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| **Health Consultation** |
| Evaluation of Residential Indoor Air near the Walton and Lonsbury Superfund Site in Attleboro, Massachusetts  EPA Facility ID: MAD001197755 |
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| September 18, 2014 |
| Prepared by: |
| Massachusetts Department of Public Health  Bureau of Environmental Health  Community Assessment Program  Under a Cooperative Agreement with the  Agency for Toxic Substances and Disease Registry  Atlanta, Georgia |

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Summary

**Introduction:** The Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH), under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), assesses the presence and nature of health hazards at sites proposed to the National Priorities List (NPL). MDPH/BEH initiated this health consultation in response to the Walton and Lonsbury site in Attleboro, Massachusetts being proposed to the NPL. The purpose of this evaluation is to address community concerns related to environmental contaminants in the indoor air of nearby residences and the potential for health effects.

Possible exposure pathways associated with this site include inhalation of contaminated indoor air as well as dermal exposure to and incidental ingestion of contaminated soil, sediment, and surface water. The indoor air pathway was selected for evaluation first because of the potential for nearby residents to be exposed to volatile chemicals in their homes throughout the year, while potential exposure to other pathways is seasonal. Potential exposure via other pathways will be evaluated in future health consultations.

MDPH/BEH evaluated cancer incidence near the site to address concerns voiced by community members regarding cancer. Because recent scientific evidence indicates that exposure to trichloroethylene, one of the primary site contaminants of concern, may result in a small increased risk of cardiac malformations in the developing fetus, MDPH/BEH also searched the birth defects registry for reports of cardiac malformations in the neighborhood near the site.

The top priority of ATSDR and MDPH/BEH is to ensure that the community has the best information possible to safeguard its health.

**Conclusions:** MDPH/BEH has reached four important conclusions in the health consultation.

**Conclusion 1:** MDPH/BEH concludes that breathing levels of trichloroethylene and tetrachloroethylene detected in houses in the vicinity of the Walton and Lonsbury Superfund Site is not expected to harm people’s health.

**Basis for Decision:** From 2000 through 2010, indoor air samples were collected from 13 houses near the Walton and Lonsbury site and analyzed for volatile organic compounds (VOCs), including trichloroethylene and tetrachloroethylene. Trichloroethylene and tetrachloroethylene had been previously identified as primary contaminants of concern in contaminated groundwater beneath the site.

During its review of the indoor air sampling data, MDPH/BEH identified some limitations (e.g., limited number of samples and some data quality concerns) such that additional sampling is desirable and would improve the reliability of the data. Given the available data, MDPH/BEH concludes that the detected levels of trichloroethylene and tetrachloroethylene were below levels known to result in harmful, non-cancer health effects. In addition, MDPH/BEH does not consider the detected trichloroethylene and tetrachloroethylene indoor air levels to present an elevated cancer risk.

**Next steps:**

* Because of the limited number of indoor air samples collected from the 13 houses and because no samples have been collected since 2010, MDPH/BEH recommends that EPA re-sample indoor air and soil gas at all 13 houses to confirm that the trichloroethylene and tetrachloroethylene indoor air levels are still below levels that could harm people’s health.
* Three of the 13 houses (houses I, J, and K) currently have sub-slab depressurization systems, which help prevent VOCs (including trichloroethylene and tetrachloroethylene) from entering the air inside the houses. MDPH/BEH recommends that EPA re-sample the indoor air of houses I, J, and K to verify that these systems are operating as intended. MDPH/BEH also recommends that homeowners ensure that the systems are properly maintained and serviced.

**Conclusion 2:** MDPH/BEH concludes that breathing other non-site related VOCs detected in the indoor air of houses near the Walton and Lonsbury site is not expected to harm people’s health.

**Basis for Decision:** A number of VOCs besides trichloroethylene and tetrachloroethylene were detected in the indoor air samples collected from 13 houses near the Walton and Lonsbury site. These contaminants are most likely not site-related considering that they have not been identified as site contaminants of concern and indoor air typically contains VOCs from many common household products, such as paints, glues, cleaners, and cigarette smoke. In any case, the levels of VOCs detected in the indoor air of houses near the Walton and Lonsbury site were below levels known to result in harmful, non-cancer health effects. In addition, MEPH/BEH does not consider the detected VOC levels to present an elevated cancer risk.

**Next steps:** Although health effects from exposure to non-site related VOCs in the indoor air of houses near the site are not expected, MDPH/BEH recommends that homeowners consider the following tips to improve their indoor air quality:

* Be aware of household products that contain VOCs. Don’t buy more chemicals than you need.
* Store chemicals (e.g. gasoline, paints, pesticides) in tightly-sealed containers outdoors or in a detached garage/shed, if possible, and away from family living spaces.
* Keep your home properly ventilated by using exhaust fans or opening windows when doing home renovations, do-it-yourself projects, or hobbies that require the use of chemicals.
* Remove unused products from the home. Check with your city/town government about properly disposing of unwanted paints, solvents and other related products (e.g., at hazardous waste collection events).
* Don’t smoke indoors.

**Conclusion 3:** MDPH/BEH cannot conclude whether trichloroethylene or tetrachloroethylene vapors from contaminated groundwater beneath the Walton and Lonsbury site are currently affecting the indoor air of downgradient houses that have not been sampled yet or if conditions have changed in previously sampled houses.

**Basis for Decision:** Trichloroethylene continues to be detected (based on sampling data collected in 2012) in off-site groundwater monitoring wells near houses J and K, and tetrachloroethylene is present in soil gas beneath houses M and N. However, no indoor air or soil gas samples have been collected at houses along North Avenue south of houses M and N. Thus, MDPH/BEH cannot determine whether vapor intrusion is currently affecting houses near the leading edge of the trichloroethylene/tetrachloroethylene groundwater plume.

**Next steps:**

* MDPH/BEH recommends that EPA collect indoor air and soil gas samples from all 13 houses previously sampled and from additional houses to the south along North Avenue to further characterize the extent of vapor intrusion.
* MDPH/BEH recommends that any additional indoor samples be collected from the main living areas of the home to better estimate actual exposures.
* Upon request, MDPH/BEH will review and provide health-based input on EPA sampling plans developed to evaluate the vapor intrusion pathway in the vicinity of the Walton and Lonsbury site.

**Conclusion 4:** Based on a review of cancer and birth defects surveillance data, no significant elevations or unusual trends in cancer incidence and no cardiac malformations were identified in the area surrounding the Walton and Lonsbury site.

**Basis for Decision:** MDPH/BEH completed an evaluation of cancer incidence in the census tract that the Walton and Lonsbury site is located in (6313). MDPH/BEH evaluated the incidence of those cancer types that are most strongly associated with exposure to the primary site contaminants of concern, as well as childhood cancer, for a 25-year period (1983-2007). No unusual trends were noted for any cancer types. Data from the MDPH birth defects registry were reviewed for cardiac malformations because exposure to trichloroethylene during pregnancy is associated with a small increased risk of fetal cardiac defects. The birth defects surveillance data were queried for all live births to residents of the neighborhood near the Walton and Lonsbury site between 2000 and 2010 for these types of birth defects. This 11-year time period constitutes the period for which the most recent and complete data were available at the initiation of this analysis. No cardiac malformations were identified in the vicinity of the site.

**Next steps:** None

**For More Information:** If you have concerns about your health, you should contact your health care provider. You may also call ATSDR at 1-800-CDC-INFO or MDPH/BEH at 617-624-5757 and ask for information on the Walton and Lonsbury Superfund Site.

Background and Statement of Issues

The Walton and Lonsbury site is located at the corner of North Avenue and Walton Street (78 North Avenue) in Attleboro, Massachusetts, approximately 0.2 miles west of Interstate 95. North and west of the site are industrial properties located along Walton Street. East of the site, across North Avenue, is the City of Attleboro’s Hayward Recreation Center, which includes a community pool, playground, and athletic fields. Southeast of the site are residential properties located along North Avenue and Paulette Lane. Residences southeast of the site are adjacent to a small brook (Bliss Brook) flanked by a narrow strip of wetlands (EPA 2010). Adjacent to (south of) the site is a wetland area that extends approximately 1,000 feet south to Deanville Road and abuts residential properties along North Avenue. The closest residential property is located approximately 50 feet east at the intersection of North Avenue and Paulette Lane (Figure 1) (MassGIS 2005).

The former Walton and Lonsbury facility occupied a site consisting of approximately 2.7 acres identified as lots 1, 2D, 4, and 5 on Attleboro Tax Map 89 (Attleboro 1995; EPA 2010). From about 1940 to 2007, Walton and Lonsbury, Inc. operated a chrome plating operation on site. Additional processes conducted included some copper plating, parts degreasing, stripping with acids, aqueous rinsing, grinding, and polishing. Chemicals historically used on site included but were not limited to chromic oxide; cyanide; lead sulfate; paint thinner; 1,1,1-trichloroethane; and trichloroethylene (EPA 2010; RCA 2001).

On-site building materials, soil, sediment, and groundwater were contaminated by historical disposal of industrial process waste to on-site wetlands and possibly directly to groundwater via a dry well, as well as by chemical spills, leaking tanks and localized deposition of airborne contaminants from facility air vents. Contaminants of concern identified by EPA and Resource Control Associates (RCA) (a former environmental contractor for Walton and Lonsbury) include metals (chromium, hexavalent chromium, cyanide, and lead), polycyclic aromatic hydrocarbons [benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and dibenzo(a,h)anthracene], petroleum hydrocarbons, and volatile organic compounds [1,1-dichloroethylene, 1,1-dichloroethane, cis-1,2-dichloroethylene, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, and vinyl chloride] (EPA 2010; RCA 2001).

Surface water flow in the wetland area adjacent to (south of) the site is toward the south and has re-distributed contaminants throughout this wetland area. Groundwater beneath the site flows to the southeast toward Bliss Brook, a wetland area, and residential neighborhoods located along North Avenue and Paulette Lane. Groundwater primarily contaminated with metals, including hexavalent chromium, extends southeast from the site beneath several residential properties on North Avenue and Paulette Lane and discharges to Bliss Brook. Groundwater primarily contaminated with chlorinated solvents extends off site to the south-southeast beneath several residential properties along North Avenue (Figure 1) (EPA 2010, RCA 2001).

Starting in 1983, numerous environmental investigations have occurred at the Walton and Lonsbury site (MassDEP 2013a). Several rounds of indoor air and soil gas sampling were conducted between 2000 and 2010. Houses were selected for indoor air and soil gas testing based on detections of elevated concentrations of VOCs in groundwater samples collected from nearby monitoring wells (RCA 2000). During this time, indoor air samples were collected from 13 houses downgradient of the Walton and Lonsbury site and houses were sampled an average of 3 times (Table 1). Soil gas samples were also collected from 10 of these houses on 1 or 2 occasions (Table 2) (EPA 2008a, 2009, 2010; MassDEP 2010a and 2010b; RCA 2001). In 2009, Massachusetts Department of Environmental Protection (MassDEP) requested the U.S. Environmental Protection Agency’s (EPA’s) assistance to address the site (EPA 2010). In 2010, EPA contractors conducted a Preliminary Assessment/Site Inspection (PA/SI) of the closed facility. During the PA/SI, chromic acid and large quantities of chromic acid sludge were discovered in the four plating tanks. Additionally, the PA/SI concluded that on- and off-site soil, sediment, and groundwater have been impacted by releases from the facility. Contaminated surface soil was identified on several residential properties abutting Bliss Brook along Paulette Lane and the east side of North Avenue (EPA 2010; 2011a).

On October 15, 2010, at the request of the City of Attleboro’s Health Director (Mr. James Mooney), the MDPH/BEH completed an evaluation of cancer incidence in the census tract where the Walton and Lonsbury site is located (6313). MDPH/BEH evaluated the incidence of those cancer types that are most strongly associated with exposure to the primary site contaminants of concern, as well as childhood cancer, for a 25-year period (1983-2007). No statistically significant elevations were noted and for most cancer types evaluated the number of observed diagnoses were approximately equal to the number of expected diagnoses. In summary, no unusual trends were noted for any cancer types. See Appendix A for details of the cancer evaluation and the cancer incidence summary tables.

On October 19, 2010, EPA’s Superfund Removal Program initiated a removal action. To date, the EPA Removal Program has removed hazardous materials and waste (e.g., asbestos and chromium hydroxide sludge), removed plating tanks, demolished the building, and initiated remediation of contaminated soil and sediment on site and in the adjacent wetland area. The EPA Removal Program is also in the final stages of excavating and capping contaminated soil at residential properties located along North Avenue and Paulette Lane (EPA 2011a and 2013b).

On September 18, 2012, the Walton and Lonsbury site was proposed to the EPA National Priorities List (NPL) and the site was added to the NPL on May 24, 2013 (Federal Register 2012 and 2013). Additional delineation of the extent of contamination and additional off-site remediation activities will be continued by the Superfund Remedial Program (EPA 2013b).

MDPH/BEH, under a cooperative agreement with the U.S. Agency for Toxic Substances and Disease Registry (ATSDR), assesses the presence and nature of health hazards at sites proposed to the NPL. As part of this agreement, MDPH/BEH reviewed indoor air data collected in residential homes near the Walton and Lonsbury site to evaluate whether VOCs detected in indoor air pose a public health threat to area residents. Possible exposure pathways associated with this site include inhalation of contaminated indoor air as well as dermal exposure to and incidental ingestion of contaminated soil, sediment, and surface water. The indoor air pathway was selected for evaluation first because of the potential for nearby residents to be exposed to volatile chemicals in their homes. Potential exposure via other pathways, such as direct contact with soil, is expected to be seasonal and intermittent. Because recent scientific evidence indicates that exposure to trichloroethylene, one of the primary site contaminants of concern, may result in a small increased risk of cardiac malformations in the developing fetus, MDPH/BEH also requested that the MDPH’s Bureau of Family Health and Nutrition, Center for Birth Defects Research and Prevention (MCBDRP) search the birth defects registry for reports of cardiac malformations in the neighborhood near the site.

Methods

Information on site history and existing indoor air and soil gas sampling data, as well as recent groundwater data, was reviewed. Indoor air concentrations of VOCs were compared to ATSDR health-based comparison values for screening purposes; these include comparison values cancer risk evaluation guides (CREGs) and environmental media evaluation guides/minimal risk levels (EMEGs/MRLs). The ATSDR comparison values are specific concentrations of a chemical in a media (in this case, air) that are used by health assessors to identify environmental contaminants that require further evaluation. These comparison values are developed based on health guidelines and assumed exposure situations that represent conservative (worst case) estimates of human exposure. When a chemical is detected at a concentration less than its respective comparison values, exposure is not expected to result in adverse health effects. It should be noted that chemicals detected at concentrations that exceed their respective comparison values do not necessarily represent a health threat, but that these chemicals warrant a more detailed, site-specific evaluation (ATSDR 2005; ATSDR 2012).

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 the compound. Therefore, if a concentration of a chemical is greater than the appropriate comparison value, the potential for exposure to the chemical should be further evaluated to determine whether exposure is occurring and whether health effects might be possible as a result of that exposure (ATSDR 2005).

For chemicals that do not have ATSDR comparison values, EPA Reference Concentrations (RfCs) or EPA Regional Screening Levels (RSLs) for Chemical Contaminants at Superfund Sites were used. An RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure of a chemical to the human population (including sensitive subpopulations) that is likely to be without risk of deleterious noncancer effects during a lifetime. RSLs are chemical-specific concentrations for individual contaminants in air, drinking water, and soil that EPA uses to determine whether further investigation or site cleanup is warranted. RSLs are based on default exposure parameters and factors that represent Reasonable Maximum Exposure conditions for long-term/chronic exposures that are protective of humans, including sensitive populations.

In addition to comparison values, MDPH compared indoor air concentrations to concentrations detected in ambient (outdoor) air samples where available and to typical concentrations detected in homes. It is important to note that indoor air typically contains VOCs from sources such as household products, building materials, chlorinated tap water, industry, and motor vehicle traffic. Typical concentrations of chemicals are commonly referred to as “background” concentrations. In general, the most commonly detected VOCs in indoor air include benzene and chlorinated solvents, such as chloroform, carbon tetrachloride, tetrachloroethylene, 1,1,1-trichloroethylene, and trichloroethylene (EPA 2011b). The background concentrations used for comparison in this report are from EPA and the New York Department of Health (EPA 2011b; NYDOH 2006).

Previous site investigations identified certain VOCs as site contaminants of concern. Thus, this evaluation will focus on the contaminants of concern detected in indoor air above comparison values. Evaluation of other VOCs detected in indoor air (e.g., from normal use of household products) are presented in Appendix B and briefly summarized in this report.

Environmental Sample Results

Indoor air

Between 2000 and 2010, nine rounds of indoor air samples were collected from residences near the Walton and Lonsbury site. Over a 1 year period between April 2000 and April 2001, Resource Control Associates, Inc. (RCA) on behalf of Walton and Lonsbury, Inc., collected four rounds of indoor air samples from a total of 12 homes (five homes twice and seven homes once) near the site (Houses A, B, D, and F through N), as well as one home located approximately 0.4 miles north of (up-gradient of) the site to characterize background indoor air conditions (background house). One outdoor air sample was collected during the 2000 - 2001 period. Samples were collected over 24-hours in evacuated stainless steel canisters. No sources of chlorinated VOCs were identified in the basements of the homes sampled during a pre-sampling inspection; however, a heating oil tank (a potential indoor air source of petroleum-related VOCs) was identified in house K. Basement windows were closed at the time of sampling, but no information was available regarding windows on other levels of the home (RCA 2001). MDPH/BEH’s evaluation of the sample results indicated a data quality issue with sample results collected by RCA in January 2001. Unlike other sampling events, trichloroethylene results for the January 2001 samples were very similar, uniformly between 1-2 ppb, across all houses sampled including an upgradient house located approximately 0.4 miles northwest of the site sampled to establish background conditions. MDPH/BEH believes that the similarity of these results across houses, including the background house, is an indication that sample quality was compromised (e.g., by cross contamination). No information on quality control samples was included in the RCA report and no outdoor air samples were collected. No other chemicals reported during the January 2001 sampling round appear to be affected. Due to the questionable validity of the January 2001 sample results for trichloroethylene, these results are not included in the quantitative exposure evaluation.

From 2008 through 2010, EPA, MassDEP, and MassDEP’s contractor, Environmental Strategies and Management (ESM), conducted five rounds of indoor air sampling. Outdoor air samples were also collected during three rounds. Depending on the round of sampling, samples were collected from one to nine houses (houses D through N) near the site (Figure 1). Samples in four out of five rounds were collected over a 24-hour period in evacuated stainless steel canisters. Samples collected in February 2010 were grab samples (collected over a short period of time) collected in Tedlar bags and analyzed in a mobile laboratory (EPA 2008a, 2009, 2010; MassDEP 2010a and 2010b). Samples collected during the February 2010 sampling round had high detection limits, with the detection limits for trichloroethylene and tetrachloroethylene much higher than their ATSDR comparison values, thus the results of this sampling round provide limited information at best regarding possible risks from vapor intrusion.

During sample collection in 2008, EPA personnel noted the presence of several potential indoor air sources of VOCs (e.g., wood stains, paints, glues, cleaners, and a heating oil tank (in house J only)) in the basements of houses I and J. During sample collection in house J on January 14-15, 2010, ESM personnel noted the presence of several potential indoor air sources of VOCs (e.g., paints, cleaners, and citronella torch fuel) in the basement. No information was available regarding the presence of potential indoor air sources of VOCs for the other sampling rounds in 2009 and 2010. With the exception of the January 2010 sampling round, no information was available regarding whether the windows in the houses were open or closed at the time of sampling for samples collected during 2008-2010 (EPA 2008a, 2009, 2010; ESM 2010; MassDEP 2010a and 2010b). Without such information it is impossible to determine whether results are representative of typical indoor concentrations given the dilution effects that open windows would provide.

Overall, about one third of the houses were sampled on four or five occasions, one third on three occasions, and one third on one or two occasions. A total of 59 indoor air samples were collected over all sampling rounds from the 13 houses plus one background house (Table 1) (EPA 2008a, 2009, 2010; MassDEP 2010a and 2010b).

Results of indoor air sampling from 2000-2010 showed a total of 41 VOCs detected in one or more of the 59 samples collected (not all VOCs were detected in all samples or all houses). Of these, 14 VOCs (acrylonitrile; benzene; benzyl chloride; bromodichloromethane, 1,3-butadiene; carbon tetrachloride; chloroform; dibromochloromethane; 1,4-dichlorobenzene; dichlorodifluoromethane; 1,2-dichloroethane; tetrachloroethylene; trichloroethylene; and 1,2,4-trimethylbenzene) were detected in some houses at concentrations above health-based comparison values. Of the VOCs detected in indoor air at concentrations above comparison values, tetrachloroethylene and trichloroethylene have been identified as contaminants of concern at the Walton and Lonsbury site by EPA and RCA, based on detections of these contaminants in environmental media at the site. Hence, these chemicals are the primary focus of this evaluation (Tables 3, 4, and 5). The evaluation of other chemicals besides tetrachloroethylene and trichloroethylene detected in indoor air that exceeded comparison values is presented in Appendix B and briefly summarized in the Discussion section of this report. Details regarding indoor air concentrations of chemicals evaluated are included in the Exposure and Health Effects Evaluation section, Tables 3-5, and Appendix B of this report.

Soil gas

The concentrations of volatile chemicals in soil gas below the foundation of a building can be helpful in assessing the potential for chemicals to migrate from sub-surface sources such as contaminated groundwater or soil to indoor air of overlying buildings. However, migration of chemical vapors from the subsurface into indoor air varies substantially between buildings due to differences in geology and building characteristics. Thus elevated soil gas concentrations do not always mean that indoor air will be impacted. Also, under the right conditions, changes in the pressure differential between indoor air and soil gas may cause chemicals from indoor sources to migrate into soil gas beneath homes making source attribution difficult (ITRC 2007).

In three out of four indoor air sampling rounds, from 2008 through 2010, MassDEP and EPA collected soil gas samples from beneath the foundations of 10 houses (Houses D through H and J through N), all of which had indoor air samples collected on or starting the day of soil gas sampling (Figure 1 and Tables 1 and 2). Four houses were sampled on two occasions and six houses were sampled once for a total of 20 sub-slab soil gas samples (Table 2). Soil gas samples were analyzed for VOCs, including trichloroethylene and tetrachloroethylene; however, tetrachloroethylene results for soil gas samples collected from Houses D and E were rejected by MassDEP laboratory analysts due to sample analysis quality control issues (Table 6) (EPA 2008a and MassDEP 2010a and b).

Trichloroethylene was detected in soil gas samples collected from six of the 10 houses sampled (houses D, E, J, K, M, and N). Lower concentrations (0.2-0.3 ppb) of trichloroethylene were detected in soil gas samples from houses D, E, and K and higher concentrations (11.2-155 ppb) were detected in soil gas samples from houses J, M, and N (Table 6). Trichloroethylene has been detected in indoor air samples in houses J, M, and N, but not in samples collected from houses D, E, and K (Table 4).

Tetrachloroethylene was detected in soil gas samples collected from four of the 8 houses sampled for which valid sample results were available (houses H, J, M, and N) with concentrations ranging from 0.5 ppb to 24.7 ppb (Table 6). Tetrachloroethylene has been detected in indoor air samples collected from house J, but not from the other three houses (Table 5).

Groundwater

In 2011, measured depth to groundwater ranged from 3-16 feet below ground surface in on- and off-site monitoring wells (ES&M 2011). Groundwater beneath the site flows to the southeast toward Bliss Brook, a wetland area, and residential neighborhoods located along North Avenue and Paulette Lane. Groundwater primarily contaminated with chlorinated solvents extends off site to the south-southeast beneath several residential properties along North Avenue. Groundwater primarily contaminated with metals, including hexavalent chromium, extends southeast from the site beneath several residential properties on North Avenue and Paulette Lane and discharges to Bliss Brook (Figure 1) (EPA 2010, RCA 2001). VOCs were first analyzed for and detected in groundwater samples collected from monitoring wells on site in 1987 and in samples collected from off-site monitoring wells in 1998 (RCA 2001). VOCs in groundwater began migrating from the site sometime after the facility began operating (1940) and before VOCs were first detected in the off-site monitoring wells (1998).

MDPH/BEH reviewed VOC results for 54 recent groundwater samples collected on or near the Walton and Lonsbury site during 2011 and 2012. Groundwater contaminant concentrations were compared with MassDEP’s GW-2 standards, which were developed to protect indoor air from volatile contaminants in groundwater. During 2011 and 2012, trichloroethylene and tetrachloroethylene were detected in groundwater samples collected from seven of nine on-site and three of 31 off-site monitoring wells (Figure 1). Trichloroethylene was detected at concentrations exceeding its MassDEP GW-2 standard (30 ppb) in samples collected from one on-site well (MW-6S) and two off-site wells (MW-15 and RCA-17). Tetrachloroethylene was not detected above its MassDEP GW-2 standard (50 ppb) (Table 7) (ES&M 2011; MassDEP 2012).

Exposure and Health Effects Evaluation

To evaluate the potential for health effects from exposure to contaminants in indoor air, exposure dose and cancer risk estimates were calculated for the inhalation pathway for trichloroethylene and tetrachloroethylene. Exposure dose and cancer risk calculations were completed using conservative (health protective) assumptions. Residents were assumed to be exposed to the maximum detected concentration for 33 years[[1]](#footnote-2), 52 weeks per year, 7 days per week, and 24 hours per day.

To evaluate potential cancer effects, detected indoor air concentrations of contaminants of concern were multiplied by EPA cancer slope factors (CSF) to estimate the potential cancer risk (Table 9). See Appendix C for example exposure concentration and cancer risk calculations.

To evaluate potential non-cancer health effects, detected contaminant concentrations were directly compared to ATSDR EMEGs/MRLs (0.37 ppb for trichloroethylene and 40 ppb for tetrachloroethylene) and EPA RfCs (0.37 ppb for trichloroethylene and 5.9 ppb for tetrachloroethylene) and evaluated for potential health concerns (Table 8).

The ATSDR EMEG/MRL for trichloroethylene is taken from the EPA RfC, which is based on three experimental studies of animals exposed to trichloroethylene in drinking water (ATSDR 1997).

The most sensitive adverse effects identified by these studies were cardiac malformations in the developing fetus (Johnson 2003) and adverse effects to the immune system (Keil 2009) and kidney (NTP 1988). EPA selected the studies showing fetal cardiac malformations and effects to the immune system, specifically decreased thymus weight, as the primary studies upon which to derive an RfC and identified the kidney study as an important supporting study. EPA used physiologically based pharmacokinetic modeling to estimate what a dose in drinking water given to study animals might equate to for humans exposed to trichloroethylene in air (human-equivalent dose). EPA applied uncertainty factors to the human-equivalent doses from each of the studies to produce preliminary RfCs. Uncertainty factors are applied as a way to account for sources of uncertainty (e.g., extrapolation from animal studies to humans) in the calculation of RfCs (EPA 2011c). Below are brief summaries of the two primary studies and the supporting study:

* Johnson et al. (2003) reported increased rates of fetal cardiac malformations in the offspring of pregnant rats administered TCE in drinking water throughout pregnancy. EPA used this study to predict internal dose levels of TCE in the rat for each of the TCE in drinking water exposure groups used in the study. EPA then used statistical modeling to predict an internal dose level that was below any level that the study authors reported as producing an adverse effect. The model-estimated “point of departure,” or POD, was then converted, using conservative assumptions, into a “human equivalent concentration” (HEC) of TCE in air of 20 µg/m3. The HEC represents a level of TCE in air that is unlikely to result in adverse effects (in this case, fetal cardiac malformations following exposure throughout pregnancy). EPA added a margin of safety of 10 to the HEC to derive a reference concentration (RfC) of 0.37 ppb (2 µg/m3) (EPA 2011c).
* Keil et al. (2009) found decreased thymus weight in female mice exposed to trichloroethylene in drinking water. EPA modeling estimated a human equivalent dose of 33 ppb (190 µg/m3). EPA applied an uncertainty factor of 100 to this concentration resulting in a preliminary RfC of 0.33 ppb (2 µg/m3) (EPA 2011c).
* The supporting National Toxicology Program study (1988) showed kidney effects (toxic nephropathy) in female rats exposed to trichloroethylene in drinking water. EPA modeling estimated a human equivalent dose of 5.6 ppb (30 µg/m3). EPA applied an uncertainty factor of 10 to this concentration resulting in a preliminary RfC of 0.6 ppb (3 µg/m3) (EPA 2011c).

EPA selected the final RfC (0.37 ppb / 2 µg/m3) based primarily on the preliminary RfCs from the two primary studies (EPA 2011c).

The ATSDR EMEG/MRL for tetrachloroethylene was based on a 1992 study of occupational exposures. The EPA RfC was derived more recently and thus considers additional studies. The EPA RfC for tetrachloroethylene is based on studies of occupational exposures resulting in neurotoxic effects (e.g., changes in cognitive function and color vision). The lowest exposure concentration at which neurotoxic effects (changes to color vision) were observed (known as the lowest observable effect level (LOAEL)) was 42,000 µg/m3 (6,200 ppb) or 15,000 µg/m3 (2,200 ppb) after adjusting the concentration to account for 24-hour exposures. The LOAEL for changes in cognitive function was 156,000 µg/m3 (23,000 ppb) or 56,000 µg/m3 (8,300 ppb) after adjusting the concentration to account for 24-hour exposures. To derive the RfC (5.9 ppb / 40 µg/m3), the midpoint of the two LOAELs was taken and an uncertainty factor of 1,000 was applied to account for inter-individual variability, extrapolation from a LOAEL instead of a no observable effects level (NOAEL), and database uncertainty (EPA 2012c).

Childhood Inhalation Exposure

Early childhood may be an especially vulnerable lifestage for cancer and noncancer health effects for two main reasons: (1) increased susceptibility of children to some contaminants; and (2) elevated air intake in children due to ventilation rate per body weight (EPA 2005 and 2008b).

EPA’s inhalation unit risk factors are based on epidemiological studies of adults, primarily in occupational settings, and animal bioassays that are not designed to isolate the effects of early life exposures (EPA 2005). EPA’s Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens recommends two approaches for assessing cancer risks of chemicals with a mutagenic mode of action (MOA): 1) using chemical-specific risk estimates that account for early-life susceptibility or 2) applying default age-dependent adjustment factors (ADAFs) (EPA 2005). Because MOAs have not been determined for many chemicals and carcinogens may have multiple modes of action during different life stages, a conservative approach is to apply ADAFs to mutagenic chemicals, as well as to chemicals without an established mode of action (OEHHA 2009). Thus, ADAFs were included in all child cancer risk calculations. See Appendix C for example exposure concentration and cancer risk calculations.

Trichloroethylene

As stated previously, MDPH considers the trichloroethylene results from one of the nine sampling rounds, the January 2001 sampling round, to be of insufficient sample quality, and thus, these results are not included in the following quantitative evaluation.

Trichloroethylene has been detected in indoor air samples from four (houses G, I, J, and M) out of the 13 houses sampled near the site. Trichloroethylene was detected in all four of these houses at concentrations exceeding the ATSDR CREG of 0.045 ppb. Two of the four houses, houses J and I, had detections of trichloroethylene above the ATSDR EMEG/MRL of 0.37 ppb in indoor air samples (house J 0.87 ppb in April 2000, 0.53 ppb in December 2008, and 0.40 ppb in March 2009 and house I 0.82 ppb in April 2000) (Table 4).

To reduce the indoor air trichloroethylene levels, in 2010, MassDEP installed a sub-slab depressurization system in house J designed to mitigate the migration of volatile chemicals from the subsurface into indoor air (ESM 2010). Additionally, it should be noted that houses I and K have radon mitigation systems (a type of sub-slab depressurization system). The system in house I was installed in 2008 (Burkhamer 2014). The system in house K was installed prior to the first indoor air sampling round in 2001 (RCA 2001).

MDPH/BEH evaluated past exposure scenarios for houses I and J because these houses had the maximum concentrations detected among all the houses sampled and no other house has exceeded the EPA RfC and ATSDR EMEG/MRL for trichloroethylene. The maximum detected concentration at house J (0.87 ppb) and house I (0.82 ppb) were selected as the exposure concentrations. For houses J and I, exposure estimates exceed the EPA RfC and ATSDR EMEG/MRL of 0.37 ppb (2 µg/m3) (Table 8).

Because detected concentrations of trichloroethylene were higher than comparison values, MDPH/BEH conducted a more detailed evaluation. For both houses I and J indoor air concentrations exceeding the EMEG/MRL were limited to the basement areas (Table 4). For house I, the basement is unfinished, and hence exposure opportunities would be expected to be infrequent compared to those in the rest of the house. The basement of house J is finished and reportedly is used often. The ATSDR EMEG/MRL assumes 24 hour per day exposure; however, exposure opportunities in the basement would likely be less than 24 hours per day, 7 days per week, for 52 weeks of the year.

The maximum concentration detected was four times lower than the 3.7 ppb concentration (human equivalent dose) derived by EPA from the Johnson study and described as presenting a small risk of cardiac malformations in the fetus as a result of in utero exposures. Additionally, the maximum concentration detected was six times and nearly 40 times lower, respectively, than the human equivalent doses for the Keil study (33 ppb) and the NTP study (5.6 ppb). Considering the lower exposure opportunities along with the maximum concentrations detected, as well as the conservative derivation of the ATSDR EMEG/MRL, we would not expect non-cancer health effects to result from the measured indoor air levels.

Cancer risks from exposure to trichloroethylene in indoor air were calculated based on the maximum detected concentration at house J (0.87 ppb) and house I (0.82 ppb) to represent a worst-case exposure scenario. Estimated cancer risks for these two houses were 1.6 in 100,000 for children (both houses I and J) and 7.7 in 1,000,000 for adults in house I and 8.1 in 1,000,000 for adults in house J (Table 9). This means that for children the risk estimate would be approximately 2 excess cancer diagnoses in a population of one hundred thousand and for adults between approximately 7-8 extra cancer diagnoses in a population of one million. Environmental regulatory agencies consider cancer risks less than 1 in 10,000 (or one excess cancer diagnosis in a population of 10,000) to represent no unusual cancer risk. Thus, exposure to trichloroethylene at the levels found inside the houses sampled would not present an unusual cancer risk to children or adults living in those houses. These exposure estimates are very conservative and are intended to indicate worst-case scenarios.

Note that including the invalid sample data from the January 2001 sampling round in the above quantitative evaluation would not have changed the results. The maximum concentrations of trichloroethylene detected during this sampling round exceeded the ATSDR EMEG/MRL but did not exceed the toxicity values (human equivalent doses) that this comparison value is based on. Additionally, including the maximum concentration detected from this sampling round would not have resulted in an unusual cancer risk. All houses in the vicinity of the Walton and Lonsbury site included in the January 2001 sampling round were sampled on at least two other occasions and, with the exception of houses I and J, none of the houses had concentrations exceeding the ATSDR EMEG/MRL (Table 4). As noted previously, sub-slab depressurization systems designed to mitigate the migration of volatile chemicals from the subsurface into indoor air were installed in houses I and J.

Tetrachloroethylene

Tetrachloroethylene has been detected in indoor air samples from four houses (G, I, J, and L) out of 13 sampled at concentrations ranging from 0.06 to 6.1 ppb. Tetrachloroethylene concentrations exceeded the ATSDR CREG (0.57 ppb) in indoor air samples collected from house L (3.47 ppb basement sample and 6.1 ppb first floor sample collected on 1/18/01) and house J (0.59 ppb basement sample collected on 4/22/2000). Additionally, the tetrachloroethylene concentration in one sample collected from house L slightly exceeded the EPA RfC of 5.9 ppb (6.1 ppb), but no concentrations exceeded the ATSDR Chronic EMEG/MRL of 40 ppb (Table 5).

As mentioned above, tetrachloroethylene was detected in the two samples collected from house L in 2001; however, it has not been detected in three samples collected from house L in 2010 (there was a long period with no sampling in house L). Similarly, for house J, tetrachloroethylene concentrations have not exceeded the ATSDR CREG in any indoor air samples since 2000.

The maximum concentration of tetrachloroethylene was 6.1 ppb (41.4 ug/m3), which was detected in house L. This slightly exceeds the EPA RfC of 5.9 ppb. However, the maximum detected concentration was about four orders of magnitude below the lowest levels associated with neurotoxic effects in occupational studies. Over four rounds of sampling in house L, only one sample exceeded the RfC and tetrachloroethylene was not detected during three out of the four rounds. No other houses had an exceedance of the RfC. Thus, based on the available sampling data, non-cancer health effects from exposure to tetrachloroethylene in indoor air would not be expected.

Based on the maximum detected concentration of tetrachloroethylene, the calculated cancer risk is approximately 4.6 in 1,000,000 for adults and 8.8 in 1,000,000 for children which means for adults the risk estimate is approximately five excess cancer diagnoses in a population of one million and for children approximately 9 excess cancer diagnoses in a population of one million. Thus, even the maximum tetrachloroethylene concentration detected in all houses would pose no unusual cancer risk.

Other VOCs

In indoor air samples collected from 2000-2010, 39 VOCs were detected in addition to tetrachloroethylene and trichloroethylene. Of these, 11 VOCs (acrylonitrile; benzene; benzyl chloride; bromodichloromethane, 1,3-butadiene; carbon tetrachloride; chloroform; dibromochloromethane; 1,4-dichlorobenzene; dichlorodifluoromethane; 1,2-dichloroethane; and 1,2,4-trimethylbenzene) not identified by EPA or RCA as site contaminants of concern were detected at concentrations above health-based comparison values. These VOCs are evaluated in Appendix B of this report. Based on MDPH/BEH’s evaluation, exposure to these VOCs in indoor air would not be expected to result in non-cancer health effects or unusual cancer risks.

Discussion

This health consultation reviewed available indoor air sampling data for residences located downgradient from the Walton and Lonsbury site where groundwater is contaminated by VOCs, particularly trichloroethylene and tetrachloroethylene. Data from nine indoor air sampling rounds conducted over an approximately 10 year period at a total of 13 houses indicated the following:

1. For trichloroethylene, houses I and J had indoor air concentrations that exceeded the ATSDR EMEG/MRL of 0.37 ppb in, but only in their basements. House I has an unfinished basement, while house J has a finished basement. The maximum concentration detected in either house was below the toxicity values from the key studies forming the basis of the EMEG/MRL. Thus, based on the maximum detected concentration and house-specific exposure assumptions, it is unlikely that residents of these two houses experienced past non-cancer health effects from exposure to trichloroethylene in their indoor air.
2. Trichloroethylene results from one round of sampling (January 2001) are considered invalid by MDPH/BEH based on similar trichloroethylene concentrations being detected in all 13 samples (uniformly between 1-2 ppb), including a background house nearly a half mile upgradient from the site. Thus, MDPH/BEH did not include these results in the quantitative evaluation. However, a qualitative evaluation of these results indicates that noncancer and cancer health effects would be unlikely.
3. For tetrachloroethylene, one sample in one house (house L) had a concentration at approximately the EPA RfC (6.1 ppb detected versus the EPA RfC of 5.9 ppb) but did not exceed levels identified in studies as being associated with health effects and tetrachloroethylene was not detected during three out of four sampling rounds at this house. Thus, we would not expect residents of houses near the Walton and Lonsbury site to experience non-cancer health effects from exposure to tetrachloroethylene in their indoor air.
4. Based on this evaluation, we would not expect an unusual cancer risk from the contaminants of concern (trichloroethylene and tetrachloroethylene) or the other VOCs at the levels detected in indoor air.

The two houses most impacted, based on the past indoor air sampling data, were houses I and J, both of which have mitigation systems to reduce or eliminate vapor intrusion into the houses. Available soil gas testing indicated higher levels for trichloroethylene for houses J, M, and N (no soil gas data samples were available from house I).

It seems that soil gas contaminant concentrations were not a good predictor of indoor air contaminant concentrations. For example, the maximum soil gas concentration in any sampling round was for house M (155 ppb), but indoor air quality testing for two floors in this house for the 24-hour period starting at the time of the soil gas sample showed concentrations ranging from 0.07-0.1 ppb.

More recent groundwater testing results have indicated that trichloroethylene may be of most concern near house J. Trichloroethylene was detected in three of 31 off-site groundwater monitoring wells tested during 2011-2012. In two of these wells (MW-15 and RCA-17), the detected trichloroethylene levels indicated possible vapor intrusion. These wells are adjacent to houses J and K. Trichloroethylene was never detected in indoor air samples from house K, most likely because the house has had a radon mitigation system since before 2000.

Houses A through H are not believed to be located in the area of the site where groundwater is contaminated by VOCs. These houses generally had low or no detectable levels of trichloroethylene in both indoor air and soil gas samples. The one exception occurred during the invalid January 2001 sampling round when the trichloroethylene concentrations detected at houses G and F were similar to concentrations detected at the other houses sampled.

Under current conditions, houses I and J (as well as K) have mitigation systems to minimize possible exposure from vapor intrusion. Trichloroethylene and tetrachloroethylene were not detected in a post-installation air sample collected from house J in January 2010. Low levels of trichloroethylene and tetrachloroethylene were detected in air samples collected from house I after the mitigation system was installed in 2008; however, these levels are below levels of concern for non-cancer effects and no unusual cancer risk is expected. However, there has not been any indoor air testing conducted since 2010 in houses I and J to confirm that these systems continue to be effective. Trichloroethylene continues to be detected (based on most recent groundwater sampling data) in offsite monitoring wells in the vicinity of houses K and J, and soil gas testing also indicates the presence of trichloroethylene in soil gas below houses M and N. Thus, it is possible that vapor intrusion could be occurring at the leading edge of the groundwater plume (as depicted in Figure 1), with houses I through N the most likely impacted of those houses that have been tested to date.

MDPH/BEH has determined that the indoor air quality sampling data are limited by the following factors:

1. No residence was tested more than five times over an approximately 10 year period.
2. The round of sampling (in January 2001) that had the highest levels of any round for trichloroethylene may have had quality control issues. Quality control issues are likely because all of the samples had similar levels of trichloroethylene (about 1-2 ppb), including the background house, and no sample quality control data were available for this round of sampling. Because of uncertainty about these data, MDPH/BEH excluded these data from the quantitative evaluation, but discussed them qualitatively.
3. A second round of testing conducted in February 2010 had very high detection limits for trichloroethylene (9.3 ppb), over two orders of magnitude higher than the ATSDR EMEG/MRL for this compound. Thus, results from this sampling round provide little information to help assess possible risks from vapor intrusion.
4. Indoor air concentrations typically vary seasonally and under different use conditions in the house, thereby making it difficult to extrapolate across time. Of the nine sampling rounds conducted at these residences, six were during December-February (two of these were limited by data quality issues or high detection limits), and three were during March or April, or late winter/early spring. Typically, worst-case conditions would be during the winter, when houses are tight and indoor air concentrations can build up if vapor intrusion is occurring.

In addition to evaluating environmental data, we asked the MCBDRP to search the birth defects registry for reports of cardiac malformations near the site. Data from the birth defects registry was reviewed for cardiac malformations because exposure to trichloroethylene during pregnancy is associated with a small increased risk of fetal cardiac defects. For this evaluation, the birth defects surveillance data were queried for all live births to residents of the neighborhood near the Walton and Lonsbury site between 2000 and 2010 for these types of birth defects. This 11-year time period constitutes the period for which the most recent and complete data were available at the initiation of this analysis. No cases of cardiac malformations were reported to the MCBDRP for the neighborhood near the site.

Conclusions

MDPH/BEH concludes that breathing levels of trichloroethylene and tetrachloroethylene detected in houses in the vicinity of the Walton and Lonsbury Superfund Site is not expected to harm people’s health.

From 2000 through 2010, indoor air samples were collected from 13 houses near the Walton and Lonsbury site and analyzed for volatile organic compounds (VOCs), including trichloroethylene and tetrachloroethylene. Trichloroethylene and tetrachloroethylene had been previously identified as primary contaminants of concern in contaminated groundwater beneath the site.

During its review of the indoor air sampling data, MDPH/BEH identified some limitations (e.g., limited number of samples and some data quality concerns) such that additional sampling is desirable and would improve the reliability of the data. Given the available data, MDPH/BEH concludes that the detected levels of trichloroethylene and tetrachloroethylene were below levels known to result in harmful, non-cancer health effects. In addition, MDPH/BEH does not consider the detected trichloroethylene and tetrachloroethylene indoor air levels to present an elevated cancer risk.

MDPH/BEH concludes that breathing other non-site related VOCs detected in the indoor air of houses near the Walton and Lonsbury site is not expected to harm people’s health.

A number of VOCs besides trichloroethylene and tetrachloroethylene were detected in the indoor air samples collected from 13 houses near the Walton and Lonsbury site. These contaminants are most likely not site-related considering that they have not been identified as site contaminants of concern and indoor air typically contains VOCs from many common household products, such as paints, glues, cleaners, and cigarette smoke. In any case, the levels of VOCs detected in the indoor air of houses near the Walton and Lonsbury site were below levels known to result in harmful, non-cancer health effects. In addition, MDPH/BEH does not consider the detected VOC levels to present an elevated cancer risk.

MDPH/BEH cannot conclude whether trichloroethylene or tetrachloroethylene vapors from contaminated groundwater beneath the Walton and Lonsbury site are currently affecting the indoor air of downgradient houses that have not been sampled yet or if conditions have changed in previously sampled houses.

Trichloroethylene continues to be detected (based on sampling data collected in 2012) in off-site groundwater monitoring wells near houses J and K, and tetrachloroethylene is present in soil gas beneath houses M and N. However, no indoor air or soil gas samples have been collected at houses along North Avenue south of houses M and N. Thus, MDPH/BEH cannot determine whether vapor intrusion is currently affecting houses near the leading edge of the trichloroethylene/tetrachloroethylene groundwater plume.

Based on a review of cancer and birth defects surveillance data, no significant elevations or unusual trends in cancer incidence and no cardiac malformations were identified in the area surrounding the Walton and Lonsbury site.

MDPH/BEH completed an evaluation of cancer incidence in the census tract that the Walton and Lonsbury site is located in (6313). MDPH/BEH evaluated the incidence of those cancer types that are most strongly associated with exposure to the primary site contaminants of concern, as well as childhood cancer, for a 25-year period (1983-2007). No unusual trends were noted for any cancer types. Data from the MDPH birth defects registry were reviewed for cardiac malformations because exposure to trichloroethylene during pregnancy is associated with a small increased risk of fetal cardiac defects. The birth defects surveillance data were queried for all live births to residents of the neighborhood near the Walton and Lonsbury site between 2000 and 2010 for these types of birth defects. This 11-year time period constitutes the period for which the most recent and complete data were available at the initiation of this analysis. No cardiac malformations were identified in the vicinity of the site.

Recommendations

Although health effects are not expected based on indoor air sample data collected to date we recognize the limitations of the available data. Thus, because of the limited number of indoor air samples collected from the 13 houses and because no samples have been collected since 2010, MDPH/BEH recommends that EPA re-sample indoor air and soil gas at all 13 houses to confirm that the trichloroethylene and tetrachloroethylene indoor air levels are still below levels that could harm people’s health. At a minimum, indoor air and soil gas samples should be analyzed for cis-1,2-dichloroethylene and vinyl chloride (breakdown products of trichloroethylene), as well as trichloroethylene and tetrachloroethylene. Three of the 13 houses (houses I, J, and K) currently have vapor intrusion mitigation systems to help prevent VOCs (including trichloroethylene and tetrachloroethylene) from entering the air inside the houses. However, no indoor air samples have been collected from these two houses since 2010 to confirm their continued effectiveness. MDPH/BEH recommends that EPA re-sample the indoor air of houses I, J, and K to verify that the vapor intrusion mitigation systems are operating as intended. MDPH/BEH also recommends that homeowners ensure that the systems are properly maintained and serviced, including periodic inspections by a professional to verify systems are in good working order.

Although health effects from exposure to non-site related VOCs in the indoor air of houses near the site are not expected, MDPH/BEH recommends that homeowners consider the following tips to improve their indoor air quality:

* Be aware of household products that contain VOCs. Don’t buy more chemicals than you need.
* Store chemicals (e.g. gasoline, paints, pesticides) in tightly-sealed containers outdoors or in a detached garage/shed, if possible, and away from family living spaces.
* Keep your home properly ventilated by using exhaust fans or opening windows when doing home renovations, do-it-yourself projects, or hobbies that require the use of chemicals.
* Remove unused products from the home. Check with your city/town government about properly disposing of unwanted paints, solvents and other related products (e.g., at hazardous waste collection events).
* Don’t smoke indoors.

MDPH/BEH is aware that the EPA Remedial Program plans to conduct an investigation into the nature and extent of contamination at the Walton and Lonsbury site and recommends that further investigation of the vapor intrusion pathway associated with the site be included in the scope of work. No indoor air or soil gas samples have been collected in houses along North Avenue south of houses M and N. Given that trichloroethylene has been detected in indoor air and soil gas samples collected from houses M and N and in groundwater samples collected from nearby monitoring wells, MDPH/BEH recommends that EPA consider expanding the vapor intrusion investigation to include additional houses located south of the site along North Avenue.

Additionally, MDPH/BEH suggests collecting indoor air samples from the main living level(s) of the houses in addition to the basement. Samples collected from the areas of the home where residents spend the most time (e.g., bedrooms and living room) are important to understanding the concentrations residents are exposed to the majority of the time.

To date, MDPH has taken public health actions to review cancer and birth defects data in the neighborhood near the site. In addition to these actions and the evaluation of indoor air, MDPH/BEH will conduct evaluations of additional potential pathways of exposure (e.g., exposure to contaminated soil) as sampling data is generated by EPA as part of the Remedial Investigation. MDPH/BEH will review and provide health-based input on future EPA sampling plans.

Report Preparation

This health consultation for the Walton and Lonsbury site was prepared by the Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH), under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

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# Figure

**Figure 1**

**Walton and Lonsbury Area Map**

Figure 1. - Walton and Lonsbury Area Map

# Tables

**Table 1. Summary of indoor air sampling rounds and number of samples collected**



**Table 2. Summary of soil gas sampling rounds and number of samples collected**



**Table 3. Maximum concentrations of contaminants detected in indoor air in residences near the Walton and Lonsbury site that exceed comparison values**



**Table 4. House-specific trichloroethylene concentrations (ppb) in indoor air by sampling round**



**Table 5. House-specific tetrachloroethylene concentrations (ppb) in indoor air by sampling round**



**Table 6. House-specific concentrations of trichloroethylene and tetrachloroethylene detected in soil gas (ppb)**



**Table 7. Concentrations of trichloroethylene and tetrachloroethylene (ppb) detected in groundwater on or near the Walton and Lonsbury site during 2011-20121**



**Table 8. Non-cancer exposure concentrations for tetrachloroethylene and trichloroethylene in indoor air**



**Table 9. Estimated cancer risk for exposure to tetrachloroethylene and trichloroethylene in indoor air**

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# Appendices

Appendix A

Cancer Incidence Evaluation Summary

On October 15, 2010, at the request of the City of Attleboro’s Health Director, Mr. James Mooney, MDPH/BEH completed an evaluation of cancer incidence in the census tract where the Walton and Lonsbury site is located (6313). These data are available to CAP through MDPH’s Massachusetts Cancer Registry (MCR). MDPH/BEH evaluated the incidence of those cancer types that are most strongly associated with exposure to the primary site contaminants of concern (hexavalent chromium and trichloroethylene). Those cancers include liver, kidney/renal pelvis, lung & bronchus, and stomach cancer as well as leukemia. Cancer incidence rates were calculated for a 25-year period (1983-2007), 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 the analysis[[2]](#footnote-3). In addition, MDPH/BEH reviewed the incidence of childhood cancer for the same 25-year period. To assess whether any unusual temporal patterns occurred over the 25-year period, cancer incidence was examined for five separate time intervals.

To assess the incidence of cancer in the census tract containing the site, a statistic called the standardized incidence ratio (SIR) was calculated. The SIR is a comparison of the number of diagnoses in the census tract to the number of expected diagnoses based on the statewide rate. Specifically, an SIR is the ratio of the observed number of cancer diagnoses in an area to the expected number of diagnoses multiplied by 100. Age-specific statewide incidence rates were applied to the population distribution of the census tract to calculate the number of expected cancer diagnoses.

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

For the six types of cancer evaluated, some differences were observed between the cancer experience in census tract 6313 versus the state of Massachusetts as a whole. Some cancers occurred less frequently than expected and some slightly more than expected. Importantly, no statistically significant elevations of the cancer types evaluated were noted in census tract 6313. In most instances, the number of observed diagnoses in census tract 6313 was approximately equal to the number of expected diagnoses. In summary, no unusual trends were noted for any cancer types. For childhood cancer, two diagnoses (non-Hodgkin lymphoma and acute myeloid leukemia) were reported to the Massachusetts Cancer Registry (MCR) over the 25-year period compared to approximately five diagnoses that would have been expected.

To determine whether an elevation is occurring among individuals diagnosed with cancer in a community or census tract (CT), cancer incidence data are tabulated by gender according to eighteen age groups to compare the observed number of cancer diagnoses to the number that would be expected based on the statewide cancer rate.

Specifically, an SIR is the ratio of the observed number of cancer diagnoses in an area to the expected number of diagnoses multiplied by 100. Age-specific statewide incidence rates are applied to the population distribution of a community to calculate the number of expected cancer diagnoses. The SIR is a comparison of the number of diagnoses in the specific area (i.e., community or census tract) to the number of expected diagnoses based on the statewide rate. Comparison of SIRs between communities or census tracts is not possible because each of these areas has different population characteristics.

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

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

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

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

To help interpret or measure the stability of an SIR, the statistical significance of an SIR can be assessed by calculating a 95% confidence interval (95% CI) to determine if the observed number of diagnoses is “statistically significantly different” from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). Specifically, a 95% CI is the range of estimated SIR values that has a 95% probability of including the true SIR for the population. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or “normal” population. “Significantly different” means there is less than 5% percent chance that the observed difference (either increase or decrease) in the rate is the result of random fluctuation in the number of observed cancer diagnoses.

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

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

**TABLE 1**

**Cancer Incidence**

**CT 6313 – Attleboro, Massachusetts**

**1983 - 1987**



**TABLE 2**

**Cancer Incidence**

**CT 6313 – Attleboro, Massachusetts**

**1988 - 1992**



**TABLE 3**

**Cancer Incidence**

**CT 6313 – Attleboro, Massachusetts**

**1993 - 1997**



**TABLE 4**

**Cancer Incidence**

**CT 6313 – Attleboro, Massachusetts**

**1998 - 2002**



**TABLE 5**

**Cancer Incidence**

**CT 6313 – Attleboro, Massachusetts**

**2003 - 2007**



Appendix B

Evaluation of VOCs (Other Than TCE and PCE) Detected in Indoor Air

Introduction

Sample results indicated that 41 VOCs were detected in indoor air samples collected from homes near the Walton and Lonsbury site. As mentioned previously, indoor air typically contains VOCs from household sources, as well as from ambient (outdoor) air. It can be difficult to differentiate between VOCs originating from site-related versus non-site related sources. Previous investigations have identified two of the VOCs detected in indoor air at concentrations exceeding comparison values (tetrachloroethylene and trichloroethylene) as contaminants of concern at the Walton and Lonsbury site (EPA 2010; RCA 2001). MDPH/BEH evaluated these two VOCs in the main body of this report. This appendix contains the MDPH/BEH evaluation of the other VOCs detected in indoor air using the methods described earlier in this report.

Indoor air screening evaluation

Results of indoor air sampling from 2000-2010 showed 41 VOCs detected. Of these, 14 VOCs (acrylonitrile; benzene; benzyl chloride; bromodichloromethane, 1,3-butadiene; carbon tetrachloride; chloroform; dibromochloromethane; 1,4-dichlorobenzene; dichlorodifluoromethane; 1,2-dichloroethane; tetrachloroethylene; trichloroethylene; and 1,2,4-trimethylbenzene) were detected at concentrations above health-based comparison values and all but three VOCs (benzene, bromodichloromethane, and dibromochloromethane) were also detected at concentrations above typical indoor air background levels (Table B1). Typical sources of benzene in indoor air include tobacco smoke, exhaust from motor vehicles, household products (e.g., such as some glues, paints, furniture wax, and detergents), and storage of gasoline indoors or in attached garages (ATSDR 2007). The typical source of bromodichloromethane and dibromochloromethane in indoor air is chlorinated drinking water. These two chemicals are byproducts formed when chlorine is added to drinking water to kill disease-causing organisms (ATSDR 1989; EPA 2013c).

In addition to the VOCs discussed above, six VOCs (cis-1,2-dichloroethylene; dichlorotetrafluoroethane; ethanol, 4-ethyl toluene; heptane; and 1,3,5-trimethylbenzene) did not have an ATSDR or EPA comparison value. In these cases we compared the maximum detected concentrations with ATSDR or EPA comparison values for structurally similar compounds or other available guidance levels. In all cases, the maximum concentrations of these six compounds were lower than the background and/or comparison values (Table B2). Thus, these six VOCs were not evaluated further in this health consultation.

VOCs detected at concentrations above both background and comparison values were selected for further evaluation, including acrylonitrile; 1,3-butadiene; carbon tetrachloride; chloroform; 1,4-dichlorobenzene; dichlorodifluoromethane; 1,2-dichloroethane; tetrachloroethylene; trichloroethylene; and 1,2,4-trimethylbenzene (Table B3). Trichloroethylene and tetrachloroethylene were evaluated earlier in this report.

Exposure and Health Effects Evaluation

To evaluate for potential health effects, exposure concentrations were compared to noncancer comparison values and cancer risk estimates were calculated for the inhalation pathway for VOCs that exceeded background and comparison values (Tables B4 and B5).

Exposure concentrations for 1,4-dichlorobenzene and 1,2,4-trimethylbenzene exceeded health comparison values for non-cancer health effects. Additionally, calculated cancer risks for children for 1,2-dichloroethane and for both children and adults for chloroform slightly exceeded EPA’s typical risk range of 1 in 10,000. Thus, these four VOCs were evaluated further and are discussed in more detail below.

1,4-Dichlorobenzene

1,4-Dichlorobenzene was detected in three samples collected from two houses (Houses D and G) at concentrations exceeding typical indoor air background concentrations (<0.5-0.63 ppb) and the ATSDR Chronic EMEG/MRL (10 ppb). 1,4-Dichlorobenzene was detected in one out of three samples collected from house D at a concentration of 12 ppb (basement 12/15/10) and in two out of five samples collected from house G at 14.1 ppb (basement 1/18/01) and 17.7 ppb (first floor 1/18/01). MDPH selected the maximum detected concentration of 17.7 ppb as the exposure concentration to represent a worst-case scenario. The maximum concentration exceeded the ATSDR EMEG/MRL of 10 ppb (60 µg/m3) but not the EPA RfC of 133 ppb (800 µg/m3). The ATSDR EMEG/MRL was based on an animal study and was calculated from a benchmark dose of 9,510 ppb for nasal lesions in female rats with an uncertainly factor of 3 applied for extrapolation from animals to humans and a uncertainty factor of 10 applied for human variability (ATSDR 2006). The maximum detected concentration is two orders of magnitude lower than the benchmark dose that the ATSDR EMEG/MRL is based on; thus, non-cancer health effects are not expected (Table B4). The U.S. National Toxicology Program (NTP) classifies 1,4-dichlorobenzene as reasonably anticipated to be a carcinogen and the International Agency for Research on Cancer (IARC) classifies it as possibly carcinogenic to humans based on limited human evidence and less than sufficient evidence in animals (ATSDR 2013). Currently there is no EPA inhalation unit risk factor available for 1,4-dichlorobenzene, thus a quantitative evaluation of cancer risk could not be completed.

1,2,4-Trimethylbenzene

1,2,4-Trimethylbenzene was detected in four samples collected from three houses (Houses B, G, and M) at concentrations exceeding typical indoor air background concentrations (1.4-2.0 ppb) and the EPA RSL (1.5 ppb). RSLs are chemical-specific concentrations for individual contaminants in air, drinking water, and soil that EPA uses to determine whether further investigation or site cleanup is warranted. This VOC was detected in one out of two samples collected from house B at a concentration of 2.2 ppb, in one out of five samples collected from house G at a concentration of 162.7 ppb, and in two out of five samples collected from house M at 2.9 ppb and 5.8 ppb. MDPH selected the maximum detected concentration of 162.7 ppb as the exposure concentration to represent a worst-case scenario. The maximum detected concentration exceeded the EPA draft RfC of 20 ppb (98 µg/m3). An RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure of a chemical to the human population (including sensitive subpopulations) that is likely to be without risk of deleterious noncancer effects during a lifetime. The draft EPA RfC is based on an animal study and was calculated from a point of departure of 15,800 µg/m3 (after calculating the human equivalent dose using a physiologically-based pharmacokinetic model) for decreased pain sensitivity with a composite uncertainty factor of 1,000. The RfC was then rounded for a draft RfC of 20 µg/m3. The worst-case scenario exposure concentration is two orders of magnitude lower than the point of departure for the RfC, indicating that non-cancer effects are unlikely (Table B4). The draft EPA Toxicological Review of Trimethylbenzenes concludes that there is insufficient evidence to evaluate the carcinogenicity of trimethylbenzenes (EPA 2012b).

Chloroform

Chloroform was detected in two samples collected from house F at concentrations exceeding the background value of 1.27 ppb and the ATSDR CREG of 0.0089 ppb. Chloroform was detected in three out of the four samples collected from house F. Samples collected on January 18, 2001 from the basement (2.17 ppb) and first floor (4.3 ppb) exceeded background and comparison values and a sample collected from the basement on December 15, 2010 (0.06 ppb) was below typical background concentrations for indoor air. Chloroform is a by-product produced when chlorine is added to drinking water to make it safe for consumption and is commonly detected in the indoor air of homes served by public water supplies (EPA 2013c). Exposure concentrations calculated using the maximum detected value of 4.3 ppb did not exceed the ATSDR Chronic EMEG/MRL, thus non-cancer effects are not expected (Table B4). It is more appropriate to calculate cancer risk based on average exposures; however, there were too few chloroform samples available to calculate a reliable average concentration for house F. Thus, the maximum and minimum concentrations detected were used to calculate a range of cancer risks. Calculated cancer risks based on the maximum concentration (4.3 ppb) were 2 in 10,000 (2 x 10-4) for adults and 4 in 10,000 (4 x 10-4) for children. Based on the minimum concentration (0.06 ppb), calculated cancer risks were 3 in one million (3 x 10-6) for adults and 6 in one million (6 x 10-6) for children (Table B5). Calculated cancer risk based on the two sampling rounds differed by about two orders of magnitude. Typical exposure concentrations, and thus cancer risks, likely fall somewhere between these values and thus, no unusual cancer risk is expected.

1,2-Dichloroethane

1,2-Dichloroethane was detected in indoor air samples collected from four houses (houses G, H, J, and N) at concentrations exceeding the typical indoor air background concentration of 0.099 ppb and the ATSDR CREG of 0.0095 ppb, but not the ATSDR Chronic EMEG/MRL of 600 ppb. This VOC was detected in two of the five indoor air samples collected from house G (0.76 ppb in the basement and 1.8 ppb on the first floor collected 12/15/10), in one of three samples collected from house H (0.83 ppb in basement 12/5/10), in four of eight samples collected from house J (0.18 ppb in basement 12/2/2008, 0.095 ppb in basement 3/3/09, 0.28 ppb on first floor 3/3/09, and 0.36 ppb on second floor 3/3/09), three of which were above the background concentration, and in one out of six samples collected from house N (0.28 ppb on first floor on 12/15/10 and not detected in the basement sample from the same day). A pattern of higher concentrations of 1,2-dichloroethane on the higher levels of homes (e.g., first floor versus basement) may be an indication that the chemical is not originating from a sub-surface source. The maximum detected value of 1.8 ppb from house G did not exceed the ATSDR Chronic EMEG/MRL, thus non-cancer effects are not expected (Table B4). It is more appropriate to calculate cancer risk based on average exposures; however, there were too few 1,2-dichloroethane samples available to calculate reliable average concentrations for most of the houses. Thus, a range of cancer risks for each of the four houses was calculated based on available data. Calculated cancer risks based on the maximum concentration detected in house G on 12/15/10 were 8 in 100,000 for adults (8 x 10-5) and 1.6 in 10,000 for children (1.6 x 10-4). This substance was not detected in house G during other sampling rounds, thus, a concentration equal to the laboratory detection limit (0.45 ppb) was used as the minimum concentration to calculate cancer risk resulting in estimated cancer risks of 2 in 100,000 (2 x 10-5) for adults and 4 in 100,000 (4 x 10-5) for children. Given that 1,2-dichloroethane was only detected in one out of the three rounds of sampling at house G, it unlikely that the long-term average concentration is elevated and unusual cancer risk is unlikely. Calculated cancer risks for house H, the house with the next highest detection of 0.83 ppb, were 4 in 100,000 (4 x 10-5) for adults and 7 in 100,000 (7 x 10-5) for children. Because the cancer risks for the next highest detection did not exceed a risk of 1 in 10,000, unusual cancer risks are not anticipated in houses H, J, and N (Table B5).

**Table B1. Maximum concentrations of contaminants detected in indoor air in residences near the Walton and Lonsbury site that exceed comparison values1**



**Table B2. Maximum concentrations of contaminants detected in indoor air without ATSDR or EPA comparison values1**



**Table B3. House-specific concentrations of contaminants detected in indoor air that exceed background AND comparison values**



**Table B4. Non-cancer exposure concentrations for VOCs other than tetrachloroethylene and trichloroethylene**



**Table B5. Estimated cancer risk for exposure to VOCs other than tetrachloroethylene and trichloroethylene**



Appendix C

Sample Exposure Calculations

Adult Residential Cancer Exposure Estimate Equation

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Where:

EC (μg/m3) = exposure concentration;

CA (μg/m3) = contaminant concentration in air;

ET (hours/day) = exposure time;

EF (days/year) = exposure frequency;

ED (years) = exposure duration; and

AT (78 year lifetime x 365 days/year x 24 hours/day) = averaging time

Cancer Risk Estimate Equation

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Where:

EC (μg/m3) = exposure concentration;

IUR (μg/m3) -1 = Inhalation Unit Risk (EPA’s chemical-specific cancer risk factor)

Adult Residential Cancer Exposure Estimate Sample Calculation for Trichloroethylene (Maximum Concentration)

Exposure Concentration = 

Adult Residential Cancer Risk Estimate Sample Calculation for Trichloroethylene (Maximum Concentration)

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Child Residential Cancer Exposure Estimate Equation

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Where:

EC (μg/m3) = exposure concentration;

Age Dependent Adjustment Factors

Birth to <2 years = 10

2 to <16 years = 3

16 to <18 years = 1

CA (μg/m3) = contaminant concentration in air;

ET (hours/day) = exposure time;

EF (days/year) = exposure frequency;

ED (years) = exposure duration; and

AT (78 year lifetime x 365 days/year x 24 hours/day) = averaging time

ADAFs = Age Dependent Adjustment Factors

Child Residential Cancer Exposure Estimate Sample Calculation for Trichloroethylene (Maximum Concentration)

EC (birth to <2yrs) =

EC (2 to <16yrs) = 

EC (16 to <18yrs) = 

Total Child Exposure Concentration = 

Child Residential Cancer Risk Estimate Sample Calculation for Trichloroethylene (Maximum Concentration)

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1. The national upper bound percentile for length of time at one residence (95th percentile value) of 33 years was used for the duration of exposure (US EPA 2011d). [↑](#footnote-ref-2)
2. The data summarized in this report are drawn from data entered into the MCR before October 2010. 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. [↑](#footnote-ref-3)