



Public Health Assessment for

**LEEDS METAL NPL SITE
LEEDS, MAINE
EPA FACILITY ID: MEN00103584
APRIL 9, 2013**

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE**

Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i) (6) (H) for their information and review. The revised document was released for a 60-day public comment period. Subsequent to the public comment period, ATSDR will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

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PUBLIC HEALTH ASSESSMENT

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Agency for Toxic Substances and Disease Registry

Summary

Introduction

The Agency for Toxic Substances and Disease Registry’s (ATSDR) top priority is to ensure that the people living in Leeds, Maine have the best information possible to safeguard their health.

The Leeds Metal Site is a former automobile shredder and scrap metal recovery facility. Material remaining after metal recovery (called “auto fluff”) makes up 4 large piles on site, and elevated levels of polychlorinated biphenyls (PCBs), metals, and other contaminants have been detected on the site. In addition, volatile organic compounds (VOCs) including trichloroethylene (TCE) have been detected in private wells near the site. The Maine Department of Environmental Protection (ME DEP) has been monitoring private wells in the area, and the U.S. Environmental Protection Agency (EPA) has added the site to the National Priorities List (NPL, or “Superfund”). ATSDR is required to conduct public health activities on all sites proposed for the NPL.

The purpose of this Public Health Assessment (PHA) is to determine whether the community is or was harmed by exposure to chemicals from the site and what public health actions need to be taken to reduce harmful exposures.

Conclusions

ATSDR reached three important conclusions in the PHA:

Conclusion 1

People using private wells near the site are currently at no risk of harmful effects from trichloroethylene (TCE) in water. Private well water may contain harmful levels of arsenic, which may not be directly related to the site.

Basis for Conclusion

All private wells have TCE too low to cause harmful effects or have filters to remove TCE. Only one well has TCE above EPA’s Maximum Contaminant Level (MCL). This well is filtered to remove TCE. Past exposure to TCE in this well through reported use of the well was unlikely to result in harmful cancer or noncancer effects. Several private wells contain arsenic at levels that could increase the risk of developing skin lesions and could increase the lifetime risk of cancer, especially for people who drink higher-than-average amounts of the water. This is an issue in private wells in many parts of Maine.

Next Steps

- ATSDR recommends ME DEP continue sampling all private wells to ensure TCE-removing filters are operating properly and to evaluate whether filters may be needed for any additional wells.
- Homeowners whose private wells contain arsenic above drinking water standards should consider installing a reverse osmosis treatment system or switching to another source for drinking water. Carbon filters will not remove arsenic from water.

- Homeowners are encouraged to consult <http://wellwater.maine.gov> for more information about treatment systems for private wells.
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Conclusion 2

While trespassing on the site is illegal and discouraged, exposure of trespassers to contaminants in on-site soil is unlikely to result in harmful health effects. However, the site contains physical hazards that could cause injury.

Basis for Conclusion

Estimated exposures to contaminants in soil during occasional trespassing, digging on site, or ATV riding are too low to result in harmful health effects. However, many physical hazards exist on the site, including steep and possibly unstable slopes, sharp pieces of metal and rebar that may cause injury, and at least one concrete pit containing water.

Next Steps

- Property owners and/or EPA should install effective fencing, gates, and warning signs to keep trespassers from entering the site.
-

Conclusion 3

Further investigation of the nature and extent of contamination at the site is needed.

Basis for Conclusion

This PHA is based on limited data available through EPA’s NPL listing process and reported current uses of the site. Changing use of the site would require a re-evaluation of potential health effects of pathways. Several potential exposure pathways, including surface water, sediment, and vapor intrusion could not be fully evaluated at this time due to a lack of data.

Next Steps

- EPA should continue investigating the nature and extent of contamination associated with the site.
- The site should not be used for other purposes without additional investigation and public health evaluation.
- ATSDR will evaluate, upon request, additional data that become available on the site.

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List of Abbreviations

ADAF	Age Dependent Adjustment Factor
ATSDR	Agency for Toxic Substances and Disease Registry
ATV	All-terrain vehicle
CREG	Cancer Risk Evaluation Guide
CV	Comparison Value
EMEG	Environmental Media Evaluation Guide
EPA	U.S. Environmental Protection Agency
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
IUR	Inhalation unit risk
MCL	Maximum Contaminant Level
ME DEP	Maine Department of Environmental Protection
mg/kg	Milligram per kilogram
mg/L	Milligram per liter
mg/m ³	Milligram per cubic meter
MRL	Minimal Risk Level
NPL	National Priorities List
NTP	National Toxicology Program
PCB	Polychlorinated Biphenyl
PCE	Tetrachloroethylene (or Perchloroethylene)
PHA	Public Health Assessment
RfC	Reference Concentration
RfD	Reference Dose
RMEG	Reference Media Evaluation Guide
RSL	Regional Screening Level
TCE	Trichloroethylene (or Trichloroethene)
µg/L	Microgram per liter
µg/m ³	Microgram per cubic meter
VOC	Volatile Organic Compound

Purpose and Health Issues

The Leeds Metal Site was proposed for the National Priorities List (NPL) on September 16, 2011; the listing was finalized on September 18, 2012. The Agency for Toxic Substances and Disease Registry (ATSDR) is required by Congress to conduct public health activities on all sites proposed for the NPL. This public health assessment evaluates whether and in what ways the Leeds Metal Site could affect public health. ATSDR reviewed available environmental data, ways in which people could come in contact with contaminants from the site, and community health concerns to determine whether adverse health effects are possible as a result of contamination on and near the site.

Public Comment

ATSDR released a draft of this PHA for public comment on November 28, 2012. The PHA was available for public review and comment at the Leeds Town Office in Leeds, Maine. The document was also available for viewing or downloading from the ATSDR web site. The public comment period was open from November 28, 2012 through January 28, 2013.

The public comment period was announced to local media outlets. ATSDR discussed the findings of the PHA with community members at an informal open house and presented the findings at a Board of Selectmen meeting on December 12, 2012 in Leeds, Maine. Copies of the PHA and fact sheets summarizing the findings were also provided to the community during the open house.

No public comments were received on the PHA.

Background

Site Description, History, and Features

This background information is from site documents available through EPA's NPL listing documentation [1]. The Leeds Metal Site consists of a 36-acre property along Blue Rock Road in Leeds, Androscoggin County, Maine. The current owner is Pan Am Railways. Before 1969, the property was mined for sand and gravel. From about 1969 until 1984, various automobile shredding and scrap metal recovery operations took place on the site. The automobile shredding operation took junked automobiles, mechanically fragmented them into small pieces, and used magnets to recover the ferrous material for recycling. The remaining nonferrous material (from seats, plastic, glass, etc.), known as "auto fluff", was discarded on site. Reportedly, fluids such as gasoline, brake fluid, and antifreeze from the cars were dumped on the ground. Parts of the site were also reportedly used for concrete manufacture and disposal, storage of transformers and drums, and dumping of various items. Elevated levels of volatile organic compounds (VOCs), polychlorinated biphenyls (PCBs), and metals have been documented on the site. In addition, VOCs, primarily tetrachloroethylene (PCE) and trichloroethylene (TCE), have been detected in site groundwater and in private wells near the site.

Site investigational history is detailed in the EPA site listing documentation and will only be briefly summarized here [1–3]. Beginning in 1983, the Maine Department of Environmental Protection (ME DEP) responded to complaints regarding leaking transformers and abandoned

drums on the site and began working with property owners to characterize the site and take needed response actions. Initial Site Discovery and groundwater monitoring investigations published in the late 1990s indicated probable releases from the site [4,5]. Beginning in 2000, ME DEP initiated a private well monitoring program for residences potentially impacted by site contamination [6]. In the 2000-2005 time frame, contractors working on behalf of the property owners conducted additional site studies, including further groundwater investigation, debris pile evaluation, and a targeted environmental investigation to determine conditions at a specific area of interest on the site [7–10].

In 2008, ME DEP requested EPA to become involved to improve cooperation of the property owner and ensure the site receives appropriate and timely response [11]. EPA completed a Preliminary Assessment/ Site Investigation in 2009 and a Site Investigation in 2010 [2,3]. In 2010, the state of Maine revised and lowered its maximum exposure guideline (MEG) for PCE in drinking water from 7 to 0.6 micrograms per liter ($\mu\text{g/L}$) [12,13]. With this change, five of the private wells being monitored exceeded the action level of one-half the MEG. In early 2011, the property owner installed, at ME DEP's direction, carbon filters on these five private wells [14]. The site was proposed for listing to the NPL in September 2011 [1].

Features of the site are shown in Figure 1 [1]. The main source areas on the site are on the eastern and southern portions of the site and include the former operations area and four large auto fluff/debris piles, estimated to have a total volume of 40,000 cubic yards. The northern and western portions of the property are wooded and contain two lagoon areas and an area of concrete disposal. The property is bordered to the south by a wooded area, a wetland, and residential properties. Maine Route 106 and residential properties border the west side of the site, and a former concrete manufacturing and gravel storage area border the site to the north. The east of the property is bordered by a railroad track and wooded area.

Demographics

Figure 2 shows demographic information for a 1-mile radius around the site. The total population living within a one-mile radius of the site is 568. The population includes the following potentially sensitive groups: about 18% women of childbearing age; about 11% children aged 6 and younger; and about 12% adults aged 65 and older.

Geology, Groundwater Flow, and Natural Resource Use

The site is underlain by a sand and gravel overburden, containing a moderate-to-good producing aquifer, above bedrock present about 100-150 feet below the ground surface [1]. The bedrock and overburden aquifers are connected – that is, there is no confining layer preventing contaminants from moving between the two groundwater aquifers. The flow of groundwater beneath the site has been subject to some debate and is not completely characterized [1]. The available groundwater information from the site does not exceed 47 feet in depth, and contaminants such as PCE and TCE measured at the site are heavier than water and will tend to sink. State officials noted that deeper ground water flow likely follows the regional surface water drainage pattern towards the Dead River located southwest of the site [1].

Private wells in the area draw water from various depths. Most of the wells near the site obtain water from the bedrock aquifer.

Figure 1. Features of Leeds Metal Site, Leeds, Maine (Source: HRS Package [1])

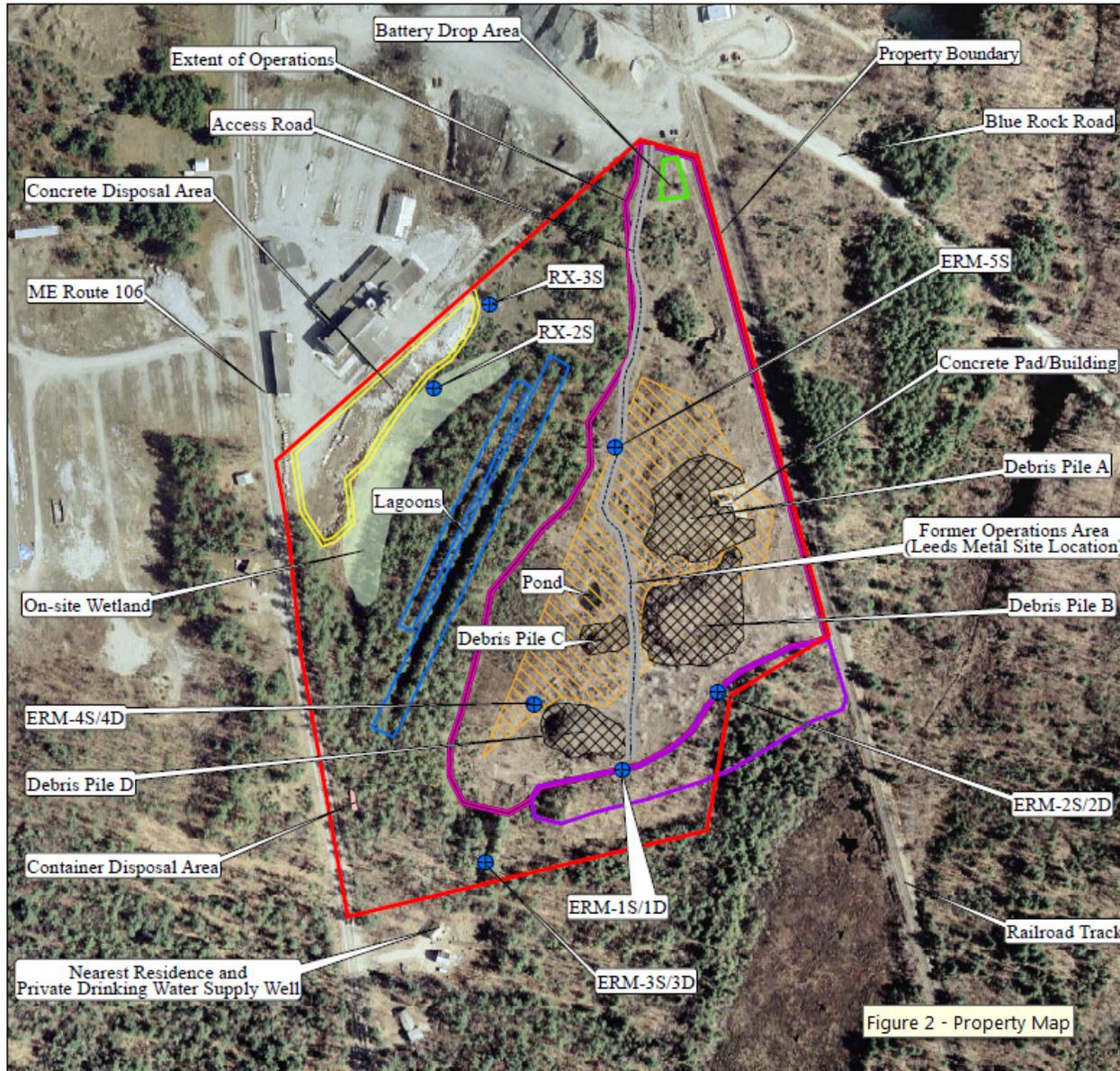


Figure 2 - Property Map

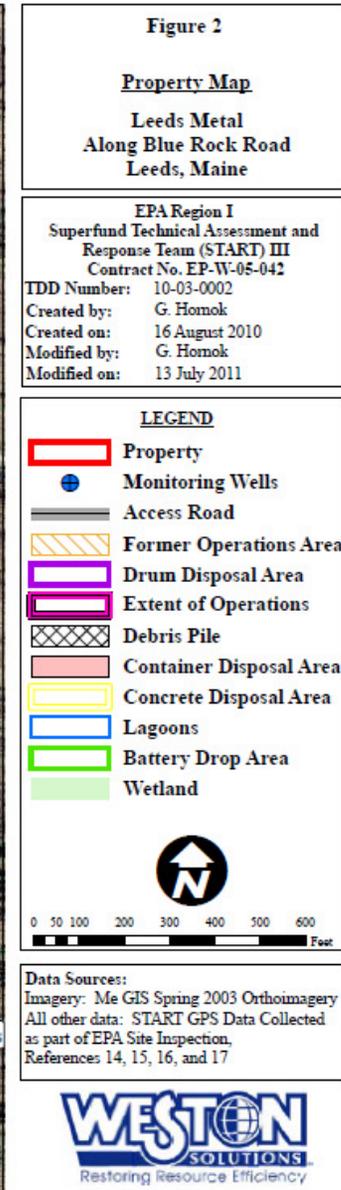
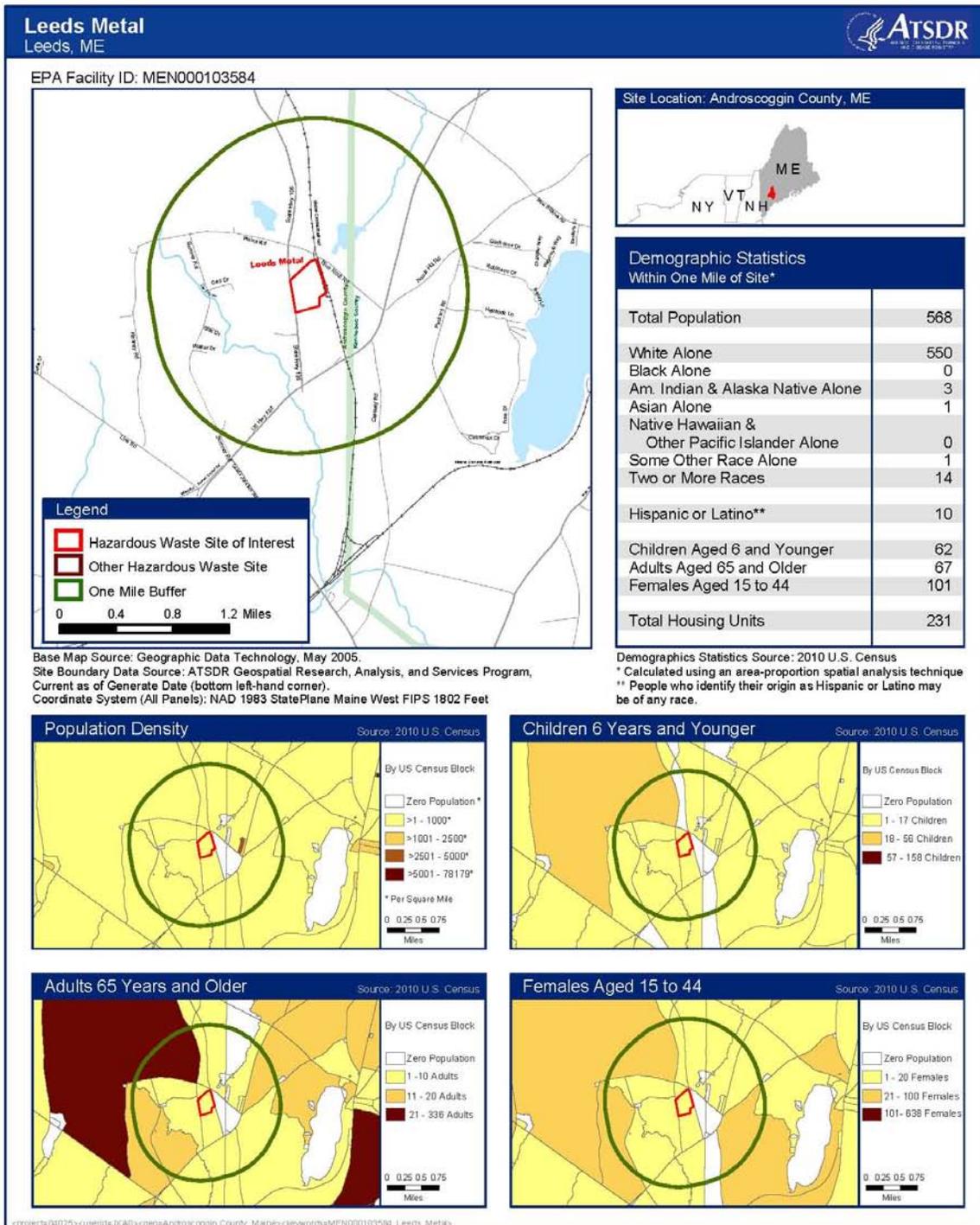


Figure 2. Site Location and Demographic Information for the Leads Metal Site, Leeds, Maine



As part of its site investigation, EPA and its contractors conducted a site visit and interviewed local residents [2]. Evidence of hunting and recreational vehicle use was observed on site, and residents reported that hikers and hunters use the site frequently.

Discussion

Data Used

The major source of data evaluated in this report is EPA's NPL listing documentation; references listed in this documentation were provided electronically by EPA Region 1 [1]. Additional well sampling data were provided by ME DEP [15]. Data evaluated and used directly in this PHA included:

- The August 2011 Final Site Inspection Report detailing results of soil, groundwater, surface water, sediment, and private well sampling at the site by EPA [2];
- A spreadsheet containing results of private well sampling for dates ranging from 2000 to 2011 by ME DEP [15]; and
- The February 2010 Preliminary Assessment / Site Inspection Report detailing results of soil, groundwater, surface water, sediment, and private well sampling at the site by EPA [3].

While not used directly in this PHA, ATSDR examined data from additional studies [4–10]. The data were examined to ensure that they were consistent with the above studies. The three data sources listed above were considered adequate to represent site conditions and potential exposures.

ATSDR visited the site¹ to better understand the physical setting of the site and its relationship to the people living and working nearby. The visit included a walk-through of the entire site, including access paths, the former shredding area, auto fluff piles, and wooded areas. During the site visit, the following observations were made:

- The site was accessible to the public. Minimal fencing was present. Parts of the site were wooded, and former processing areas had young trees and vegetation starting to take over.
- A dirt road/path led into the site, past a “battery drop” area where high levels of lead were reportedly detected, and to the main former processing area.
- The former processing area was paved, but the concrete was cracked and



Figure 3. Former main processing area

¹ATSDR Staff (Jill Dyken, Tarah Somers, Gary Perlman, Donna Chaney, and Debra Joseph) visited the site on November 30, 2011 accompanied by the EPA on-scene coordinator (John McKeowan), remedial project manager (Anni Loughlin) and other representatives of EPA, Maine DEP, and PanAm Railways.

deteriorating. Structures formerly housing the shredder and other equipment were present. Debris such as old tires and concrete pieces covered the area, and large pieces of machinery had been abandoned in this area (see Figure 3).

- Behind the former processing area, 4 piles of auto fluff were located. The material in the piles was fine and appeared soil-like from a distance; but on close inspection could be seen to consist of small particles of cloth, plastic, and metal. The piles had a springy feel when walked on. Small vegetation and some trees were growing in some of the fluff piles.
- The largest fluff pile (Pile D) provided a good view of the surrounding area and was approximately 45 feet high (see Figure 4).
- Although not visible from the site, the nearest residences were reported to be about 250 yards southwest of pile D.
- Physical hazards were apparent throughout the site. Large tangles of metal rebar with pointed ends were lying on the ground, and metal rods were protruding from auto fluff piles (see Figure 5). The auto fluff piles themselves (especially pile D) had steep slopes that were potentially unstable. Concrete fragments, rusty crushed drums, and large piles of tires presented potential hazards to people who may be on site. Near the former processing area, a concrete pit with water in it posed a potential injury or drowning hazard.
- During the site visit, the group observed two men in a truck who appeared to be collecting scrap metal from the site. A representative of the property owner advised the men to leave the area.
- Other evidence of trespassing included beverage cans and bottles and dumping observed in a few areas.
- The group also toured a portion of the site that was not used for auto shredding operations. This section was wooded and did not show evidence that it was used for dumping. The area contained two long trenches, filled with water during the site visit, which may have been dug during the site's past use as a gravel operation.



Figure 4. Pile D



Figure 5. Close-up of auto fluff with physical hazard

Pathway Analysis

ATSDR determines whether people may have come into contact with chemicals from a site by examining *exposure pathways*. Exposure pathways consist of five elements which must all be present (in the past, now, or in the future) for exposure to occur: a contamination *source*; *transport* of the contaminant through an environmental medium like air, soil, or water; an *exposure point* where people can come in contact with the contaminant; an *exposure route* whereby the contaminant can be taken into the body; and an *exposed population* of people actually coming in contact with site contaminants.

Completed exposure pathways are those for which all five pathway elements are evident, and indicate that exposure to a contaminant has occurred in the past, is now occurring, or will occur in the future. If one or more elements is missing or has been stopped (e.g., by removal of the exposed population from coming in contact with the contaminant, the pathway is *incomplete*, and exposure cannot occur. *Potential* exposure pathways are those for which exposure seems possible, but one or more of the elements is not clearly defined. Potential pathways indicate that exposure to a contaminant could have occurred in the past, could be occurring now, or could occur in the future.

The identification of an exposure pathway does not necessarily mean that health effects will occur. Even if exposure has occurred, is now occurring, or is likely to occur in the future, human health effects might not result. Further evaluation of the exposure is necessary to determine the likelihood for health effects from exposure.

At the Leeds Metal Site, the contamination source was the automobiles and other waste processed at the site during operations. Today, the remaining auto fluff piles can be considered a contamination source, as well. The following two pathways are considered of greatest likelihood for harmful exposures at the Leeds Metal Site:

- Private Well Water – site contaminants entered the groundwater beneath the site and moved to where private wells extract water. People drink the water and use it for other household purposes. This pathway is considered complete for past and current exposures, unless the well water is filtered or treated to effectively remove the contaminants.
- On-Site Soils – contaminants present in soils or auto fluff piles on the site may be contacted by people trespassing on the site, resulting in exposure. Because trespassing has been reported and observed on site, this pathway is considered complete for both past and current time periods.

These pathways will be evaluated for potential health effects in this PHA. Although private wells draw from groundwater, groundwater data from the site itself are not evaluated because this water is not used directly by anyone in the community. Other potential exposure pathways, including exposure to surface water and sediment on the site, are not evaluated in this document because there is little sampling data available to evaluate these pathways at this time. The major activities identified at the site (trespassing, digging for scrap metal, and all-terrain vehicle (ATV) riding) may bring people in contact with soil and are the evaluated further in this document using the available on-site soil data.

Evaluation Process

The typical process by which ATSDR evaluates the potential for adverse health effects to result from exposure to site contaminants will be described briefly in this section.

- When presented with results of comprehensive environmental sampling for chemicals, ATSDR reduces the number of contaminants that need to be evaluated by screening the results for each chemical against *comparison values* (CVs)—concentrations of chemicals in the environment (air, water, or soil) below which no adverse human health effects would be expected to occur. If a contaminant is present at a level higher than the corresponding CV, it does not mean that adverse health effects will occur; the contaminant is merely retained for the next step of evaluation.
- The next step of evaluation focuses on identifying which chemicals and exposure situations could be a health hazard. For exposures occurring by inhalation, we compare the air concentration of the contaminant directly with health guideline air concentrations such as an ATSDR Minimal Risk Level (MRL), if available, or an EPA Reference Concentration (RfC). For other pathways, we calculate *exposure doses*—estimated amounts of a contaminant that people come in contact with and get into their bodies, on an equivalent body weight basis—under specified exposure situations, typically starting with “worst case” type assumptions to obtain the highest dose that could be expected. Each calculated exposure dose is compared against the corresponding health guideline, typically an ATSDR MRL or EPA Reference Dose (RfD), for that chemical. Health guidelines are considered safe doses; that is, if the concentration or calculated dose is at or below the health guideline, no adverse health effects would be expected.
- If the “worst case” exposure dose for a chemical is greater than the health guideline, then the exposure dose may be refined to more closely reflect actual exposures that occurred or are occurring at the site. The exposure dose is then compared to known health effect levels (for both cancer and non-cancer effects) identified in ATSDR’s toxicological profiles or EPA’s Integrated Risk Information System (IRIS). These comparisons are the basis for stating whether or not the exposure presents a health hazard.
- The estimated risk of developing cancer from exposure to carcinogens is calculated by multiplying the site-specific estimated exposure dose by an appropriate cancer slope factor or inhalation unit risk (please see details in Appendix A). The result is an estimate of the increase in lifetime risk of developing cancer from exposure to the contaminant and is compared to EPA’s acceptable risk range for Superfund of 1 in 1,000,000 to 1 in 10,000. The actual increased risk of cancer may be lower, perhaps by several orders of magnitude, than the calculated number due to several factors in the cancer risk assessment process detailed in Appendix A.

Evaluation of Leeds Metal Site Exposure Pathways

Private Well Water Pathway

Several private wells are located near the site. Since the year 2000, ME DEP has sampled wells that may have been affected by contaminants from the site. EPA also conducted sampling of private wells in 2009 and 2010. Table 1 summarizes contaminants detected in private wells near the site. Of several VOCs detected, only TCE was detected in any well at a level exceeding its corresponding CV. In addition, arsenic was detected in several wells at concentrations exceeding

its CV. Arsenic occurs naturally in several areas of Maine and may not be directly related to the Leeds Metal Site. However, we evaluate any contaminants that exceed CVs. Exposure to TCE and arsenic in private well water will be evaluated further in the ensuing sections.

Evaluation of Exposure to TCE in Private Well Water

People using private wells near the site could be exposed to TCE. This is true both in the past, before any wells were fitted with filters to remove VOCs, and currently, for wells that have not been fitted with filters. Exposure to TCE could occur through *ingestion* (drinking the water), *inhalation* (breathing in TCE evaporating from the water during showering, bathing, or other household use), or *dermal exposure* (getting contaminated water on the skin).

Table 2 shows the mean TCE concentrations (averaged per well over time) measured in private wells near the site. The table presents the wells in order of decreasing mean TCE concentration, not necessarily related to geographic proximity to the site. Four of the private wells have had carbon filters installed by ME DEP to remove VOCs¹; the third column of Table 2 indicates that sampling of these wells after filtration showed that the filters were effective at removing TCE present in the well water.

Figure 6 graphically depicts the changing TCE concentrations measured in these wells. TCE was detected at low levels in some private wells beginning in 2000, when the wells were first sampled. Most wells, at the last sampling date available, still had relatively low TCE concentrations, below the maximum contaminant level (MCL) of 5 µg/L. In 2010, one well was identified with a TCE concentration higher than the MCL (older sample results for this well are not available). This well and four other wells had filters installed in 2011 to remove contaminants [14]¹. As stated previously, the filters remove TCE and other VOCs to below detection limits [15].

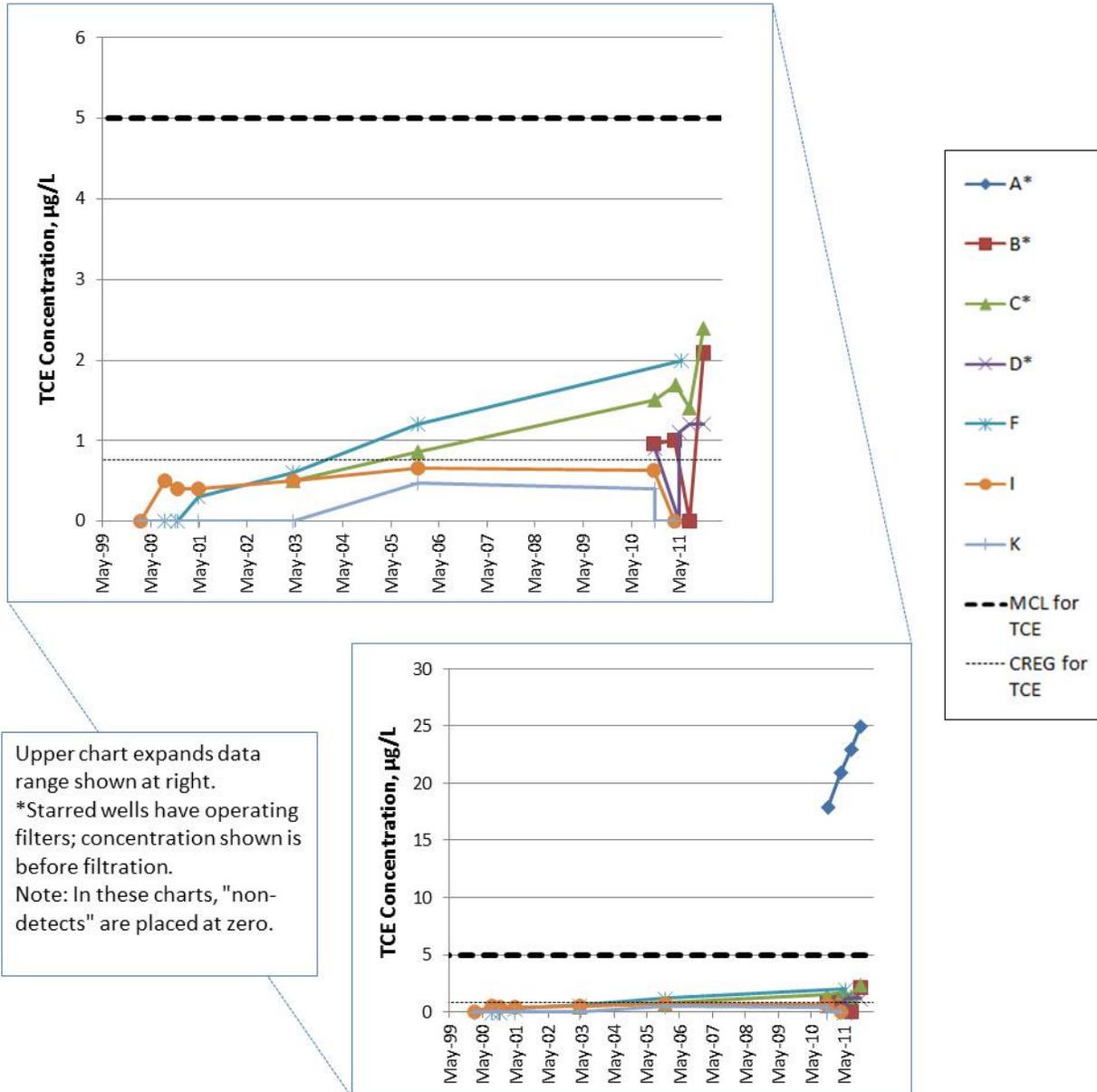
¹ Five wells were fitted with carbon filters because they exceeded the action level for PCE. One well did not have any detection of TCE, and therefore does not appear in Table 2 or Figure 6.

Table 2. Mean TCE concentration for Fourteen Private Wells With TCE Detected – Leeds Metal Site

Well ID*	Mean Detected TCE Concentration in µg/L**	TCE Concentration After Filtration, if Present
A	22.0	Not Detected [†]
B	1.4	Not Detected
C	1.4	Not Detected
D	1.1	Not Detected
E	1.0	No Filter
F	1.0	No Filter
G	0.7	No Filter
H	0.5	No Filter
I	0.5	No Filter
J	0.4	No Filter
K	0.4	No Filter
L	0.4	No Filter
M	0.2	No Filter
N	0.1	No Filter

* Wells listed in order of decreasing TCE concentration; no relation to location.
 ** Mean calculated from well-specific sample results (1-6 detections per well, before filters, if present) collected in 2000-2011.
 † Typical detection limit 1 µg/L; Results provided by ME DEP [15].
 µg/L = micrograms per liter

Figure 6. TCE Concentration Over Time in Private Wells* Near Leeds Metal Site, Leeds, Maine



With the exception of the one private well with a high TCE concentration, none of the private wells tested have ever shown TCE above the MCL. Several wells have exceeded ATSDR's cancer comparison value; however, this value is based on extremely conservative assumptions. For the wells that had TCE below the drinking water standard, current or past exposures are unlikely to result in any adverse health effects in children or adults.

To evaluate the one private well where TCE concentration exceeded the MCL, ATSDR looked at information on when the well was installed and its use. The well was installed in 2006, and the owner reported to EPA that water from the well was used for all household purposes except drinking [16]. In 2011, a carbon filter was installed on the well to remove VOCs and follow-up sampling confirmed that the filter was effective at removing TCE to nondetect levels. Therefore, exposure to TCE from the well has been stopped since 2011. No health effects from TCE exposure are currently likely in children or adults, even if they use the well water for drinking.

In the past (from 2006-2011), residents served by this private well could have been exposed to TCE while using the well water for showering and other household uses. Showering is considered the major contributor to overall exposure because hot water will result in more TCE evaporating from the water, and showering is typically done in a small, enclosed space where TCE concentrations might build up. Exposure could result from breathing in TCE that evaporates from contaminated water or by absorbing TCE through the skin.

Although Figure 6 seems to suggest that TCE concentrations in the well were lower in the past, no historical data exist to show when the TCE entered the well or what the TCE concentrations were in the past. Therefore, ATSDR evaluated potential past exposure to TCE in this one well assuming that the highest concentration of TCE measured in the well (25 µg/L) was the concentration residents were exposed to every day during showering.

ATSDR used the inhalation model developed by Andelman and dermal exposure methods documented by EPA to estimate exposures from inhalation and dermal exposure to TCE during showering and time spent in an enclosed bathroom after showering [17,18]. The exposures are put in terms of equivalent 24-hour air concentrations to allow comparison with air health guidelines for TCE. For details of the calculations, please see Appendix A. Using an assumed concentration of 25 µg/L TCE in water, this method predicts that for average shower (10-15 minutes) and average bathroom stays after showering (about 5 minutes), the 24-hour TCE air concentration would range from about 2—4 µg/m³ for infants to adults, as summarized in Table 3.²

² We recognize that infants do not shower; however estimating exposure assuming showers will be protective of bathing because more volatilization occurs during showers.

Table 3. Estimated Equivalent 24-Hour TCE Air Concentrations Resulting from Inhalation and Dermal Exposure to Water Containing 25 µg/L TCE, One Private Well Near the Leeds Metal Site, Leeds, Maine

Age Group	Inhalation Intake (µg)	Skin Intake (µg)	Equivalent 24-Hour TCE Air Concentration from Inhalation and Dermal Exposure to Water Containing 25 µg/L TCE (µg/m ³)
Pregnant Women From 16 Years Old Up To Age 45	44.3	5.8	2.3
Children from Birth Up To 1 Year Old	13.7	1.0	4.2
Children from 1 Year Old Up to Age 2	21.6	1.4	2.9
Children from 2 Years Old Up To Age 3	21.6	1.6	2.6
Children from 3 Years Old Up To Age 6	25.7	2.1	2.7
Children from 6 Years Old Up To Age 11	39.6	3.4	3.6
Children from 11 Years Old Up To Age 16	39.6	5.0	2.9
Children from 16 Years Old Up To Age 21	43.2	5.8	3.0
Adults Greater Than 21 Years Old	44.4	6.2	3.4
All estimated using mean shower and bathroom stay times tabulated by EPA [19]. Shower estimates will overestimate exposures that might occur through bathing because of lower volatilization during baths. Further details on assumptions and example calculations are given in Appendix A. µg = microgram µg/L = microgram per liter µg/m ³ = microgram per cubic meter			

Possible Noncancer Effects from Past TCE Exposure in Private Well Water

TCE-contaminated drinking water has been associated with several health effects [20]. Although the residents served by this private well did not report drinking the water, inhalation of the water and absorbing it through the skin during showering, bathing, or other household uses could result in similar or greater doses than drinking the water. Therefore, a discussion of epidemiology studies of communities exposed to TCE in drinking water follows.

Several epidemiologic studies describe noncancer effects associated with exposure to drinking water contaminated with TCE and other solvents. Note that a positive association does not mean causation. A study of a community in Arizona exposed to elevated levels (up to 239 µg/L) of TCE in drinking water showed an association between maternal exposure to TCE in water while pregnant and congenital heart defects in their newborns [21]. A study of communities in northern New Jersey with drinking water containing TCE greater than 5 µg/L (and other solvents) reported an association between TCE level and oral cleft defects, central nervous system defects, and neural tube defects [22]. A study of people in Woburn, Massachusetts exposed to up to 267 µg/L TCE in drinking water suggested an association between maternal exposure and a combination of eye and ear anomalies and a combination of central nervous system,

chromosomal, and oral cleft anomalies in newborns [23]. However, other researchers have questioned the unusual groupings of these anomalies, and all the studies are limited by the presence of other contaminants in the water which may have led to an association with the observed health effects. Other limitations include small sample sizes and poorly defined TCE exposure levels. Animal studies have confirmed some of the suggested noncancer effects from epidemiologic studies. Rat studies have identified heart defects in newborn rats whose mothers were exposed to TCE in drinking water at doses as low as 0.05 mg/kg/day [24].

EPA's newly issued chronic inhalation Reference Concentration (RfC) for TCE is 0.002 mg/m³, or 2 µg/m³ [25,26]. The estimated 24-hour equivalent air concentrations slightly exceed the RfC. The reference concentration is an estimate, with safety factors built in, of the daily, life-time exposure of human populations to a chemical that is not likely to cause noncancerous health effects. EPA based its RfC on two principal toxicological studies:

- The Johnson study showing increased rates of heart defects in newborn rats born to female rats who were exposed to TCE in drinking water [24]. EPA applied models of TCE metabolism in rats and humans to the study results to obtain a 99th percentile human equivalent concentration (HEC₉₉) of 21 µg/m³. The HEC₉₉ can be interpreted as being the applied concentration in humans for which there is 99% likelihood that a randomly selected individual will have an internal dose less than or equal to the internal dose derived from the animal study.
- A study in female adult mice showing immune system effects (decreased thymus weight) after exposure to TCE in drinking water [27]. EPA converted the study findings to obtain a HEC₉₉ of 190 µg/m³.

An additional study was also cited as supporting the RfC:

- A study showing kidney damage (toxic nephropathy) in female rats exposed to TCE by gavage for 2 years [28]. EPA obtained a HEC₉₉ of 30 µg/m³ for lifetime continuous exposure.

The estimated TCE 24-hour equivalent TCE concentrations for pregnant women and all age groups are much lower than the human equivalent concentrations on which the RfC is based. Noncancer health effects are unlikely for past exposure to TCE from showering in water from this private well. Exposure to TCE can also cause cancer, with increased susceptibility for early-life exposures. The following section provides more detail.

Possible Cancer Effects from Past TCE Exposure in Private Well Water

The National Toxicology Program (NTP) classifies TCE as reasonably anticipated to be a human carcinogen [29]. In humans, occupational exposure to TCE was associated with excess incidences of several cancers, particularly liver cancer, non-Hodgkin's lymphoma, and kidney cancer [29]. Animal studies showed that TCE exposure caused tumors in mice and rats at several different sites, including liver and kidney, by inhalation or oral exposure [29]. The International Agency for Research on Cancer (IARC) has determined that TCE is a probable human carcinogen based on epidemiological studies showing increased rates of liver cancer and non-Hodgkin's lymphoma, primarily in workers who were exposed to TCE on the job, and animal

studies showing increased numbers of liver and kidney tumors upon oral administration [30]. EPA characterizes TCE as carcinogenic to humans by all routes of exposure [25,26]. This conclusion is based on human epidemiology studies showing associations between human exposure to TCE and kidney cancer, non-Hodgkin's lymphoma, and liver cancer.

EPA's inhalation unit risk (IUR) of $4.1 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$ for TCE reflects total incidence of kidney, non-Hodgkin's lymphoma, and liver cancers [25,26]. EPA has concluded that TCE is carcinogenic by a mutagenic mode of action for induction of kidney tumors. As a result, increased early-life susceptibility is assumed for kidney cancer, and age-dependent adjustment factors (ADAFs) are used for the kidney cancer component of the total cancer risk when estimating age-specific cancer risks [26]. ADAFs are factors by which cancer risk is multiplied to account for increased susceptibility to mutagenic compounds early in life – standard ADAFs are 10 (for ages below 2 years old), 3 (for ages 2 up to 16 years old), and 1 (for ages 16 years old and greater) [31].

Although technically the ADAF is only applied to the kidney cancer component of the TCE IUR, for the purposes of this PHA we apply the ADAFs to the entire IUR. This will overestimate the potential for increased cancer risk. For a given period of exposure, the IUR is multiplied by the 24-hour equivalent TCE air concentration, appropriate ADAF, and a fraction corresponding to the fraction of a 78-year lifetime under consideration, to obtain the increased risk of cancer. For example, for the first year of life, the estimated equivalent 24-hour TCE concentration was $4.2 \mu\text{g}/\text{m}^3$ and the equation for estimating increased cancer risk from the first year of exposure is:

$$\begin{aligned} \text{Increased risk} &= 4.2 \frac{\mu\text{g}}{\text{m}^3} \times \frac{1 \text{ year of exposure}}{78 \text{ - yr lifetime}} \times 10(\text{ADAF}) \times \left(4.1 \times 10^{-6} \left(\frac{\mu\text{g}}{\text{m}^3} \right)^{-1} \right) \\ &= 0.0000022, \text{ which can also be stated } 0.022 \text{ out of } 10,000 \text{ or } 2.2 \text{ out of a million.} \end{aligned}$$

To get a “worst-case” estimate of increased cancer risk from past exposure to TCE in this well, ATSDR assumed exposure began when the well was installed in 2006, and continued until the exposure pathway was interrupted by installation of a filter on the well in 2011. ATSDR used the 24-hour equivalent air concentrations of TCE estimated for various age groups listed in Table 3 and assumed continuous exposure to these concentrations for 5 years.

Table 4 lists the estimated increased cancer risk from these calculations. As indicated in Table 4, out of 10,000 people exposed every day to the equivalent 24-hour air concentration based on the highest concentration of TCE detected in the well, less than one additional case of cancer would be predicted to occur due to the TCE exposure. This is within EPA's acceptable risk range for Superfund of 1 in 1,000,000 to 1 in 10,000 (or 0.01 to 1 in 10,000). It is unlikely that past exposure to TCE from showering in water from this private well (or from other household uses) will measurably increase the risk of cancer, even for children who were exposed from birth.

**Table 4. Estimated Increased Cancer Risk from Inhalation and Dermal Exposure to TCE
In One Private Well Near the Leeds Metal Site, Leeds, Maine**

Age Group	24-Hour Equivalent Air Concentration of TCE, $\mu\text{g}/\text{m}^3$	Years of Exposure / Lifetime	ADAF	IUR, $(\mu\text{g}/\text{m}^3)^{-1}$	Estimated Increased Cancer Risk, out of 10,000
Children from Birth Up To 1 Year Old	4.2	1/78	10	4.1×10^{-6}	0.022
Children from 1 Year Old Up to Age 2	2.9	1/78	10	4.1×10^{-6}	0.015
Children from 2 Years Old Up To Age 3	2.6	1/78	3	4.1×10^{-6}	0.004
Children from 3 Years Old Up To Age 5	2.7	2/78	3	4.1×10^{-6}	0.009
Total		5/78			0.05
ADAF = Age dependent adjustment factor IUR = Inhalation Unit Risk $\mu\text{g}/\text{m}^3$ = microgram per cubic meter					

Past and Current Exposure to Arsenic in Private Well Water

Arsenic was detected in 24 of the 29 private wells tested for arsenic near the site. Nineteen of the wells had arsenic concentrations greater than ATSDR's CV for noncancer effects ($3 \mu\text{g}/\text{L}$), and in all 24 wells the arsenic concentration exceeded the cancer CV ($0.02 \mu\text{g}/\text{L}$). Table 5 lists the arsenic concentrations detected in each of the wells. Arsenic appears to be more evenly distributed throughout the private wells (compared to the TCE, which was only present in a few wells). Arsenic does occur naturally in water formations in several parts of Maine, and the state has information on arsenic in private wells available online [32]. We recognize that arsenic in private wells near the Leeds Metal Site may not be directly related to the site. However, because this exposure might affect public health, we evaluate arsenic in this PHA to determine if exposure to it in drinking water could harm health.

Exposure to arsenic in private wells is assumed to have occurred in the past, and exposure is continuing. The carbon filters installed on 4 wells to remove TCE are not effective at removing arsenic. A different treatment known as reverse osmosis is needed to remove arsenic [32].

Table 5. Arsenic Concentration For 14 Private Wells With Arsenic Detected, Out Of 24 Wells Tested – Leeds Metal Site, Leeds, Maine

Well ID*	Detected Arsenic Concentration in µg/L**
A1	28.9
B1	26.8
C1	23.1
D1	22.2
E1	21.2
F1	17.5
G1	16.1
H1	13
I1	12.6
J1	12.5
K1	12.3
L1	12.3
M1	11.9
N1	11.1

* Table lists 14 wells that had arsenic above analytical detection limit. Wells listed in order of decreasing arsenic concentration; no particular relation to location or to well IDs listed in Table 2.
 ** Typical detection limit 1–2 µg/L
 µg/L = microgram per liter

To evaluate how exposure to arsenic might affect health, ATSDR estimated exposure doses for average and high water consumption rates. Assumptions used in these estimates are detailed in Appendix A. An example calculation for arsenic exposure from drinking water is shown below. For a child less than one year old (average weight 7.8 kg) drinking 1.1 liters of water (about 4 8-ounce glasses) containing the highest concentration of arsenic (28.9 µg/L or 0.0289 mg/L) every day:

$$Dose = \frac{0.0289 \frac{mg}{L} \times 1.1 \frac{L}{day}}{7.8 kg} = 0.004 mg / kg / day$$

Estimated exposure doses for young children and other age groups are shown in Table 6. The table includes calculations based on continuous exposure to 10 µg/L, 20 µg/L, and 30 µg/L arsenic to encompass the range of arsenic concentrations detected in the private wells; each dose is expressed as a range reflecting the dose based on average to 95th percentile water consumption rates.

Table 6. Estimated Arsenic Exposure Doses for Drinking from Private Wells Near the Leeds Metal Site, Leeds, Maine

Group	Estimated Arsenic Exposure Dose Range*, mg/kg/day					
	10 µg/L Arsenic		20 µg/L Arsenic		30 µg/L Arsenic	
	Average*	High*	Average*	High*	Average*	High*
Children from Birth Up to 1 Year Old	0.00064	0.0014	0.0013	0.0028	0.0019	0.0042
Children from 1 Year Old up to Age 2	0.00035	0.00079	0.00070	0.0016	0.0011	0.0024
Children from 2 Years Old Up To Age 3	0.00036	0.00065	0.00072	0.0013	0.0011	0.0020
Children from 3 Years Old Up To Age 6	0.00032	0.00054	0.00065	0.0011	0.0010	0.0016
Children from 6 Years Old Up To Age 11	0.00016	0.00044	0.00031	0.00088	0.00047	0.0013
Children from 11 Years Old Up To Age 16	0.00011	0.00035	0.00021	0.00070	0.00032	0.0011
Children from 16 Years Old Up To Age 21	0.00011	0.00034	0.00022	0.00067	0.00034	0.0010
Adults Greater Than 21 Years Old	0.00015	0.00038	0.00030	0.00075	0.00045	0.0011
Health Guideline, in mg/kg/day	0.0003 – chronic MRL		0.0003 – chronic MRL		0.0003 – chronic MRL	
<p>* Column heading indicates water consumption used for exposure estimate: Average= mean water consumption; High = 95th percentile water consumption; both as listed in [19]. Please see Appendix A for exposure assumptions and example calculations. Note: Highlighted cells indicate values equal to or greater than the health guideline.</p> <p>mg/kg/day = milligrams per kilogram per day µg/L = microgram per liter</p>						

Possible Noncancer and Cancer Effects from Arsenic Exposure in Private Well Water

Ingesting or breathing low levels of inorganic arsenic for a long time can cause skin changes (such as the appearance of dark spots or small "corns" or "warts" on the palms, soles, and torso), as well as changes in blood chemistry and neurologic and cardiovascular effects [33]. ATSDR's minimal risk level of 0.0003 mg/kg/day was developed on the basis of a human study that did not report any health effects at an arsenic dose of 0.0008 mg/kg/day [33]. Skin changes were observed at a chronic arsenic dose of 0.014 mg/kg/day.

Several of the estimated doses in Table 6 are higher than the minimal risk level for arsenic exposure of 0.0003 mg/kg/day, and at higher arsenic concentrations and for younger children, the estimated arsenic exposures for this site exceed the no effect level of 0.0008 mg/kg/day. But even the highest estimated dose is still 3.5 times smaller than the 0.014 mg/kg/day effect level for skin lesions. Although unlikely, regularly drinking water with higher concentrations of arsenic could increase the risk of developing dark spots and hardened, corn-like lesions on the skin.

Arsenic is classified by the NTP as a known human carcinogen, and it has been associated with liver, kidney, lung, and skin cancer (especially basal and squamous cell carcinoma) [29]. Based on EPA’s oral cancer slope factor for arsenic of $1.5 \text{ (mg/kg/day)}^{-1}$, a lifetime of drinking water with the arsenic concentrations measured in private wells at this site would result in increased risks of cancer greater than EPA’s acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000) [34]. For people who drink an average amount of water every day, the arsenic concentrations at this site may increase the estimated risk of cancer by a low to moderate amount – that is, out of 10,000 people drinking the water every day for a lifetime, 3-6 additional cases of cancer might be attributable to arsenic. For people who drink higher amounts of water than most people, the lifetime increased risk of cancer is predicted to be 7 to 18 additional cases per 10,000 people exposed. This would be considered a moderate to high increased risk of cancer. Table 7 below tabulates the increased risk of cancer for the various age groups and arsenic concentrations considered in this analysis, along with the predicted lifetime increased risk of cancer.

Table 7. Estimated Increased Cancer Risk From Arsenic Exposure From Drinking From Private Wells Near the Leeds Metal Site, Leeds, Maine

Group	Estimated Increased Cancer Risk*, estimated number of additional cases out of 10,000		
	10 µg/L Arsenic	20 µg/L Arsenic	30 µg/L Arsenic
Children from Birth Up to 1 Year Old	0.1–0.3	0.3–0.6	0.4–0.9
Children from 1 Year Old up to Age 2	0.08–0.2	0.2–0.3	0.2–0.5
Children from 2 Years Old Up To Age 3	0.08–0.1	0.2–0.3	0.2–0.4
Children from 3 Years Old Up To Age 6	0.2–0.3	0.4–0.7	0.6–1
Children from 6 Years Old Up To Age 11	0.2–0.5	0.3–0.9	0.5–1
Children from 11 Years Old Up To Age 16	0.1–0.4	0.2–0.8	0.3–1
Children from 16 Years Old Up To Age 21	0.1–0.4	0.2–0.7	0.4–1
Adults Greater Than 21 Years Old	2–4	3–8	5–12
Lifetime Increased Cancer Risk	3–6	5–12	7–18

* Range of cancer risk estimates based on average to 95th percentile water consumption as listed in [19]. Please see Appendix A for exposure assumptions and example calculations. Note: Highlighted cells indicate lifetime excess cancer risks greater than EPA’s acceptable risk range for Superfund (0.01 to 1 out of 10,000).
µg/L = microgram per liter

Summary of Possible Health Effects from Private Well Water

In summary, only one private well near the site had TCE levels above the MCL, and past exposures based on reported use of this well were too low to result in harmful effects. This well is now filtered, so exposure to TCE is not occurring. The concentrations of TCE in all other private wells tested were too low to cause noncancer or cancer health effects. Arsenic was present in several wells at levels that could cause an increased risk of skin changes (dark spots or hard, corn-like lesions on the hands, feet, or torso) and an increased lifetime risk of cancer. This exposure occurred in the past and continues at present as long as people drink from untreated wells.

On-Site Soil Pathway

People gain access to the site for recreation and to scavenge for scrap metal, despite reported efforts by the property owner to post signage and restrict access to the site. Teenagers and adults trespassing on the site could be exposed to site contaminants, particularly in soil, through *incidental ingestion* (accidentally swallowing dust and soil particles clinging to skin or raised up into the air during activities), *inhalation* (breathing in soil particles suspended in the air as dust), or *dermal exposure* (getting contaminated soil on the skin). As components of an overall exposure dose, incidental exposure is typically the main contributor, with dermal exposure important for some contaminants. Inhalation exposure is typically only important for activities that suspend large amounts of dust, like ATV-riding.

State and federal agencies have conducted several investigations including collection of soil to determine contaminants present. Because they encompassed the greatest portion of the site, ATSDR focused its evaluation on surface soil and subsurface soil results from EPA's 2010 Preliminary Assessment/ Site Inspection and 2011 Site Inspection [3,2]. These results are used for the evaluation of soil exposures in the ensuing section. ATSDR also examined data from earlier and more focused studies to ensure that they were generally consistent with the two EPA studies used for quantitative exposure estimates (they were).

The soil sampling results reported in EPA's reports were split into surface and subsurface samples, but the actual depths of soil collected for each sample varied. ATSDR typically considers surface soils available for exposure to only include the top two inches of soil; the surface soil samples in the EPA reports were collected from up to 24 inches below the ground surface. The subsurface soils listed in the EPA reports also varied; some were as deep as 10 feet below the ground surface. For ATSDR's evaluation, we split the soil samples and considered any sample that included the surface (zero inches below ground surface) to be accessible by trespassers walking or hiking on the site. For trespassers riding ATVs or digging for metal in the soil, we added samples collected from anywhere 3 feet or less below the ground surface to the soils accessible for exposure. These two categories are denoted "surface" (including the actual surface) and "subsurface" (anything collected less than 3 feet below the surface). We considered soil samples collected greater than 3 feet below the ground surface to be inaccessible by trespassers.

Table 8 presents the contaminants that were detected at least once in surface or subsurface soil above CVs for soil. For these contaminants, an exposure dose was estimated for the trespasser and adult scavenger scenarios. To evaluate chronic effects, ATSDR estimated total exposure

dose (including incidental ingestion, dermal, and inhalation exposures) assuming trespassers entered the site on an ATV and spent time riding, walking, or digging on site up to 72 days a year. Appendix A contains details of the assumptions used and example calculations for the exposure dose calculations. Trespassers were assumed to be exposed at a contaminant concentration represented by the 95th percentile of the surface soil concentrations, and adults digging for metal on-site were assumed to be exposed at a contaminant concentration represented by the 95th percentile of the surface and subsurface soil concentrations.

The available data included samples from throughout the former operational areas of the site, and no clustering of high contaminant concentrations in particular areas was apparent. Because exposure would occur throughout the site, long-term exposure is to an average rather than maximum contaminant concentration. Using the 95th percentile gives a more conservative (higher) average exposure estimate to account for the fact that the site may not be fully characterized.

The estimated doses (given in ranges encompassing average to high-end soil ingestion rates) are presented in Table 9 along with noncancer health guideline values (doses generally considered unlikely to result in any adverse noncancer health effects). Estimated doses for cancer-causing contaminants are also used to estimate the increased risk of cancer.

Table 8. Summary of Contaminants Detected Above Comparison Values* (CVs) in Soil on the Leeds Metals Site, ME

Contaminant	Highest Concentration Detected, mg/kg (# Detected Above Non-cancer CV / # of Samples)		Non-cancer CV in mg/kg	Oral Cancer CV in mg/kg; National Toxicology Program Cancer Class
	Surface Soil [†]	Subsurface Soil [†]		
Arsenic	199 (10 / 29)	205 (15 / 45)	20 – EMEG	0.5 – CREG; Known human carcinogen
Cadmium	183 (17 / 29)	306 (21 / 45)	5 - EMEG	No oral cancer CV; Known human carcinogen
Chromium	606 (16 / 29)	1,190 (21 / 45)	50 – EMEG for hexavalent chromium	0.29 – RSL based on cancer; hexavalent chromium is a known human carcinogen
Copper	38,000 (17 / 29)	180,000 (19 / 45)	500 – iEMEG	No cancer CV; Not classified
Lead	39,000 (18 / 29)	20,900 (19 / 45)	400 – SSL	No cancer CV; Reasonably anticipated to be a carcinogen
Aroclor-1254	6 (5 / 16)	27 (12 / 40)	1 –EMEG	0.4 – CREG for PCBs; Reasonably anticipated to be a carcinogen
Aroclor-1242	None detected above CV (0 / 16)	8 (1 / 40)	1 – EMEG for Aroclor-1254	0.4 – CREG for PCBs; Reasonably anticipated to be a carcinogen
Aroclor-1248	2 (1 / 16)	2 (1 / 40)	1 – EMEG for Aroclor-1254	0.4 – CREG for PCBs; Reasonably anticipated to be a carcinogen
Aroclor-1260	6 (7 / 16)	12 (12 / 40)	1 – EMEG for Aroclor-1254	0.4 – CREG for PCBs; Reasonably anticipated to be a carcinogen
Aroclor-1268	None detected above CV (0 / 16)	2 (2 / 40)	1 – EMEG for Aroclor-1254	0.4 – CREG for PCBs; Reasonably anticipated to be a carcinogen

* Please see Appendix A for definitions and additional information about CVs.

CV = comparison value

mg/kg = milligrams of contaminant per kilogram of soil

MCL = maximum contaminant level

EMEG = environmental media evaluation guide for chronic duration (greater than one year)

RMEG = reference media evaluation guide

iEMEG = environmental media evaluation guide for intermediate duration (greater than 2 weeks to one year)

RSL = regional screening level

CREG = cancer risk evaluation guide

[†]As defined in discussion on page 21.

Data sources: References [2,3]; soil data examined but not tabulated from other references as summarized in “Data Used” section beginning on page 5.

Note: Contaminants detected at values exceeding the lowest CV were selected for further evaluation.

Table 9a. Estimated Exposure Doses for On-Site Soil Pathway, Leeds Metal Site, Leeds, Maine

Group	Estimated Total Exposure Dose Range*, mg/kg/day									
	Arsenic		Cadmium		Chromium		Copper		Lead	
	Avg.*	High*	Avg.*	High*	Avg.*	High*	Avg.*	High*	Avg.*	High*
Children from 11 Years Old Up To Age 16	0.00007	0.0001	0.00005	0.0001	0.0002	0.0004	0.008	0.01	0.003	0.006
Children from 16 Years Old Up to Age 21	0.00006	0.0001	0.00004	0.00008	0.0002	0.0003	0.006	0.01	0.003	0.005
Adults Greater than 21 Years Old	0.00003	0.00006	0.00002	0.00004	0.00009	0.0002	0.003	0.006	0.001	0.002
Diggers for Metal – Adults Greater than 21	0.0001		0.0001		0.0006		0.01		0.006	
Health Guideline, in mg/kg/day	0.0003 – chronic MRL		0.0001 – chronic MRL		0.001 – chronic MRL for hexavalent chromium		0.01 – intermediate MRL		none	

* Column heading indicates exposure assumption used for exposure estimate: Avg.= mean incidental soil ingestion; High = upper percentile incidental soil ingestion; both as listed in [19]. Dose includes incidental ingestion, dermal, and inhalation exposure. MRL = minimal risk level mg/kg/day = milligrams per kilogram per day Note: Highlighted cells indicate values equal to or greater than the health guideline. Please see Appendix A for exposure assumptions and example calculations.

Table 9b. Estimated Exposure Doses for On-Site Soil Pathway, Leeds Metal Site, Leeds, Maine

Group	Estimated Total Exposure Dose Range*, mg/kg/day									
	Aroclor-1242		Aroclor-1248		Aroclor-1254		Aroclor-1260		Aroclor-1268	
	Avg.*	High*	Avg.*	High*	Avg.*	High*	Avg.*	High*	Avg.*	High*
Children from 11 Years Old Up To Age 16	0.0000001	0.0000001	0.000001	0.000002	0.000004	0.000006	0.000003	0.000005	0.0000003	0.0000005
Children from 16 Years Old Up to Age 21	0.00000009	0.0000001	0.000001	0.000001	0.000003	0.000005	0.000002	0.000004	0.0000003	0.0000004
Adults Greater than 21 Years Old	0.00000007	0.00000008	0.0000007	0.000001	0.000002	0.000003	0.000002	0.000003	0.0000002	0.0000003
Diggers for Metal – Adults Greater than 21	0.000002		0.000004		0.00001		0.000008		0.000001	
Health Guideline, in mg/kg/day	0.00002 – chronic MRL for Aroclor-1254		0.00002 – chronic MRL for Aroclor-1254		0.00002 – chronic MRL		0.00002 – chronic MRL for Aroclor-1254		0.00002 – chronic MRL for Aroclor-1254	
* * Column heading indicates exposure assumption used for exposure estimate: Avg.= mean incidental soil ingestion; High = upper percentile incidental soil ingestion; both as listed in [19]. Dose includes incidental ingestion, dermal, and inhalation exposure. MRL = minimal risk level mg/kg/day = milligrams per kilogram per day Please see Appendix A for exposure assumptions and example calculations.										

Chronic Exposure to Contaminants in On-Site Soils

Arsenic

All of the estimated arsenic doses were lower than the MRL of 0.0003 mg/kg/day. Therefore, no adverse noncancer effects are expected from exposure to arsenic in on-site soil.

Arsenic is classified by the NTP as a known human carcinogen [29,33]. Based on EPA's oral cancer slope factor for arsenic of $1.5 \text{ (mg/kg/day)}^{-1}$, the highest doses of arsenic in soil estimated for trespassers, continued for up to 20 years, would result in an increased risk of cancer of 5.0×10^{-5} , or 5 in 100,000. This is within EPA's acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000). Lower durations of exposure or lower doses would result in even lower risks. Exposure to arsenic in soils during trespassing in itself is not likely to result in a measurably increased risk of cancer. However, this exposure could contribute to an unacceptable risk if the trespasser were exposed elsewhere to arsenic (in private well water, for example).

Cadmium

Most teenage and adult trespasser estimated doses were lower than the MRL, and no non-cancer adverse health effects are likely. For the scenarios of 11-16 year old trespassers with high soil ingestions and adults digging for scrap metal, the estimated doses are equivalent to the MRL of 0.0001 mg/kg/day. This value is based on a meta-analysis of several human epidemiology studies showing increased levels of low molecular weight protein biomarkers in urine as a sign of kidney toxicity due to chronic cadmium exposure [35]. The point of departure, 0.5 micrograms of cadmium per gram creatinine in urine, was the statistical lower confidence level of the lowest reported urinary cadmium level of any study. A model was used to predict the chronic cadmium intake that would result in this urinary cadmium level at age 55; the lowest intake was found to be 0.0003 mg/kg/day.

The dose of cadmium estimated for this site is lower than the intake that could lead to kidney damage. Moreover, the estimated dose for the trespasser scenario used a number of very conservative assumptions—it is unlikely that trespassers would have exposure as high as estimated here. For these reasons, ATSDR concludes that no adverse noncancer health effects would result from exposure to cadmium in on-site soils.

Cadmium is classified as a known human carcinogen by the NTP and was shown to cause tumors when administered to experimental animals by inhalation, orally, or by injection [29]. However, EPA has not developed an oral cancer slope factor for cadmium. Therefore, no quantitative assessment of the likelihood for increased risk of cancer from the estimated oral exposure to cadmium in site soil is possible. The rat study in which oral exposure led to increases in tumors involved the rats eating food containing high levels of cadmium every day for a year and a half. It is unlikely that the occasional incidental ingestion of soil by trespassers at this site would approach the effect levels in this study [36].

EPA has developed an inhalation unit risk for cadmium of $0.0018 \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ [26]. For inhalation of cadmium in soil suspended during ATV riding, the predicted increased risk of cancer is 1.7×10^{-6} , or about 2 in 1,000,000. This is within EPA's acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000).

Chromium

All of the estimated chromium doses were lower than the MRL for hexavalent chromium of 0.001 mg/kg/day. Only a fraction of chromium in soil is present in the more toxic hexavalent form; however, even if all of the chromium in soil was hexavalent, the exposure would be unlikely to result in any harm. Therefore, no adverse noncancer effects are expected from exposure to chromium in on-site soil.

Hexavalent chromium is classified as a known human carcinogen by the NTP [29]. Not enough information exists on other forms of chromium to determine their carcinogenicity. Even if all the chromium measured in soil at the site were of the hexavalent form, it is unlikely exposure to site soil would increase the risk of cancer. California EPA's Office of Environmental Health Hazard Assessment published an oral cancer slope factor for hexavalent chromium of $0.42 \text{ (mg/kg/day)}^{-1}$ [37]. Using this cancer slope factor, and assuming all the chromium in the soil is hexavalent, the predicted increased risk of cancer is 4.9×10^{-5} , or about 5 in 100,000. This is within EPA's acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000).

Copper

Some of the estimated copper doses exceeded the intermediate MRL of 0.01 mg/kg/day. No chronic MRL is available for copper. The intermediate MRL is based on a human study of men and women ingesting copper sulfate in drinking water for 2 months [38]. No effects were observed at a dose of 0.042 mg/kg/day; at higher doses, gastrointestinal effects were observed.

All the estimated doses are lower than the no effect level observed in this study. In addition, ingesting copper as a component of soil is very different than drinking a copper compound in water; it is likely that far less copper will be absorbed from soil than in the water exposures. Therefore, no adverse effects are expected from exposure to copper in on-site soil.

Lead

Lead, even at low levels, in children's blood has been associated with small decreases in IQ and slightly impaired hearing and growth [39]. The risk is considered greatest in children under 6 years old. The Centers for Disease Control and Prevention uses a reference value based on the 97.5th percentile of the population blood lead level in children ages 1-5 (currently 5 µg/dL) to identify children and environments associated with lead-exposure hazards [40]. No children under the age of 6 are likely to trespass on the site.

Occupational and general population studies of adults have shown association of blood lead with many effects including high blood pressure, blood changes, kidney effects, and tooth and bone problems [39].

A slope factor for the increase in blood lead concentration per increase in soil lead concentration for males aged 18-65 has been calculated as 0.001—0.003 µg/dL blood per mg/kg lead in soil [39]. Assuming this slope factor is valid for teenagers and females trespassing on the site, this would suggest that regular exposure to the 95th percentile value for lead in surface soil (8,204 mg/kg) could cause a rise in blood lead levels of 8-25 µg/dL. A blood level increase this large would be of serious concern, but such an increase is not likely because the slope factor used is not really applicable for trespasser exposures. The slope factor assumes a continuous, residential

exposure; the intermittent nature of trespassing (conservatively assumed as 72 days a year, only 20% of the time) makes such a large increase in blood lead unlikely. The body removes lead from blood, with half of the lead in blood being removed in about a month [39]. Therefore, intermittent exposures will not build up in blood as much as continuous ones. These considerations suggest that trespasser exposure to lead in on-site soils is unlikely to significantly increase the risk of adverse health effects. This conclusion only applies to the trespasser scenario as evaluated here; further evaluation would be needed if the site was accessed by younger children or for other uses, such as industrial or residential.

Lead is classified by the NTP as reasonably anticipated to be a human carcinogen based on evidence in animal studies that lead exposure was associated with tumors, mainly of the kidney, but also brain, blood system, and lung [29]. Limited human epidemiology studies show a weak association between lead exposure and cancer of the lung or stomach [29]. EPA has not developed an oral cancer slope factor for lead because it determined that typical methods for quantifying cancer risk would not yield an accurate prediction of the risk from lead exposure [34]. Therefore, no quantitative assessment of the likelihood for increased risk of cancer from the estimated oral exposure to lead in site soil is possible.

Aroclor Mixtures

Aroclors are mixtures of polychlorinated biphenyls (PCBs), and are numbered as an indication of the degree of chlorination of the compounds in the mixture (e.g., Aroclor-1254 contains 54% chlorine). An MRL is available for PCBs in general, based on a study of Aroclor-1254 [41]. For this evaluation, all the Aroclor mixtures were compared to the MRL for PCBs.

All of the estimated doses for various Aroclor mixtures were lower than the MRL of 0.00002 mg/kg/day for PCBs [41]. No adverse noncancer effects are expected from exposure to Aroclor mixtures in on-site soil.

PCBs are classified by the NTP as reasonably anticipated to be carcinogenic [29]. No oral cancer slope factors for specific Aroclor mixtures are available, but an upper-bound slope factor of $2.0 \text{ (mg/kg/day)}^{-1}$ is available for high-risk, high persistence PCBs [34]. Applying this conservative slope factor to the Aroclor mixtures measured at this site results in predicted increased risks of cancer ranging from 4.9×10^{-7} to 4.3×10^{-6} , or 0.5 to 4 in 1,000,000. This is within EPA's acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000).

Cumulative Increased Cancer Risk from Exposure to On-Site Soils

Summing the predicted increased risks of cancer for all the carcinogenic compounds discussed in the previous section results in a total predicted risk of cancer of about 9.9×10^{-6} , or about 1 in 100,000. This is within EPA's acceptable risk range for Superfund (1 in 1,000,000 to 1 in 10,000). The actual increased risk of cancer from exposure to soils while trespassing is probably even lower than estimated due to the conservative assumptions used for contaminant concentration, exposure frequency and exposure duration. Exposure to contaminants in on-site soil while trespassing is unlikely to result in a measurably increased risk of cancer.

Acute Exposures from Inhalation of Dust While ATV-Riding

Some contaminants may have direct health effects when breathed in. We focus only on acute inhalation effects here (long-term exposure to contaminants from inhaling dust was included in the total exposure doses discussed previously). The only contaminant with an acute CV for air is cadmium, with an acute MRL of $0.03 \mu\text{g}/\text{m}^3$. To estimate the air concentration of cadmium suspended in the air from ATV riding on the Site, ATSDR used a particle emission factor of $1.18 \times 10^{-6} \text{ kg of soil per m}^3 \text{ of air}$, a value developed by EPA for ATV riding [45].

The estimated cadmium concentration in air during ATV riding (based on the 95th percentile of surface and subsurface soil concentrations of $116 \text{ mg}/\text{kg}$) is:

$$116 \frac{\text{mg cadmium}}{\text{kg soil}} \times 1.18 \times 10^{-6} \frac{\text{kg soil}}{\text{m}^3 \text{ air}} \times 1000 \frac{\mu\text{g}}{\text{mg}} = 0.14 \mu\text{g}/\text{m}^3 \text{ cadmium}$$

The estimated concentration of cadmium in air is higher than the acute MRL of $0.03 \mu\text{g}/\text{m}^3$. This value is based on an inhalation study in which cadmium oxide was administered to rats for 6.2 hours a day, 5 days a week for 2 weeks. Rats exposed to $88 \mu\text{g}/\text{m}^3$ and higher of cadmium showed inflammation and cellular changes in lung cells. The estimated concentration of cadmium in air during ATV riding is hundreds of times smaller than the effect level of the study. Moreover, trespassers on the site have a much less frequent exposure than in this study, and cadmium in soil is not likely to be as well absorbed as the cadmium oxide used in the study. Acute effects from inhaling cadmium are unlikely for trespassers riding ATVs on the site.

Potential Exposure Pathways

Vapor Intrusion

If VOC levels are high enough in groundwater and the groundwater is close enough to the surface, sometimes VOCs can move through the soil above the water table and/or through cracks or gaps in the subsurface. If the travel pathway leads to a building's interior through a basement, crawl space, or cracks in the foundation, it is possible for the contaminant to build up inside. This is known as *vapor intrusion*, and in some cases vapors from contaminants can reach levels of health concern. EPA recommends evaluating the potential for vapor intrusion at sites where volatile substances are suspected to be present in soil or groundwater at 100 feet of depth or less near existing or future buildings [42]. Stopping or preventing vapor intrusion may involve techniques such as sealing foundation cracks or improving ventilation of the homes to allow vapors to dissipate.

It is unlikely that vapor intrusion is a problem near the Leeds Metal Site. EPA guidance suggests vapor intrusion is not expected to be an issue at a target groundwater concentration of $5.3 \mu\text{g}/\text{L}$ for TCE [42]. All but one well had TCE concentrations in well water less than $5 \mu\text{g}/\text{L}$, too low to cause a concern for vapor intrusion. One home's well had a TCE concentration as high as $25 \mu\text{g}/\text{L}$. This well draws water from the bedrock more than 100 feet below the ground surface, and there is no information on contaminant concentrations in the groundwater directly below the house. However, because the contaminants from the site would tend to sink in the groundwater as they travel from the site because they are heavier than water, it's very unlikely that the

groundwater directly below the house has a concentration of TCE that would be of concern for vapor intrusion. As more information about the site contamination is collected, ATSDR will work with EPA and ME DEP to ensure that any issues with vapor intrusion near the site are considered and addressed.

Incidental Exposure to Surface Water or Sediment

Limited data are available on contaminant levels in surface water or sediment on the site. Given the observed and reported uses of the site, ATSDR considered incidental exposure to surface water and sediment less likely than soil to result in harmful exposures. If this information changes, and if additional data on surface water or sediment become available through the remedial investigation process, ATSDR will evaluate these potential exposure pathways.

Children's Health Considerations

ATSDR recognizes that infants and children might be more vulnerable than adults to exposures in communities with contaminated air, water, soil, or food. This potential vulnerability results from the following factors: 1) children are more likely to play outdoors and bring food into contaminated areas; 2) children are shorter and therefore more likely to contact dust and soil; 3) children's small size results in higher doses of chemical exposure per kg of body weight; and 4) developing body systems can sustain permanent damage if toxic exposures occur during critical growth stages. Because children depend completely on adults for risk identification and management decisions, ATSDR is committed to evaluating their special interests at the site.

Pregnant women and small children were potentially exposed to TCE-contaminated private well water before the filters were installed. ATSDR considered these potentially sensitive groups in estimating exposures and possible health effects. ATSDR also estimated arsenic in well water exposures assuming exposure begins at birth and continues throughout life.

Older children and teenagers may be exposed to on-site soil while trespassing or riding ATVs on the site. ATSDR used age-group specific exposure assumptions to get the best estimate of exposures experienced by these special groups.

Community Health Concerns

In producing a PHA, ATSDR attempts to respond to communities' health concerns about the site. ATSDR met with local Selectmen and officials who have worked on the site for several years and asked them to share any health concerns related to site exposures. We also met with community members in a public availability session held during the public comment period for the draft PHA. Residents expressed concerns about the rate of cancer in the area. ATSDR found that, in the current situation, exposures to site contaminants are not expected to contribute to an increased risk of cancer. We did find that some wells contain arsenic (commonly found in Maine) at levels that could contribute to an increased risk of cancer. ATSDR recommends that people not drink water from these wells without treating it to remove arsenic.

Conclusions and Next Steps

Conclusions

ATSDR reached three important conclusions in the PHA:

Conclusion 1

People using private wells near the site are currently at no risk of harmful effects from trichloroethylene (TCE) in water. Private well water may contain harmful levels of arsenic, which may not be directly related to the site.

Basis for Conclusion

All private wells have TCE too low to cause harmful effects or have filters to remove TCE. Only one well has TCE above EPA's Maximum Contaminant Level (MCL). This well is filtered to remove TCE. Past exposure to TCE in this well through reported use of the well was unlikely to result in harmful cancer or noncancer effects. Several private wells contain arsenic at levels that could increase the risk of developing skin lesions and could increase the lifetime risk of cancer, especially for people who drink higher-than-average amounts of the water. This is an issue in private wells in many parts of Maine.

Next Steps

- ATSDR recommends ME DEP continue sampling all private wells to ensure TCE-removing filters are operating properly and to evaluate whether filters may be needed for any additional wells.
- Homeowners whose private wells contain arsenic above drinking water standards should consider installing a reverse osmosis treatment system or switching to another source for drinking water. Carbon filters will not remove arsenic from water.
- Homeowners are encouraged to consult <http://wellwater.maine.gov> for more information about treatment systems for private wells.

Conclusion 2

While trespassing on the site is illegal and discouraged, exposure of trespassers to contaminants in on-site soil is unlikely to result in harmful health effects. However, the site contains physical hazards that could cause injury.

Basis for Conclusion

Estimated exposures to contaminants in soil during occasional trespassing, digging on site, or ATV riding are too low to result in harmful health effects. However, many physical hazards exist on the site, including steep and possibly unstable slopes, sharp pieces of metal and rebar that may cause injury, and at least one concrete pit containing water.

Next Steps

- Property owners and/or EPA should install effective fencing, gates, and warning signs to keep trespassers from entering the site.

Conclusion 3 Further investigation of the nature and extent of contamination at the site is needed.

Basis for Conclusion This PHA is based on limited data available through EPA’s NPL listing process and reported current uses of the site. Changing use of the site would require a re-evaluation of potential health effects of pathways. Several potential exposure pathways, including surface water, sediment, and vapor intrusion could not be fully evaluated at this time due to a lack of data.

Next Steps

- EPA should continue investigating the nature and extent of contamination associated with the site.
- The site should not be used for other purposes without additional investigation and public health evaluation.
- ATSDR will evaluate, upon request, additional data that become available on the site.

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Appendix A. Explanation of Evaluation Process

Screening Process

In evaluating these data, ATSDR used comparison values (CVs) to determine which chemicals to examine more closely. CVs are health-based contaminant concentrations found in a specific media (air, soil, or water) and are used to screen contaminants for further evaluation. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of air, water, and soil that someone might inhale or ingest each day.

As health-based thresholds, CVs are set at a concentration below which no known or anticipated adverse human health effects are expected to occur. Different CVs are developed for cancer and noncancer health effects. Noncancer levels are based on valid toxicological studies for a chemical, with appropriate safety factors included, and the assumption that small children and adults are exposed every day. Cancer levels are based on a one-in-a-million excess cancer risk for exposure to contaminated soil or drinking contaminated water every day for 70 years. For chemicals for which both cancer and noncancer CVs exist, we use the lower level to be protective. Exceeding a CV does not mean that health effects will occur, just that more evaluation is needed.

CVs used in preparing this document are listed below:

Cancer Risk Evaluation Guides (CREGs) are estimated contaminant concentrations that would be expected to cause no more than one additional excess cancer in one million persons exposed over a lifetime. CREGs are calculated from EPA cancer slope factors.

Environmental Media Evaluation Guides (EMEGs) are estimated contaminant concentrations in a media where noncancer health effects are unlikely. EMEGs are derived from the ATSDR minimal risk level (MRL).

Reference Media Evaluation Guides (RMEGs) are estimated contaminant concentrations in a media where noncancer health effects are unlikely. RMEGs are derived from EPA's reference dose (RfD).

Regional Screening Levels (RSLs) are chemical-specific concentrations developed by EPA for individual contaminants in air, drinking water and soil that may warrant further investigation or site cleanup. RSLs are not cleanup standards.

Maximum Contaminant Levels (MCLs) are enforceable standards set by EPA for the highest level of a contaminant allowed in drinking water. MCLs are set as close to MCL goals (the level of a contaminant in drinking water below which there is no known or expected risk to health) as feasible using the best available treatment technology and taking cost into consideration.

Estimation of Exposure Dose

The next step is to take those contaminants present at levels above the CVs and further evaluate whether those chemicals may be a health hazard given the specific exposure situations at this site. For exposures occurring by inhalation, the air concentration of the contaminant can be

compared directly with health guideline air concentrations. For other pathways, we estimate the *exposure dose*, or the amount of contaminant that gets into a person's body. The exposure dose is typically expressed as milligrams of contaminant per kilogram of body weight of the person exposed, per day (mg/kg/day). This allows comparison with toxicological studies which express dose in the same units. Exposure that occurs through skin absorption may be converted to either an exposure dose or equivalent air concentration, depending on the other exposure routes being considered.

To do these estimates, ATSDR made assumptions about weight and other body characteristics of children and adults exposed, how they may be exposed, and how often they may be exposed to allow estimation of site- and pathway-specific exposure dose. The following sections detail the exposure assumptions and calculation of exposure dose for the pathways evaluated in this PHA.

Inhalation and Skin Absorption of TCE During Showering

One private well contaminated with TCE was used for all household uses except for drinking. Because inhalation and skin absorption of TCE during showering can be significant, ATSDR evaluated these exposures directly. ATSDR compared the estimated equivalent 24-hour TCE concentration with health guideline values. There are several steps in estimating the equivalent 24-hour air concentration, which will be discussed below.

ATSDR used a model developed by Andelman [17] to estimate the peak TCE concentration occurring in the bathroom as a result of showering. The equation is given below.

$$Peak\ Conc.\left(\frac{\mu g}{m^3}\right) = \frac{C_w\left(\frac{\mu g}{L}\right) \times k \times F_w\left(\frac{L}{min}\right) \times T_s\left(min\right)}{V_a\left(m^3\right)}$$

where

C_w	=	Concentration of the volatile compound in water, in $\mu g/L$
k	=	volatilization coefficient, unitless (default is 0.6)
F_w	=	Flow rate of water through showerhead, in L/min (default is 8 L/min)
T_s	=	Time of shower, in min (varies with age, found in [19])
V_a	=	Volume of air in shower in m^3 (default is 10 m^3)

For example, a 10-year-old takes a 15-minute shower in water containing 25 $\mu g/L$ TCE. The peak concentration of TCE in the bathroom is:

$$\begin{aligned} Peak\ Conc.\left(\frac{\mu g}{m^3}\right) &= \frac{25\frac{\mu g}{L} \times 0.6 \times 8\frac{L}{min} \times 15\ min}{10\ m^3} \\ &= 180\ \mu g/m^3 \end{aligned}$$

The peak air concentration will be breathed in during the shower and during any time stayed in the bathroom after the shower. ATSDR used shower stay times listed in [19]. The intake of contaminant due to inhalation is given by the following:

$$Intake_{Inhalation} = Peak\ Conc. \left(\frac{\mu g}{m^3} \right) \times IR_{st} \left(\frac{m^3}{min} \right) \times (T_s + T_b) (min),$$

where

IR_{st} = short term inhalation rate in m^3/min (varies with age, found in [19], assumed to reflect “light intensity” activity)

T_s = Time of shower and/or bath, in min (varies with age, found in [19])

T_b = Time in bathroom after shower/bath, in min (varies with age, found in [19])

For example, the inhalation intake for the 10-year-old in the previous example, who has an average short term inhalation rate of $0.011\ m^3/min$ and remains in the bathroom for 5 minutes after a 15-minute shower is:

$$Intake_{Inhalation}(\mu g) = 180 \frac{\mu g}{m^3} \times 0.011 \frac{m^3}{min} \times (5 + 15) min = 39.6\ \mu g\ TCE$$

Skin Uptake While Showering

Intake also occurs during showering through skin absorption. ATSDR estimated skin intake using the general methods of EPA’s Risk Assessment Guidance for Superfund, Part E [18]. The formula for skin intake of VOCs during a shower is:

$$Intake_{skin}(\mu g) = 2 \times FA \times K_p \left(\frac{cm}{hr} \right) \times C_w \left(\frac{\mu g}{L} \right) \times \left(\frac{1\ L}{1000\ cm^3} \right) \times SA (cm^2) \times \sqrt{\frac{6 \times \tau (hr) \times T_s (min)}{60 \frac{min}{hr} \times \pi}}$$

where

C_w = Concentration of the volatile compound in water, in $\mu g/L$

FA = Fraction Absorbed, assumed to be 1.

K_p = Permeability constant for compound of interest (for TCE, $0.012\ cm/hr$)

SA = total skin surface area in cm^2 (varies with age, found in [19])

τ = lag time, in hr (estimated at 0.5715 for TCE)

T_s = Time of shower plus time of bath, in min (varies with age, found in [19])

π = pi, 3.14

For the example above, a 10-year old taking a 15 minute shower in water containing $25\ \mu g/L$ TCE will have the following skin intake of TCE:

$$Intake_{skin} = 2 \times 1 \times 0.012 \frac{cm}{hr} \times 25 \frac{\mu g}{L} \times \frac{1\ L}{1000\ cm^3} \times 10,800\ cm^2 \times \sqrt{\frac{6 \times 0.5715\ hr \times 15\ min}{60 \frac{min}{hr} \times \pi}} = 3.4\ \mu g\ TCE$$

The total intake is the sum of inhalation and skin intake. To convert to an equivalent 24-hour air concentration, the total intake is divided by the daily average breathing rate.

$$24 - Hr \text{ Equivalent Conc. } \left(\frac{\mu g}{m^3} \right) = \frac{Intake (\mu g)}{IR_{day} \left(\frac{m^3}{day} \right)}$$

For the example above, a 10-year-old has an average inhalation rate of 12 m³/day. The 24-hour equivalent TCE concentration is:

$$24 - Hr \text{ Equivalent Conc.} = \frac{(39.6 + 3.4) \mu g}{12 \frac{m^3}{day}} = 3.6 \mu g/m^3$$

Table A1 below summarizes the assumptions used in calculating the 24-hour equivalent air concentrations from inhalation and dermal exposure during showering. We recognize that very young children likely take more baths than showers. Bathing would not likely result in exposures as great as showering because showering has a high flow rate and more volatilization of VOCs. Therefore, estimating exposures assuming showers is protective of bathing scenarios as well.

The 24-hour equivalent TCE concentrations calculated using the above equations and assumptions are summarized in the body of the text in Table 3.

Table A1. Exposure Assumptions for Estimating TCE Inhalation and Dermal Exposures From Showering – Private Well Pathway, Leeds Metal Site, Leeds, Maine

Group	Total Skin Surface Area (cm ²)	Short Term Inhalation Rate, m ³ /min	Long Term Inhalation Rate, m ³ /day	Time in Shower, min	Time in Bathroom after shower, min
Pregnant Women (16 Up To 45 Years Old)	18,400	0.0123	22	15	5
Children from Birth Up to 1 Year Old	3,992	0.0076	3.5	10*	5
Children from 1 Year Old Up To Age 2	5,300	0.012	8	10	5
Children from 2 Years Old Up To Age 3	6,100	0.012	8.9	10	5
Children from 3 Years Old Up To Age 6	7,600	0.011	10.1	12	5
Children from 6 Years Old Up To Age 11	10,800	0.011	12	15	5
Children from 11 Years Old Up To Age 16	15,900	0.011	15.2	15	5
Children from 16 Years Old Up To Age 21	18,400	0.012	16.3	15	5
Adults Greater Than 21 Years Old	19,683	0.012	15.1	15	5

*Infants do not shower but estimating exposure for showers will be protective of bathing because more volatilization occurs during showers.

Sources:

- Skin surface area obtained from Table 7.1 of [19], recommended values for total body surface area, for children (sexes combined) and adults by sex. (Weighted averages used to obtain body surface area for specific age ranges/groups listed in this table.)
- Short term inhalation rate obtained from Table 6-2 of [19], mean recommended short-term exposure values for inhalation (males and females combined), light intensity activity level.
- Long term inhalation rate obtained from Table 6-1 of [19], recommended long-term exposure values for inhalation (males and females combined). Rate for pregnant women estimated using Table 6-54 of [19] and professional judgment.
- Time in shower and bathroom obtained from Table 16-32 of [19], time spent (minutes) showering and in shower room immediately after showering (minutes/shower).

cm² = square centimeters m³/min = cubic meter per minute m³/day = cubic meter per day

Ingestion of Arsenic in Drinking Water

ATSDR estimated exposure doses for users of private well water assuming the average weights and drinking water ingestions listed in Table A2 below.

Table A2. Estimates for Body Weight and Drinking Water Ingestion – Private Well Pathway, Leeds Metal Site, Leeds, Maine

Group	Body Weight in Kilograms (Weight in Pounds)	Ingestion of Drinking Water in Liters per Day (Approximate 8-ounce glasses per day)	
		High-end	Average
Children from Birth Up to 1 Year Old	7.8 kg (17 lb)	1.1 L/day (5 glasses/day)	0.5 L/day (2 glasses/day)
Children from 1 Year Old up to Age 2	11.4 kg (lb)	0.9 L/day (4 glasses/day)	0.4 L/day (2 glasses/day)
Children from 2 Years Old Up To Age 3	13.8 kg (lb)	0.9 L/day (4 glasses/day)	0.5 L/day (2 glasses/day)
Children from 3 Years Old Up To Age 6	18.6 kg (lb)	1.0 L/day (4 glasses/day)	0.6 L/day (2.5 glasses/day)
Children from 6 Years Old Up To Age 11	31.8 kg (lb)	1.4 L/day (6 glasses/day)	0.5 L/day (2 glasses/day)
Children from 11 Years Old Up To Age 16	56.8 kg (lb)	2 L/day (8 glasses/day)	0.6 L/day (2.5 glasses/day)
Children from 16 Years Old Up To Age 21	71.6 kg (158 lb)	2.5 L/day (11 glasses/day)	0.8 L/day (3.5 glasses/day)
Adults Greater Than 21 Years Old	80 kg (176 lb)	3.0 L/day (13 glasses/day)	1.2 L/day (5 glasses/day)

Sources:
 - Weight for children and adults obtained from Table 8-1 of [19], recommended values for body weight (males and females combined). (Weighted averages used to obtain body weight for specific age ranges listed in this table.)
 - Ingestion rates obtained from Tables 3-1 and 3-3 of [19], consumers-only ingestion of drinking water, High-end=95th percentile, Average=mean. (Weighted averages used to obtain ingestion for specific age ranges listed in this table.)
 kg = kilogram lb = pound L/day = liters per day

To calculate the exposure dose resulting from drinking water containing a certain concentration of a chemical, the concentration is used with exposure assumptions as listed in Tables A1 and A2. For example, a child less than one year old (average weight 7.8 kg) drinking 1.1 liters of water (about 4 8-ounce glasses) containing the highest concentration of arsenic (28.9 µg/L or 0.0289 mg/L) every day will receive a dose of:

$$Dose = \frac{0.0289 \frac{mg}{L} \times 1.1 \frac{L}{day}}{7.8 kg} = 0.004 mg / kg / day$$

The arsenic doses calculated for the drinking water pathway for this site are summarized in the body of the text in Table 6.

On-Site Soil Exposure

Incidental Ingestion of Soil

ATSDR estimated soil exposure doses for trespassers on the site assuming the average weights and incidental ingestion listed in Table A3 below. Because the site is relatively remote, ATSDR considered only older children and adults as likely to enter the site regularly. Table A3 also lists assumptions for how often and for how many years trespassers would go on site. These assumptions reduce the long-term average dose as compared to an exposure that would occur continuously.

For calculating exposure doses, ATSDR assumed that a normal trespasser would be exposed only to surface soil. For each contaminant, the concentration was assumed to be the 95th percentile of all the surface soil results (that is, the concentration at which 95% of the results were lower than that value). Adults digging on site for scrap metal were assumed to be exposed to contaminants in both surface and subsurface soil. For these dose calculations, ATSDR assumed the digger would be exposed to the 95th percentile of all the surface soil and subsurface soil results.

The exposure assumptions are conservative, because the average contaminant concentration a person would contact over time would probably be lower than the 95th percentile value. Also, we assumed that a trespasser receives the entire days' soil ingestion from the site, when it is unlikely that the trespasser will spend more than a few hours on the site on any one day.

Table A3. Estimates for Body Weight and Incidental Soil Ingestion – Trespasser / Digger Scenario, Leeds Metal Site, Leeds, Maine

Group	Body Weight in Kilograms (Weight in Pounds)	Incidental Ingestion of Soil in milligrams per day		Frequency of Exposure in Days per Year [‡]	Duration of Exposure in Years	Assumed Exposure Concentration
		Average	High-End			
Children from 11 Years Old Up To Age 16	56.8 kg (lb)	100 mg/day	200 mg/day	72	5	95 th Percentile of Surface Soil Concentrations
Children from 16 Years Old Up To Age 21	71.6 kg (158 lb)	100 mg/day	200 mg/day	72	5	95 th Percentile of Surface Soil Concentrations
Adults Greater Than 21 Years Old	80 kg (176 lb)	50 mg/day	100 mg/day	72	10	95 th Percentile of Surface Soil Concentrations
Adults Digging for Scrap Metal	80 kg (176 lb)	330 mg/day		72	10	95 th Percentile of Surface and Subsurface Soil Concentrations

Sources:

- Body weights for children and adults obtained from Table 8-1 of [19], recommended values for body weight (males and females combined). (Weighted averages used to obtain body weight for specific age ranges listed in this table.)
 - Incidental ingestion obtained from Table 5-1 of [19], recommended values for daily soil + dust ingestion, High-end=upper percentile, Average=central tendency. Value for adults digging scrap metal obtained from Exhibit 1-2 of [43], for workers with high soil exposure.
 - Frequency of exposure based on professional judgment (average of two day on site per week during non-winter months, approximately 72 days a year).
- kg = kilogram lb = pound mg/day = milligrams per day

To calculate the exposure dose resulting from incidental ingestion of soil containing a certain concentration of a chemical, the concentration is used with exposure assumptions as listed in Table A3. For example, a young teenage trespasser who ingests a “high-end” amount of soil containing 167 milligrams of arsenic per kilogram of soil (mg/kg) will receive an average dose of:

$$\begin{aligned}
 \text{Dose} &= \frac{167 \text{ mg arsenic}}{\text{kg soil}} \times \frac{200 \text{ mg soil}}{\text{day}} \times \frac{1 \text{ kg soil}}{10^6 \text{ mg soil}} \times \frac{72 \text{ days}}{365 \text{ days}} \times \frac{1}{56.8 \text{ kg}} \\
 &= 1.2 \times 10^{-4} \text{ mg/kg/day}
 \end{aligned}$$

Dermal Exposure to On-Site Soil

ATSDR estimated doses resulting from dermal exposure to on-site soil using the assumptions listed in Table A4 below. The assumed exposure concentration was the same used in the incidental exposure estimates (95th percentile of surface soil concentrations for trespasser scenarios and 95th percentile of surface and subsurface concentrations for digging scenarios).

Table A4. Estimates for Body Weight and Dermal Exposure – Trespasser / Digger Scenario, Leeds Metal Site, Leeds, Maine

Group	Body Weight in kg (lb)	Incidental Ingestion of Soil in mg/day		Skin Surface Area Available for Contact in cm ²	Mean soil adherence to skin in mg/cm ²
		High-end	Average		
Children from 11 Years Old Up To Age 16	56.8 kg (125 lb)	200	100	3,680	0.11
Children from 16 Years Old Up To Age 21	71.6 kg (158 lb)	200	100	3,836	0.11
Adults Greater Than 21 Years Old	80 kg (176 lb)	100	50	4,262	0.1336
Adults Digging for Scrap Metal	80 kg (176 lb)	330		4,262	0.1595

Sources:

- Body weights for children and adults obtained from Table 8-1 of [19], recommended values for body weight (males and females combined). (Weighted averages used to obtain body weight for specific age ranges listed in this table.)
- Incidental ingestion obtained from Table 5-1 of [19], recommended values for daily soil + dust ingestion, High-end=upper percentile, Average=central tendency. Value for adults digging scrap metal obtained from Exhibit 1-2 of [43], for workers with high soil exposure.
- Skin surface available for contact calculated from age-specific mean skin surface area information listed in Tables 7-1 and 7-2 of [19], assuming soil contacts the face, forearms, and lower legs. The face is assumed to represent 1/3 of the surface area of the head; forearms are assumed to represent 45% of the surface area of the arms; and lower legs are assumed to represent 40% of the surface area of the legs.
- Mean soil adherence values obtained from Table 7-4 of [19], recommended values for mean solids adherence to skin. Used factors for legs as a conservative assumption for the adherence for the entire body. Used values for “outdoor sports activities” to represent trespasser exposures, and used “activities with soil” values to represent digging for scrap metal.

kg = kilogram lb = pound mg/day = milligrams per day cm² = square centimeter
 mg/cm² = milligrams per square centimeter

To estimate dermal exposure, one uses a contaminant-specific dermal absorption factor as listed in Table A5 below.

Table A5. Dermal Absorption Factors Used for Trespasser Scenario, Leeds Metal Site, Leeds, Maine

Contaminant	Dermal Absorption Factor
Arsenic	0.03
Cadmium	0.001
Chromium	0.01
Copper	0.01
Lead	0.01
Aroclor-1242	0.14
Aroclor-1248	0.14
Aroclor-1254	0.14
Aroclor-1260	0.14
Aroclor-1268	0.14
Sources:	
- Exhibit 3-4 of [18], recommended dermal absorption fraction from soil (arsenic, cadmium, and Aroclors)	
- [44] (chromium, copper, and lead treated as “other metals”)	

Calculation of dermal exposure follows the example given below for an 11-to-16-year old contacting arsenic at 167 mg/kg in soil. ATSDR assumed one dermal exposure “event” per day on the site:

$$\begin{aligned}
 \text{Dermal dose} &= \frac{167 \frac{\text{mg Arsenic}}{\text{kg soil}} \times \frac{10^{-6} \text{ kg soil}}{\text{mg soil}} \times 3,680 \text{ cm}^2 \times 0.11 \frac{\text{mg soil}}{\text{cm}^2} \times \frac{72 \text{ days}}{365 \text{ days}} \times 0.03}{56.8 \text{ kg}} = \\
 &= 0.0000070 \text{ mg/kg/day} = 7.0 \times 10^{-6} \text{ mg arsenic/kg/day}
 \end{aligned}$$

Inhalation Exposure to On-Site Soil

ATSDR estimated doses resulting from inhalation exposure to on-site soil during ATV riding using the assumptions listed in Table A6 below. The assumed exposure concentration was the 95th percentile of surface soil concentrations.

Table A6. Estimates for Body Weight and Inhalation Exposure – Trespasser / Digger Scenario, Leeds Metal Site, Leeds, Maine

Group	Body Weight in kg (lb)	Particle Emission Factor in kg soil /m ³ of air	Breathing Rate in m ³ /min	Duration of Inhalation in min/day	Frequency of Exposure in days/year
Children from 11 Years Old Up To Age 16	56.8 kg (125 lb)	1.18×10 ⁻⁶ kg/m ³	0.0508 m ³ /min	180 min/day	72 days/yr
Children from 16 Years Old Up To Age 21	71.6 kg (158 lb)	1.18×10 ⁻⁶ kg/m ³	0.0532 m ³ /min	180 min/day	72 days/yr
Adults Greater Than 21 Years Old	80 kg (176 lb)	1.18×10 ⁻⁶ kg/m ³	0.0552 m ³ /min	180 min/day	72 days/yr

Sources:
 - Body weights for children and adults obtained from Table 8-1 of [19], recommended values for body weight (males and females combined). (Weighted averages used to obtain body weight for specific age ranges listed in this table.)
 - Particle emission factor derived by EPA for ATV riding based on field data [45].
 - Breathing rate from Table 6-17 of [19], mean ventilation rates for males, unadjusted for body weight, for high intensity activities. For adults greater than 21 years old, used average of mean ventilation rates from ages 21 up to age 51. (Note: male ventilation rates are higher so will be protective of female exposure.)
 kg = kilogram lb = pound kg/m³ = kilogram per cubic meter m³/min = cubic meters per minute

Calculation of inhalation exposure dose follows the example given below for an 11-to-16-year old riding an ATV and stirring up soil contacting arsenic at 167 mg/kg. The average inhalation exposure using the assumptions given above is:

$$\text{Inhalation dose} = \frac{167 \frac{\text{mg Arsenic}}{\text{kg soil}} \times \frac{1.18 \times 10^{-6} \text{ kg soil}}{\text{m}^3 \text{ air}} \times 0.0508 \frac{\text{m}^3 \text{ air}}{\text{min}} \times 180 \frac{\text{min}}{\text{day}} \times \frac{72 \text{ days}}{365 \text{ days}}}{56.8 \text{ kg}} = 0.0000063 \text{ mg/kg/day} = 6.3 \times 10^{-6} \text{ mg arsenic/kg/day}$$

Total Exposure Dose – On-Site Soil

For the examples given above, the total exposure dose to arsenic for a young teenager is the sum of all three doses:

$$\begin{aligned} \text{Total dose} &= 1.2 \times 10^{-4} \text{ (ingestion)} + 7.0 \times 10^{-6} \text{ mg/kg/day (dermal)} + 6.3 \times 10^{-6} \text{ (inhalation)} = \\ &= 1.3 \times 10^{-4} \text{ mg arsenic /kg/day.} \end{aligned}$$

Exposure dose ranges for all the scenarios evaluated are tabulated within the body of the text in Table 9.

Evaluating Noncancer Health Effects

The calculated exposure doses are then compared to an appropriate health guideline for that chemical. Health guideline values are considered safe doses; that is, health effects are unlikely below this level. The health guideline value is based on valid toxicological studies for a chemical, with appropriate safety factors built in to account for human variation, animal-to-human differences, and/or the use of the lowest study doses that resulted in harmful health effects (rather than the highest dose that did not result in harmful health effects). For noncancer health effects, the following health guideline values are used.

Minimal Risk Level (MRLs) – Developed by ATSDR

An MRL is an estimate of daily human exposure – by a specified route and length of time – to a dose of chemical that is likely to be without a measurable risk of adverse, noncancerous effects. An MRL should not be used as a predictor of adverse health effects. A list of MRLs can be found at <http://www.atsdr.cdc.gov/mrls/index.html>.

Reference Concentration (RfC) – Developed by EPA

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers both toxic effects of the respiratory system (portal-of-entry) and effects peripheral to the respiratory system (extrarespiratory effects). RfCs can be found at <http://www.epa.gov/iris>.

Reference Dose (RfD) – Developed by EPA

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. RfDs can be found at <http://www.epa.gov/iris>.

Maximum Contaminant Level (MCL) – Developed by EPA

The MCL is the highest level of a contaminant that is allowed by the EPA in public drinking water systems. MCLs are enforceable standards set as close as feasibly possible to levels below which there is no known or expected risk to health, using the best available treatment technology and taking cost into consideration.

If the estimated exposure dose for a chemical is less than the health guideline value, then the exposure is unlikely to cause a noncancer health effect in that specific situation. If the exposure dose for a chemical is greater than the health guideline, then the exposure dose is compared to known toxicological values for that chemical and is discussed in more detail in the public health assessment. These toxicological values are doses derived from human and animal studies that are summarized in the ATSDR *Toxicological Profiles* and in current scientific literature. A direct comparison of site-specific exposure and doses to study-derived exposures and doses that cause adverse health effects is the basis for deciding whether health effects are likely or not.

Evaluating Cancer Health Effects

The estimated risk of developing cancer resulting from exposure to the contaminants was calculated by multiplying the site-specific estimated exposure dose by an appropriate cancer

slope factor or inhalation unit risk (EPA values can be found at <http://www.epa.gov/iris>). The result estimates the increase in risk of developing cancer after a lifetime of continuous exposure to the contaminant.

If a substance causes cancer by a mutagenic mode of action, there is a greater risk for exposures that occur in early life. A current list of substances EPA considers mutagenic can be found at <http://www.epa.gov/oswer/riskassessment/sghandbook/chemicals.htm>. For these substances, age-dependent adjustment factors (ADAFs) are applied to the risks estimated as follows: An ADAF of 10 is applied for exposures taking place from birth up to 2 years old, and an ADAF of 3 is applied for exposures taking place from age 2 up to age 16. No adjustment is applied for exposures at age 16 or above [31].

The actual increased risk of cancer may be lower than the calculated number, which gives an estimated risk of excess cancer. The methods used to calculate cancer slope factors assume that high-dose animal data can be used to estimate the risk for low dose exposures in humans. The methods also assume that no safe level exists for exposure. Little experimental evidence exists to confirm or refute those two assumptions. Lastly, most methods compute the upper 95th percent confidence limit for the risk. The actual cancer risk can be lower, perhaps by several orders of magnitude [46].

Because of uncertainties involved in estimating cancer risk, ATSDR employs a weight-of-evidence approach in evaluating all relevant data [47]. Therefore, the increased risk of cancer is described in words (qualitatively) rather than giving a numerical risk estimate only. Numerical risk estimates must be considered in the context of the variables and assumptions involved in their derivation and in the broader context of biomedical opinion, host factors, and actual exposure conditions. The actual parameters of environmental exposures must be given careful consideration in evaluating the assumptions and variables relating to both toxicity and exposure.

Appendix B. Exposure Pathways for the Leeds Metal Site

Pathway Name	Environmental Media and Transport Mechanisms	Point of Exposure	Route of Exposure	Exposure Population	Time	Notes	Complete?
Private Well Water	Infiltration to groundwater	Drinking water taps supplied by private wells	Ingestion, inhalation, dermal exposure	Residents and workers drinking and showering in water from private wells near the site	Past, potential future	Population may include young children	Y – for untreated wells
Soil or Source	Auto fluff piles on site; dispersed to soil by wind or water erosion	On Site	Incidental ingestion, dermal exposure	Trespassers on the site	Past, present, future	Population may include teenagers; exposure to subsurface and surface soil	Y
Surface water	Ground water and surface water drainage through fluff piles into creeks	Creeks On Site	Incidental ingestion, dermal exposure	Trespassers on the site	Past, present, future	Population may include teenagers	Y
Sediment	Ground water and surface water drainage through fluff piles into creeks	Creeks On Site	Incidental ingestion, dermal exposure	Trespassers on the site	Past, present, future	Population may include teenagers	Y

Appendix C. Glossary of Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency in Atlanta, Georgia, with 10 regional offices in the United States. ATSDR serves the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases from toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces laws to protect the environment and human health. This glossary defines words used by ATSDR in this public health assessment. It is not a complete dictionary of environmental health terms. For additional questions or comments, call 1-800-CDC-INFO.

Acute exposure

Contact with a substance that occurs once or for only a short time; ATSDR defines acute exposures as occurring for periods of 14 days or less. [compare with intermediate duration exposure and chronic exposure].

Adverse health effect

A change in body function or cell structure that might lead to disease or health problems

Age Dependent Adjustment Factor (ADAF)

A factor used to account for age-related differences in toxicity of cancer-causing chemicals which allows integration of varying toxicity and exposures over relevant age intervals.

Aquifer

A layer of underground porous rock, gravel, sand, or silt containing enough groundwater to supply springs or wells.

Aroclor

A commercial name for various mixtures of polychlorinated biphenyls (PCBs) that were produced from about 1930-1979. The Aroclor designation is followed by a number indicating the chlorine content of the mixture; e.g., Aroclor 1254 contains 54% chlorine by weight.

Association

In statistics, a relationship between two measured quantities that means changes in one quantity can predict changes in the other. The relationship is not necessarily causal; that is, changes in one quantity do not necessarily cause the changes observed in the other quantity..

Auto fluff

Material remaining after automobiles are shredded and recyclable metals are removed. Also known as automotive shredder residue (ASR), this material is made up of fibrous textiles, polyurethane foams, plastics, rubber, and a wide variety of light metals and may also contain residual engine oils and coolants as well as contaminating dirt and stone.

Basal cell carcinoma

A type of skin cancer that arises in cells in the deepest layer of the skin, the basal layer. Basal carcinoma, the most common form of skin cancer, very rarely spreads to other parts of the body.

Bedrock

Solid rock underlying unconsolidated (loose) materials such as soil, sand, clay, or gravel.

Cancer

Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk

A predicted risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen

A substance that causes cancer.

Central nervous system

The part of the nervous system that consists of the brain and the spinal cord.

Chronic exposure

Contact with a substance that occurs over a long time; ATSDR defines chronic exposures as occurring for periods of one year or more. [compare with acute exposure and intermediate duration exposure]

Comparison value (CV)

Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway

[see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)

CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. The Superfund Amendments and Reauthorization Act (SARA) later amended this law.

Concentration

The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Confining layer

Underground layer of low-permeability geological material that does not allow significant movement of water. Aquifers may be present on top of confining layers or between two confining layers.

Congenital

Of or pertaining to a condition present at birth, whether inherited or caused by environmental influences.

Contaminant

A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Demographic

Pertaining to statistical characteristics of human populations.

Dermal

Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact

Contact with (touching) the skin [see route of exposure].

Detection limit

The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Dose

The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Environmental media

Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Epidemiologic study

A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Epidemiology

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

Exposure

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

Exposure pathway

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

Groundwater

Water beneath the earth’s surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Ingestion

The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation

The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intelligence Quotient (IQ)

A score derived from one of several standardized tests designed to assess intelligence. An IQ of 100 represents the average score within a particular age group.

Intermediate duration exposure

Contact with a substance that occurs for weeks or months; ATSDR defines intermediate duration exposures as occurring over periods greater than 14 days but less than 365 days (one year) [compare with acute exposure and chronic exposure].

Meta-analysis

A method of analysis in which results of multiple research experiments or studies of the same subject are combined and contrasted with the goal of identifying overall patterns and trends and improving statistical strength of findings.

Metabolism

The conversion or breakdown of a substance from one form to another by a living organism.

Metabolic byproduct

Any product of metabolism.

Minimal risk level (MRL)

An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

National Priorities List (NPL, also known as the Superfund list)

EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

Percentile

The value of a variable below which a certain percent of observations fall. For example, 95 out of 100 observations are expected to fall below the 95th percentile.

Physical hazard

Property or circumstance in the physical (as opposed to chemical) environment that could cause bodily harm or injury to people nearby.

Point of departure

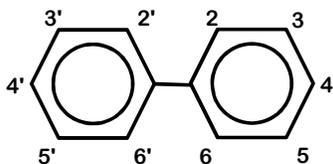
In risk assessment, the dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound on dose for an estimated incidence or change in response level and may be derived from modeling or from toxicological effect levels.

Point of exposure

The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Polychlorinated Biphenyls (PCBs)

A group of synthetic organic chemicals formerly used for insulating purposes but now banned due to toxicity. The base of every PCB is the biphenyl molecule, two benzene rings joined together. Chlorine atoms can substitute for hydrogen at any of the 10 numbered carbon atom locations shown. The 209 possible combinations that can be formed are known as PCB congeners.



Biphenyl Chemical Structure

Population

A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Prevention

Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public health assessment (PHA)

An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health.

Public health surveillance

The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Reference concentration (RfC)

An EPA estimate, with uncertainty or safety factors built in, of a continuous inhalation exposure of a chemical that is unlikely to cause harmful noncancer effects during a lifetime.

Reference dose (RfD)

An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Reverse Osmosis

A process of purifying water or other liquids by forcing them through a semipermeable membrane that removes many types of large molecules and ions.

Risk

The probability that something will cause injury or harm.

Route of exposure

The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Sample

A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size

The number of units chosen from a population or an environment.

Source of contamination

The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Squamous cell carcinoma

A type of skin cancer that arises in flat cells just under the outermost layer of skin. Squamous cell carcinoma, the second most common form of skin cancer, rarely spreads to other parts of the body.

Substance

A chemical.

Superfund [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)]

Superfund Amendments and Reauthorization Act (SARA)

In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Toxicological profile

An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology

The study of the harmful effects of substances on humans or animals.

Transport mechanism

Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

Vapor Intrusion

Vapor intrusion is a way that volatile chemicals in the ground or groundwater can get into indoor air. Volatile gases, or vapors, can move up from the groundwater into pockets of air underground. Then the vapors can travel through the ground. Vapors can enter homes through cracks in foundations, dirt floors, sump pump pits, utility conduits, floor drains, and damaged or poorly constructed plumbing. Once vapors are in the home, they may not be able to leave if the home is airtight and does not get fresh air. In some cases, the vapors can build up to harmful levels inside a home.

Volatile organic compounds (VOCs)

Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.