frac{350\ \text{days/year} \times 30\ \text{years}}{30\ \text{years} \times 365\ \text{days}} = 0.96$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{2740\ \text{mg/kg} \times 100\ \text{mg/day} \times 0.96\ \text{x}\ 10^{-6}}{70\ \text{kg}} = 0.0038\ \text{mg/kg/day}$$

b. Older Child

$$\text{Noncancer Health Effects Exposure Factor} = \frac{52\ \text{days/year} \times 10\ \text{years}}{10\ \text{years} \times 365\ \text{days}} = 0.14$$

$$\text{Noncancer Health Effects Exposure Dose} = \frac{2740\ \text{mg/kg} \times 200\ \text{mg/day} \times 0.14\ \text{x}\ 10^{-6}}{70\ \text{kg}} = 0.0011\ \text{mg/kg/day}$$

NOTES:

1. There is no ATSDR MRL or EPA RfD available for lead. The calculated exposure dose for lead was less than that which was calculated for exposure via ingestion of on-site surface soil. As indicated in Appendix A, the exposure dose from surface soil was input into the U.S. Environmental Protection Agency's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows® (IEUBKwin32, Lead Model Version 1.0, Build 264) to estimate blood lead (PbB) levels in children exposed to lead-contaminated media. The IEUBK model results indicated that exposure to the average concentration of lead in on-site surface soil (836 mg/kg) would not result in a predicted mean blood lead concentration above 10 µg/dL, which the CDC defines as a level of concern. Since the exposure dose from ingestion of on-site ASR is less than that from ingestion of on-site surface soil, exposure to the maximum concentration of lead from on-site ASR would also not result in a predicted mean blood lead concentration above the CDC level of concern.
2. The EPA has categorized lead as a probable human carcinogen; however, they have concluded that existing scientific information cannot determine whether or not exposure to lead can cause cancer in humans; thus, no EPA Oral Cancer Slope Factor has been developed for lead. Due to the lack of evidence for cancer health effects in humans, cancer risk was not calculated for lead.

APPENDIX C: RISK FACTOR INFORMATION FOR LUNG AND BRONCHUS CANCERS

How to Use this Factsheet

This risk factor summary was developed to serve as a general fact sheet. It is an overview and should not be considered exhaustive. For more information on other possible risk factors and health effects being researched, please see the References section.

A risk factor is anything that increases a person's chance of developing cancer. Some risk factors can be controlled while others cannot. Risk factors can include *hereditary conditions*, *medical conditions or treatments*, *infections*, *lifestyle factors*, or *environmental factors*. Although risk factors can influence the development of cancer, most do not directly cause cancer. An individual's risk for developing cancer may change over time due to many factors and it is likely that multiple risk factors influence the development of most cancers. Knowing the risk factors that apply to specific concerns and discussing them with your health care provider can help to make more informed lifestyle and health-care decisions.

For cancer types with environmentally-related risk factors, an important factor in evaluating cancer risk is the route of exposure. This is particularly relevant when considering exposures to chemicals in the environment. For example, a particular chemical may have the potential to cause cancer if an individual breathes the chemical in. That same chemical may not increase the risk of cancer similarly if an individual comes into contact with the chemical by touching it. In addition, an individual must generally be exposed to a chemical at a sufficient dose and for a sufficient duration of time for an adverse health effect to occur.

Gene-environment interactions are another important area of cancer research. An individual's risk of developing cancer may depend on a complex interaction between their genetic make-up and exposure to an environmental agent (for example, a virus or a chemical contaminant). This may explain why some individuals have a fairly low risk of developing cancer as a result of an environmental factor or exposure, while others may be more vulnerable.

Key Statistics

Lung and bronchus cancer is the second most common cancer in both men (after prostate cancer) and women (after breast cancer). The American Cancer Society estimates 222,520 individuals will be diagnosed with lung and bronchus cancer in the U.S. in 2010: 116,750 men and 105,770 women. In Massachusetts, lung and bronchus cancer is expected to account for about 14% of all cancers diagnosed within the state in 2010. These cancers mainly occur in older individuals, with roughly two-thirds of those diagnosed older than 65 years of age. Fewer than 3% of diagnoses occur in individuals under the age of 45. The incidence of lung and bronchus cancer is greater among men than women. African American men are more likely to develop lung and bronchus cancer than white men, though the incidence rate is about the same in African American and white women. For several years, the incidence rate dropped among men but

remained fairly stable among women.

Types of Lung and Bronchus Cancer

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. 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).

Lung and bronchus tumors can be either malignant (cancerous) or benign (non-cancerous). The lung and bronchus are sites where both primary and secondary tumors can arise; secondary lung and bronchus tumors generally originate elsewhere in the body and then metastasize, or spread, to the lung or bronchus. There are two main types of primary lung and bronchus cancers: small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC). About 85% to 90% of lung and bronchus cancers are NSCLC, of which there are three subtypes: adenocarcinoma, squamous cell carcinoma, and large-cell (undifferentiated) carcinoma. Adenocarcinoma is usually found in the outer region of the lung and is the most common subtype in the U.S., accounting for about 40% of lung and bronchus cancer diagnoses. Squamous cell carcinoma accounts for about 25% to 30% of lung and bronchus cancers and tends to be found in the middle of the lungs, near a bronchus. Large-cell carcinoma accounts for about 10% to 15% of lung and bronchus cancers and may appear in any part of the lung. SCLC also accounts for about 10% to 15% of all lung and bronchus cancers but often starts in the bronchi. Additional rare types of lung and bronchus cancers include carcinoid tumors, adenoid cystic carcinomas, and hamartomas, among others.

Established Risk Factors

Hereditary Conditions

Siblings and children of those who have had lung or bronchus cancer may have a slightly higher risk themselves; however, it is not clear whether this risk may be attributed to hereditary conditions or to shared exposures (such as tobacco smoke or radon). Genetics do seem to play a role in some families with a strong history of lung and bronchus cancer. Individuals who inherit certain DNA changes are more likely to develop lung and bronchus cancer. Although these changes cannot be routinely tested for at this time, research is ongoing.

Medical Conditions

Individuals who have had lung or bronchus cancer have a higher risk of developing a second lung or bronchus tumor. In addition, individuals who have had radiation therapy to the chest for cancer are at higher risk for lung and bronchus cancer, particularly if they smoke. Typical patients are those treated for Hodgkin disease or women who get radiation after a mastectomy for breast cancer. Women who receive radiation therapy to the breast after a lumpectomy do not appear to have an elevated risk of lung and bronchus cancer.

Lifestyle Factors

Smoking is by far the most important risk factor for lung and bronchus cancer. For example, SCLC is almost always caused by smoking and rarely develops in an individual who has never smoked. The risk of lung and bronchus cancer increases with the quantity and duration of cigarette consumption. Smoking of cigars and pipes is almost as likely to cause lung and bronchus cancer as cigarette smoking. Furthermore, smoking low tar or "light" cigarettes increases the risk of lung and bronchus cancer just as much as regular cigarettes. Mentholated cigarettes are thought to increase the risk of lung and bronchus cancer even more since the menthol allows smokers to inhale more deeply.

Approximately 85% to 90% of deaths from lung and bronchus cancer are thought to result from smoking. If an individual stops smoking before a cancer develops, the damaged lung tissue gradually repairs itself. No matter the age of an individual or how long someone has smoked, quitting may help an individual to live longer. Information about quitting smoking and related services are available from the Massachusetts DPH Tobacco Control Program at 1-800-Try-To-Stop or 1-800-879-8678.

Breathing in the smoke of others (called secondhand smoke) at home and in the workplace also increases an individual's risk of developing lung and bronchus cancer. A nonsmoker who lives with a smoker has about a 20% to 30% greater risk of developing lung cancer. Some evidence suggests that some people may be more susceptible to the cancer-causing effect of tobacco smoke than others.

Environmental Exposures

Exposure to radon (a naturally occurring radioactive gas produced by the breakdown of uranium in soil and rocks) has been identified as the second leading cause of lung and bronchus cancer, and the leading cause among nonsmokers. The level of radon that occurs outdoors is not dangerous. However, indoor levels of radon can be more concentrated and may increase the risk of developing lung and bronchus cancer. According to the World Health Organization, radon may account for up to 15% of lung cancers worldwide. Houses that are built on soil with natural uranium deposits can create high levels of indoor radon, particularly in basements. If you are concerned about radon exposure, contact the Massachusetts Radiation Control Program, Radon Unit at 1-800-723-6695.

Occupational exposure to asbestos is an important risk factor for lung and bronchus cancer. Asbestos may occur in mines, mills, textile plants, shipyards, and where insulation is used. In recent years, government regulations have reduced the use of asbestos in commercial and industrial products. It is still present in many homes and commercial buildings but is not usually considered harmful as long as it is not released into the air by deterioration, demolition, or renovation.

In addition to asbestos and radon, chemical compounds that are also occupational risk

factors for lung and bronchus cancer include arsenic, beryllium, cadmium, silica, vinyl chloride, nickel compounds, chromium compounds, coal products, mustard gas, chloromethyl ethers, diesel exhaust and radioactive ores such as uranium. The risk of lung and bronchus cancer from each of the above mentioned substances is even higher for smokers.

Possible Risk Factors

Medical Conditions

Having certain lung diseases, such as tuberculosis or bronchitis, for many years may increase the risk of developing lung and bronchus cancer.

Lifestyle Factors

Some evidence suggests that a diet high in fruits and vegetables may help protect against lung and bronchus cancer. But any positive effect of fruits and vegetables on reducing risk would be much less than the negative effects of smoking in increasing risk. It should be noted, however, that two large studies examining the possible role of antioxidant supplements in reducing the risk of lung and bronchus cancer found that smokers who took beta carotene supplements actually had an *increased* risk. The results of these studies suggest that smokers should avoid taking beta carotene supplements.

Environmental Exposures

High levels of arsenic in drinking water may increase the risk of lung cancer. In addition, air pollution appears to slightly raise the risk of lung and bronchus cancer in cities.

Other Risk Factors That Have Been Investigated

Marijuana use is believed to increase the risk of lung and bronchus cancer due to its high tar content. However, the effects of this possible association have proven difficult to study due to its illegal nature and the fact that many marijuana users also smoke cigarettes.

Talc is a mineral that may contain asbestos in its natural form. Previous studies suggested that talc miners and millers have a higher risk of lung and bronchus cancer due to their exposure to industrial grade talc but recent studies did not found this association. Talcum powder is made from talc. Since 1973, all home-use talcum products, such as baby, body, and facial powders, have been asbestos-free by law.

Lung and Bronchus Cancers in Children

Fewer than 3% of lung and bronchus cancers occur in individuals under the age of 45. Pleuropulmonary blastoma (PPB) is a rare type of childhood lung cancer that occurs most often in children under the age of four. As few as 10 to 20 individuals are diagnosed with

PPB each year in the United States.

For More Information / References

Much of the information contained in this summary has been taken directly from the following sources. This material is provided for informational purposes only and should not be considered as medical advice. Persons with questions regarding a specific medical problem or condition should consult their physician.

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