EnvironmentalSafetyHealthGeotechnical

O'Reilly, Talbot & Okun
[A S S O C I A T E S]
ENGINEERING

69 Milk Street
Suite 210
Westborough, MA 01581
Tel 508 366 6409
Fax 508 366 9826
www.oto-env.com

October 19, 2015
File No: 5193-01-01

Prepared for:
Alliance Environmental Group, Inc.
100 Jefferson Boulevard, Suite 200
Warwick, RI 02888

METHOD 3 RISK CHARACTERIZATION
Former Universal Steel & Trading Company
297 – 305 Bridge Street
Salem, Massachusetts 01970
MassDEP RTN 3-11726

Prepared by:
O'Reilly, Talbot & Okun Associates, Inc.
69 Milk Street, Suite 210
Westborough, Massachusetts 01581

O'Reilly, Talbot & Okun [ASSOCIATES] ENGINEERING

TABLE OF CONTENTS

TABLE OF CONTENTS (Cont'd.)

1.0 INTRODUCTION

This Method 3 Risk Characterization (RC) has been prepared by O'Reilly, Talbot & Okun Associates, Inc. (OTO) to assess whether a release of oil and/or hazardous material (OHM) at the former Universal Steel & Trading Company property located at 297 - 305 Bridge Street in Salem, Massachusetts (the "Site") represents a Condition of No Significant Risk within the meaning of the Massachusetts Contingency Plan (MCP: 310 CMR 40.0990).

This Method 3 Risk Characterization was based on Site information and analytical data collected at the Site and provided to OTO by Alliance Environmental Group, Inc. (AEG). These Site information and analytical data were summarized in the Weston & Sampson report *Phase III Remedial Action Plan (RAP) and Permanent Solution Statement with Conditions Statement* (PSS) dated January 2015; and the AEG *Release Abatement Measure (RAM) Plan* dated October 2015. The PSS included an Activity and Use Limitation (AUL) which was applied to the Site in December 2014. AEG prepared the RAM Plan on behalf of F.W. Webb Company to facilitate proposed construction activities at the Site.

OTO has not conducted independent testing or Site characterization activities and relies on the Weston & Sampson and AEG Reports to provide an adequate characterization of the nature and extent of Site contamination, as defined in the MCP.

1.1 Site History and Investigations

In October 1994, the Massachusetts Department of Environmental Protection (MassDEP) assigned Release Tracking Number (RTN) 3-11726 to the Site following notification of a release to soil and groundwater of polychlorinated biphenyls (PCBs), metals, and petroleum. The release was identified during soil and groundwater assessment activities completed at the Site from November 1993 to July 1994. The source of contamination was attributed to the former metals recycling and reclamation activities conducted by Universal Steel & Trading Company at the Site.

Since the release was discovered, several subsurface investigations and preliminary response actions have been conducted at the Site. In 2011, Weston & Sampson completed a Phase II Comprehensive Site Assessment (CSA) to evaluate the current nature and extent of contamination at the Site, and a site-specific Method 3 RC to evaluated risk to human health, safety, public welfare, and the environment. PCB soil impacts at depths of up to 12 feet below ground surface (bgs) were identified across the Site. In general, the majority of PCB contamination was located in the top 3 feet of soil, while deeper impacts (i.e., greater than 4 feet bgs) were relatively limited. The 2011 Method 3 RC indicated that a Condition of No Significant Risk (NSR) did not exist due to the potential exposure to PCBs in soil.

Based on the findings of the Phase II CSA and Method 3 RC, Weston & Sampson discussed and evaluated several remedial options for the Site under the direction of the

MassDEP and the United States Environmental Protection Agency (EPA) from June to October 2011. Subsequently, a multi-agency team consisting of MassDEP, EPA, the City of Salem and MassDevelopment agreed to fund and implement a risk-based cleanup of the Site. The risk-based cleanup approved by MassDEP and EPA included the excavation and removal of the top 1 foot of soil and concrete across the Site and off-site (92 and 102 Federal Street) and the select removal of deeper PCB-impacted soils with concentrations greater than 50 parts per million (ppm). At the conclusion of removal actions, the construction of a pavement cap and implementation of an Activity and Use Limitation (AUL) was proposed to mitigate future direct contact exposure to residual PCB impacts at the Site.

The excavation and removal of PCB-impacted materials was completed as a Removal Action under EPA's Emergency Response and Removal Program (ERRP). The Removal Action was initiated in December 2012 and was completed in September 2013. In total, approximately 6,380 cubic yards of PCB-impacted soil and concrete were excavated and disposed off-site as part of the Removal Action, and 81 post-excavation confirmatory soil samples were collected to verify the limits of remediation. EPA subsequently backfilled and compacted the Site with gravel, and Manter Construction installed a paved parking lot and storm water control features (i.e., sediment forebays). The parking lot cap construction was completed in October 2013.

Weston & Sampson utilized the 2013 EPA post-excavation soil analytical results and historical data sets for contaminants of concern (COCs) remaining below the final EPA excavation depths to perform an updated Method 3 RC for the Site. The updated Method 3 RC indicated that the MassDEP and EPA risk-based cleanup achieved a Condition of NSR for current and future Site use with the construction of a pavement cap and implementation of an AUL to mitigate and control the future direct exposure to residual PCB impacts at the Site. Therefore, the requirements for a Permanent Solution with Conditions Statement had been met.

In general, the AUL was placed on the entire parcel to restrict future residential use and other Site activities that would result in greater exposure to residual contaminated soil at the Site. The AUL allows for industrial and commercial uses; and landscaping above the geotextile liner at 1.5 feet bgs; and underground utility and/or construction activities below the geotextile liner at depths greater than 1.5 feet bgs or more provided that a Soil Management Plan (SMP) and Health and Safety Plan (HASP) is implemented. The AUL also specifies maintenance of the existing asphalt, and no disturbance and direct contact with soil under the geotextile liner, except for underground utility and/or construction activities as described previously.

The purpose of the subject Method 3 RC is to evaluate that the proposed developed use of the Site as a retail plumbing and heating supply company are consistent with a Condition of NSR with the AUL. The subject Method 3 RC is based on the 2013 post-excavation soil analytical results and the proposed condition of the Site primarily capped by commercial buildings and pavement, with small landscaped islands, vegetative buffers, and sediment forebays along the eastern and southern perimeters of the Site.

Three (3) Risk Characterization methods, which vary in detail and circumstances of use, have been developed under the MCP to evaluate MCP sites, as described in 310 CMR 40.0941(3) and 40.0942. These three (3) methods provide equivalent levels of protection to human health, public welfare, and the environment. A Method 3 approach was considered applicable for the Site because Site-specific methodologies (e.g., exposure assumptions concerning Site use) have been used. As defined in 310 CMR 40.0990, the Method 3 Risk Characterization evaluated the risks to human health, public welfare, safety, and the environment for all current and reasonably foreseeable future site activities and uses.

This Method 3 Risk Characterization has been completed in accordance with the MCP, 310 CMR 40.0900, and applicable MassDEP guidance (MassDEP, 1995 through 2015). Human health risks for the Site are assessed in Section 2.0 of this report. Risk of harm to public welfare, including comparisons to Upper Concentration Limits (UCLs), is presented in Section 3.0. Characterization of risk of harm to safety is described in Section 4.0. The environmental risk characterization is in Section 5.0. Section 6.0 presents conclusions regarding the overall significance of Site risk.

1.2 Data Used in the Method 3 Risk Characterization

The objective of the Method 3 Risk Characterization is to determine if concentrations of OHM at the Site represent a Condition of No Significant Risk under current and foreseeable future Site Conditions based on a commercial use of the Site and implementation of the existing AUL.

The analytical data representative of current post-excavation conditions for the subject Site were evaluated for use in the Method 3 Risk Characterization. These data were presented in the Weston & Sampson PSS. Following the excavation and removal of the PCB-impacted materials, confirmatory soil samples were collected to verify the limits of the excavation. The confirmatory samples were collected across the entire Site using an approximately 25-foot square grid, with one confirmatory soil sample collected from the approximate center of each grid cell. A total of 81 samples were collected, including 79 samples on-Site and one each from of the two off-Site remediated properties (92 and 102 Federal Street). Sample collection ranged from 1.5 to 4 feet bgs on-Site and 1 foot bgs off-Site; at the bottom of the excavation.

Each of the confirmatory soil sample concentrations for PCBs were less than 50 mg/kg, with the exception of the samples collected from the confirmatory grids B-150, B-175, B-200, C-125, and E-150. These five samples were each collected at depths of 3.5 feet bgs. The concentrations of PCBs in confirmatory samples from 92 and 102 Federal Street were below the MCP's IH criteria of 10 mg/kg.

In general, groundwater was not significantly impacted by soil contamination at the Site. Groundwater was not impacted with PCBs, and during groundwater sampling conducted in conjunction with the Phase II CSA in 2011, exceedances of applicable MCP Method 1

standards were limited to cadmium and chlorobenzene in one groundwater monitoring well exceeding the Method 1 GW-3 and GW-2 standards, respectively.

2.0 HUMAN HEALTH RISK ASSESSMENT

2.1 Current and Reasonably Foreseeable Future Site Use

Site Location/Description: The Site is approximately 1.2 acres in size and was historically used for metal recycling and reclamation activities including processing and sorting of scrap metals and demolition debris, dismantling and processing of transformers, and stockpiling of automotive batteries.

Historically, the Site was developed and contained several structures, including a two-story warehouse building, two large concrete pads, and several ancillary features (i.e., truck scale, and paved loading areas). The historical warehouse building was demolished in 2012. The remaining Site features were demolished and removed as part of the Site remediation completed in 2013.

Currently, the Site is used as a temporary parking lot for a nearby Massachusetts Bay Transportation Authority (MBTA) train station. The parking lot was opened by the City of Salem in October 2013 following Site remediation. The parking lot consists of a large central paved area with small landscaped islands, and vegetative buffers along the perimeter of the Site. In addition, two sediment forebays are also located at each corner of the Site along Bridge Street.

The proposed use of the Site includes the construction of a 15,875 square foot (ft^2) warehouse and 10,225 ft^2 self-service retail store, for a total building footprint of approximately 26,100 ft^2. Parking spaces will occupy the majority of the remainder of the Site. The existing areas of small landscaped islands, vegetative buffers, and sediment forebays would remain.

Utilities: The Site is served by municipal water and sewer. Water, sewer, and natural gas lines enter the Site from Bridge Street. Drainage lines will be located around the eastern and southern perimeters of the proposed building and installed at depths between 3 and 4 feet bgs and primarily located within a clean fill corridor.

Electrical utilities are located overhead.

Surrounding Areas: The Site is located in an area of mixed commercial and residential use. The Site is abutted by Bridge Street to the north, across which is an unpaved parking area with railroad tracks beyond. A F.W. Webb plumbing and heating supply store is located directly adjacent to the Site the east. Residential properties border the Site to the south. Beckford Street is located to the west of the Site. A used automobile sales lot is located further to the west beyond Beckford Street.

No sensitive institutions such as schools, hospitals, daycare centers and long-term

healthcare facilities are located within a 500-foot radius of the Site.

According to the Site Scoring Map, no Areas of Critical Environmental Concern (ACECs), certified vernal pools, wetlands, or sensitive habitat areas as mapped by the Natural Heritage and Endangered Species Program (NHESP) are located within a one-half mile radius of the Site. Several areas of Protected Open Space are located within 500 feet of the Site to the north, south, east, and west.

Foreseeable Future Use: The Site is expected to remain as commercial, and the surrounding areas a mix of residential and commercial in the foreseeable future.

2.1.1 Soil and Groundwater Categories

Categories for soil and groundwater have been developed by the MassDEP to facilitate the characterization of risk at MCP sites. The identification of applicable groundwater and soil categories at the Site has been performed in accordance with 310 CMR 40.0993(2).

Soil Category

Identification of the applicable soil category requires an assessment of three (3) factors identified in Section 40.0930 of the MCP. These are (i) accessibility, (ii) frequency of use, and (iii) intensity of use. Each of these factors must be assessed for the current use scenario and for a reasonably foreseeable future use scenario.

Post-excavation soil analytical data indicates that residual contaminated soils are located at depths of approximately 1.5 to 15 feet bgs. The majority of impacted soil is currently located under pavement and under the proposed use will be located under Site buildings and pavement. Commercial and office workers may be present at a high frequency, while landscapers would be present at a low frequency.

The soil located under pavement are categorized in the MCP as “potentially accessible” to exposure. The soil located under the Site building are categorized in the MCP as “isolated” from exposure. Small landscaped areas and vegetative buffers are located along the eastern and southern perimeters of the Site. The soil in these areas would be generally categorized in the MCP as “accessible”. However, impacted soil are located at 1.5 feet bgs and greater and under the geotextile liner and are not readily “accessible”. The AUL restricts landscaping activities to the upper 1.5 feet of soil located above the geotextile liner. Therefore, there is no potential exposure to impacted soil for commercial and office workers, and landscapers.

Potential receptors of the public (as patrons and trespassers), may be present at the Site at a high frequency. Members of the public include children and adults. The intensity of use for soil by these receptors will be low under most current and future Site conditions as no soil with impacts will be “accessible” as soil are located under liner and greater than 1.5 feet bgs.

Construction and utility work may occur at the Site. The frequency of this activity was considered low, and the intensity of use was considered high (i.e., short-term excavation activities occurring at infrequent and irregular intervals). As specified in the AUL, these activities are only allowed below the geotextile liner at depths greater than 1.5 feet or more if these activities are conducted in accordance with a SMP and HASP.

The AUL restricts uses of the Site for residences, schools, and day care centers. Therefore, these receptors will not be present at the Site.

Under current and proposed future commercial use, the applicable soil categories are S-2 and S-3. The AUL restricts future use of the Site for S-1 type uses.

Groundwater Category

MassDEP has identified three groundwater exposure categories (GW-1, GW-2, and GW-3) under the MCP, each reflective of a type of risk that may be posed by OHM in groundwater. Different combinations of these criteria are applicable at MCP sites depending upon the relevant site groundwater resource characteristics.

GW-1

The GW-1 category is applicable to locations where groundwater is, or may in the future be, a drinking water source.

No private drinking water supply wells used for drinking water purposes are known to be located within 500 feet of the Site and properties in the vicinity of the Site are serviced by the municipal water supply. There are no Current or Potential Drinking Water Source Areas within a one-half mile radius of the Site.

According to the MassDEP Site Scoring Map dated September 11, 2015, the Site is not located within the boundaries of Current or Potential Drinking Water Source Area, including:

1. A MassDEP Approved Zone II of a Public Water Supply Well,
2. Potentially Productive Aquifer (PPA),
3. Interim Wellhead Protection Area (IWPA), and
4. Zone A of a Class A Surface Water Body.

In addition, the Site is not located within the boundaries of:

5. An area designated by a municipality specifically for the protection of groundwater quality to ensure its availability for use as a source of potable water supply, and
6. U.S. Environmental Protection Agency (USEPA) Sole Source Aquifer.

The Site is underlain by medium and high yield aquifers designated as Non-Potential Drinking Water Source Areas (NPDWSA).

Therefore, GW-1 category is not applicable to the Site.

GW-2

The GW-2 category applies to locations where OHM may volatilize from the groundwater and migrate into an occupied structure.

Wells with an average annual depth to groundwater of fifteen (15) feet or less and located within 30 feet of a currently occupied or future planned building meet the criteria of GW-2.

Currently there is no building on the Site. However, proposed future use of the Site includes buildings. The groundwater sampling results in 2011 indicate depth to groundwater as approximately 4 to 6 feet bgs. Therefore, the GW-2 category is applicable to the future conditions of the Site.

GW-3

The GW-3 category is intended to protect environmental receptors in surface water that may be exposed to OHM when groundwater from an MCP site discharges to surface water. For all MCP sites, the GW-3 groundwater category is applicable.

There are no surface water bodies located on the Site. The nearest surface water body to the Site is the North River Canal located approximately 530 feet north of the Site. The North River Canal connects to the North River on the eastern side of the North Street Bridge. The North River discharges into the Danvers River approximately 1 mile east of the Site. Both the North River and the Danvers River are tidal rivers, which are connected to Beverly Harbor, which is located northeast of the Site. It is noted that the Site is located within the Federal Emergency Management Agency (FEMA) 100-year Floodplain.

Applicable Groundwater Categories

Given this set of circumstances, the applicable groundwater categories for the Site are GW-2 and GW-3.

2.2 Hazard Identification

2.2.1 Identification of Constituents of Concern

In accordance with MCP guidance (MassDEP, 1995), each detected compound should be considered a COC, unless one (1) of the following conditions is true:

- The chemicals are present at a low frequency of detection and in low concentration; or
- The chemicals are present at levels that are consistent with "background" concentrations for the area and there is no evidence that their presence is related to activities at the site; or
- The chemicals are field or laboratory contaminants.

Background Concentrations

The compounds detected in pre- and post-excavation soil samples collected at the Site were PCBs, extractable petroleum hydrocarbons (EPH) and volatile petroleum hydrocarbons (VPH) fractions and target analytes, metals, and volatile organic compounds (VOCs). The MassDEP has not formally established background concentrations for these constituents in soil, except for EPH target polycyclic aromatic hydrocarbons (PAHs) and metals.

Seventeen PAHs were detected in at least one soil sample. The maximum detected concentrations of each PAHs was greater than the MassDEP background concentration in "natural" soil (MassDEP, 2002a). Therefore, each of the detected PAHs was selected as a soil COC and carried through the Method 3 Risk Characterization.

Seven metals (arsenic, barium, cadmium, chromium, lead, mercury, and silver) were detected in at least one pre-excavation soil sample. The maximum detected concentrations of each metals were greater than the MassDEP background concentration in "natural" soil (MassDEP, 2002a). Therefore, these seven metals were selected as COCs for soil and carried through the Method 3 Risk Characterization.

Soil

The soil data representative of current post-excavation Site conditions were used to select soil COCs. The data represent samples that were from a depth ranges of 1.5 feet bgs or greater.

The compounds selected as COCs were the detected PCBs, EPH and VPH fractions and target analytes including seventeen PAHs, seven metals and chlorinated VOCs.

Groundwater

No COCs were selected for groundwater.

2.2.2 Toxicity Profiles

Toxicity profiles describe the potential human health effects posed by the constituents of concern, when doses are high enough to elicit an effect. Toxicity profiles for the constituents of concern at the Site are included in Appendix A.

2.2.3 Identification of Applicable or Suitably Analogous Standards

Applicable or suitably analogous standards are formally promulgated standards intended to protect human health and the environment from adverse effects of hazardous agents. Such standards are medium-specific.

There are no applicable or suitably analogous soil standards available for Site COCs. In accordance with MassDEP policy, MCP Method 1 Risk Characterization standards are not considered applicable or suitably analogous standards for Method 3 risk assessments. However, they are included on data tables in this Risk Characterization for reference.

Federal and state drinking water standards (such as Massachusetts Maximum Contaminant Levels [MMCLs]) are applicable or suitably analogous enforceable standards for sites classified as GW-1. Drinking water guidelines (e.g., MassDEP Office of Research and Standards Drinking Water Guidelines [ORSGL]) are not enforceable standards. The drinking water standards are applicable to a site, if the site groundwater is categorized as GW-1. The GW-1 category is not applicable to Site groundwater. Therefore, drinking water standards are not applicable to the subject Site.

Massachusetts Surface Water Quality Standards are applicable or suitably analogous standards to a site, if surface water is present and potentially impacted by a site. The potential for impact to surface water related to the Site has not been identified. Therefore, surface water quality standards are not applicable to the Site.

2.3 Dose-Response Assessment

Dose-response information describes the health effects observed in humans or animals associated with particular doses of a constituent. Based on the observed effect and target organ identified, a numerical value is developed to estimate the magnitude of the health effect associated with a dose. Dose-response values are derived differently for non-carcinogenic and carcinogenic effects, as discussed below.

Toxicity values were compiled for COCs in the media that serve as potential exposure points for human receptors. For this Site, soil serves as a potential exposure point.

The dose-response information for COCs are encoded in the MassDEP Method 3 Shortforms and are presented in the Shortforms Vlookup Version v0315 (MassDEP, 2015). The primary source of the toxicity values used by MassDEP was United States Environmental Protection Agency (USEPA) Integrated Risk Information System (IRIS) as of May 2012. IRIS was checked to identify any toxicity values for COCs that have been updated (USEPA, 2015). The toxicity values for COCs are presented in Table 1.

2.3.1 Threshold (Non-carcinogenic) Effects

For non-carcinogenic effects, there is believed to be a threshold level below which no

adverse health effects will occur.

Dose-response values for non-carcinogenic oral effects are referred to as Reference Doses (RfDs). For inhalation effects, these values are referred to as Reference Concentrations (RfCs). RfDs and RfCs represent provisional estimates of the threshold dose that will not pose risk of an adverse health effect to sensitive humans.

RfDs and RfCs are developed by applying uncertainty factors and modifying factors to the critical dose or concentration. This dose or concentration is usually either the Lowest-Observed-Adverse-Effect Level (LOAEL) or the No-Observed-Adverse-Effect Level (NOAEL) from toxicological studies, which are typically performed with test animals.

Uncertainty factors are used to account for interspecies variability, variation in sensitivity within the human population, differences in the route of administration among tests, and other variables that may lend uncertainty to the extrapolation of test data to environmental settings.

Units for RfDs are mg/kg/day, representing a dose of chemical (in milligrams) per receptor body weight (in kilograms) per day. For inhalation exposures, the RfC value is expressed as a concentration in air in mg/m^3 for continuous, 24 hour/day exposure.

Oral RfDs and inhalation RfCs for the COCs were summarized in Table 1.

2.3.2 Non-threshold (Carcinogenic) Effects

In accordance with MCP guidance, we have assumed that for carcinogenic effects there is no threshold level; that is, every non-zero exposure to a carcinogen is believed to be associated with some increased incremental risk. Dose-response values derived for carcinogenic compounds are Cancer Slope Factors (CSFs).

CSFs are calculated as the largest linear slope of the dose-response curve, which is generally extrapolated from the low-dose end of the curve. Oral CSFs are expressed in $(mg/kg/day)^{-1}$, and inhalation unit risks are expressed in $(ug/m^3)^{-1}$. Both values assume that the received dose is averaged over a lifetime.

USEPA's weight-of-evidence cancer classifications for each of the COCs at the Site were compiled. These classifications indicate whether existing human and animal data are sufficient to confirm whether there is an association between exposure to the compound and the occurrence of cancer.

Arsenic has a weight-of-evidence classification of Group A (i.e., Human Carcinogen - sufficient evidence in epidemiological studies to support causal association between exposure and cancer in humans). Cadmium, carcinogenic PAHs, lead, and PCBs have a Group B1/B2 weight-of-evidence classification (i.e., Probable Human Carcinogen). Trichloroethylene (TCE) has a Group C-B2 weight-of-evidence classification (i.e.,

Possible-Probable Human Carcinogen). The remaining COCs have been given a classification of D (i.e., Not Classifiable - inadequate or no human and animal evidence of carcinogenicity) or a classification is not available.

Carcinogenic values for inhalation exposures based on oral CSFs, called unit risks, are calculated by dividing the slope factor by the body weight (70 kg) and multiplying by the air inhalation rate (20 cubic meter [m^3]/day) for risk associated with unit concentration in air. Multiplication by 10^{-3} is necessary to convert mg (milligrams) to µg (micrograms).

Dose-response information for carcinogenic effects associated with Site COCs were summarized in Table 1.

2.3.3 Relative Absorption Factors

Relative absorption factors (RAFs) are used to account for the differences in absorption likely to occur between exposures under Site conditions and those that occurred under the experimental conditions that form the basis of the toxicity values. Absorption differences may result from matrix effects (e.g., doses absorbed from soil versus water) as well as from routes of administration (e.g., oral versus dermal exposure). RAFs adjust the calculated Site dose to make it comparable to the available toxicity information.

RAFs used in this risk assessment for exposure are encoded in the Method 3 Shortforms (MassDEP, 2015). In the absence of compound-specific data for inhalation exposures, a default RAF of one (1) was used, which conservatively assumes 100% uptake. The RAFs for Site COCs are presented in Table 2.

2.4 Exposure Assessment

The objectives of the Exposure Assessment are to:

1. Qualitatively and quantitatively describe the settings and conditions under which human exposures to Site OHM may reasonably be expected to occur, and
2. Calculate doses of Site OHM that human receptors may receive.

Achieving these goals entails the identification of receptors that may be on-Site, evaluation of exposure pathways, and the calculation of Exposure Point Concentrations (EPCs) to which receptors may be exposed.

2.4.1 Development of Exposure Profiles

Exposure profiles provide a narrative description of how exposures may take place at the Site. The profiles identify factors related to potential exposures and estimate their magnitude.

These factors include variables such as the receptors' body weights, intake rates, frequency of exposure, and duration of exposure. Exposure profiles are provided for each receptor identified under current and reasonably foreseeable future uses of the Site, including unrestricted residential use of the Site, in order to evaluate the need for an AUL in accordance with the MCP.

2.4.1.1 Identification of Potential Human Receptors and Exposure Points

Exposure points represent the locations where human or ecological receptors may come into contact with OHM at a site. These locations may be either single discrete points or areas/zones of affected media.

Potential human receptors were identified based on the current and reasonably foreseeable future use of the Site.

Under current Site conditions, there are "potentially accessible" impacted soil located under the parking lot and geotextile liner at depths of greater than 1.5 feet bgs. Under foreseeable future conditions, there will also be "isolated" impacted soil located under the Site building.

Under the AUL implemented at the Site, these impacted soils would not serve as an exposure point for residents, school employees and students, patrons, trespassers, commercial and office workers, and landscapers. MassDEP does not consider utility worker soil exposure to be a potentially significant risk unless the COCs have significant acute health effects (i.e., cyanide). In addition, the one utility drainage line that will traverse impacted soil will primarily be located in a corridor of clean fill.

Direct contact exposures to soils may potentially occur to impacted soil below the liner to construction workers during Site redevelopment. Soil may be directly contacted by construction workers during the installation of the building foundation and drainage line. As specified in the AEG RAM Plan, the building may be installed using a stone pier system to support the building slab. The system minimizes both the potential for direct contact to soil, as well as soil that would need to be excavated.

The AUL specifies that any excavation, movement and handling of soil below the geotextile liner at depths greater than 1.5 feet of more be conducted in accordance with a SMP and a HASP. The requirements for a SMP and a HASP are also presented in the AEG RAM Plan.

It is noted that potential exposures by construction workers to Site soils were considered to be protective of potential soil exposure to lesser-exposed utility workers, landscapers, and commercial workers who would not be conducting intrusive activities on a frequent and regular basis.

Potable water is supplied to the Site by a municipal source. Therefore, there is no current direct exposure pathway to groundwater as a drinking water supply.

Vapor intrusion from groundwater categorized as GW-2 (located at a depth of 15 feet or less and within 30 feet of an existing or planned future building) and soil (located within 6 feet horizontally and 10 feet vertically from the building) may be an indirect exposure pathway for building occupants.

The depth to groundwater at the Site is less than 15 feet bgs. Soil with low levels of volatile compounds do exist in close proximity to the proposed Site building foundation footings as impacted soil are located at depths of 10 feet bgs or less.

The proposed plan for the building is to place a vapor barrier under the foundation to mitigate the potential intrusion of vapors from underlying soil.

2.4.1.2 Identification of Exposure Routes

The exposure route describes how a receptor may contact contaminants at a site. The exposure routes identified for quantitative analysis in this Method 3 Risk Characterization are the inadvertent ingestion of constituents in soil and the dermal absorption of constituents from soils in contact with the skin and the inhalation of airborne particulates by construction workers.

Other exposure routes that were considered for quantitative analysis in the evaluation of current and unlimited future use, but were determined not to be complete or not to contribute significantly to overall risk, were:

- Incidental ingestion of and dermal contact with soil by Site commercial workers, utility workers, landscapers, trespassers, and visitors;
- Inhalation of fugitive dust by residents, workers and lesser-exposed receptors; and
- Dermal contact with groundwater and inhalation of volatiles released from groundwater in a trench by construction/utility workers.

Soil exposure is possible for utility workers and landscapers. Soil exposures were not quantitatively evaluated for these receptors as the evaluation of construction worker and resident exposure to Site soils, respectively, were considered protective of these lesser-exposed receptors.

Commercial workers, trespassers, and visitors are not expected to be engaged in regular activities that would result in direct contact exposure to soil.

The inhalation of fugitive dusts by Site workers, residents, and lesser-exposed receptors was not quantitatively evaluated. The inhalation of fugitive dusts was evaluated for construction workers. Under the condition that the impacted soils were excavated in the future, it is likely that the construction workers would be the maximum exposed individuals, and potential exposures to commercial and utility workers, residents, and other receptors would be lower.

Pathways for the construction/utility workers that involve groundwater exposures, including dermal contact with groundwater and inhalation of volatiles in ambient air released from groundwater in a trench, were not evaluated quantitatively. Subsurface excavation for utility work is typically to a depth of 6 feet, while construction work may occur to a depth of 15 feet. No significant groundwater impacts were encountered during site assessment and remediation activities.

In addition, direct contact with groundwater would generally be experienced only occasionally by laborers during dewatering, an activity that would result in short-term exposure (MassDEP, 1996b). It is not considered standard procedure for workers to enter an excavation containing water. We also assumed that Occupational Safety and Health Administration (OSHA) regulations and standard health and safety procedures regarding sloping of trench excavations, water in trenches, and atmospheres in trenches would be followed, which would further limit potential exposure.

For construction and utility excavation in the water table, dermal absorption of volatiles in groundwater and in contact with the skin and inhalation of volatiles by construction and utility workers were not considered significant exposure pathways.

2.4.1.3 Exposure Profile Summary

Exposure profile summaries bring together the different elements of the exposure profile to develop the relevant complete exposure pathways for each receptor.

Receptor (time frame)	Age Group	Time Frame	Medium	Exposure Route
Construction Worker (current/future)	Adult	Subchronic	Soil	Incidental Ingestion
				Dermal Contact
				Inhalation - Gastrointestinal
				Inhalation - Pulmonary

2.4.2 Development of Exposure Factors

Exposure factors, also referred to as exposure assumptions, are numerical estimates of the magnitude and duration of exposures that receptors may have to Site OHM. For the potential exposure to soil by construction workers, the MassDEP *Shortforms for Human Health Risk Assessment under the MCP sf12cw* with the Vlookup Version v0315 (MassDEP, 2015) was used for exposure assumptions and equations.

The exposure assumptions and equations for estimating exposures are presented in the following tables:

Table 3 - Construction Worker - Incidental Ingestion of and Dermal Contact with Soil, and Inhalation of Airborne Particulates

2.4.3 Exposure Point Concentrations (EPCs)

EPCs are the concentrations of OHM in Site media that are representative of the concentrations a receptor may be exposed to over the course of an exposure. EPCs are calculated separately for each OHM in each medium. The soil, groundwater, and indoor air analytical data discussed in Section 2.2.1 of this report were evaluated for the development of EPCs.

Groundwater EPCs are generally represented by detected concentrations in each individual well, while soil EPCs may represent an arithmetic average concentration within an affected area or over the entire site. The Site data were first evaluated to determine if a "hot spot" area(s) is (are) present.

Hot Spots

A hot spot is defined as a discrete area where concentrations of OHM or the thickness of non-aqueous phase liquid (NAPL) are substantially higher than those present in the surrounding area. A hot spot can be identified based on consideration of both the concentrations or thickness of an OHM within a contaminated area and the spatial pattern of that contamination.

A discrete area where the average concentration within the area is greater than ten (10) but less than 100 times the average concentration in the immediate surrounding area is a hot spot, unless there is no evidence that the discrete area would be associated with greater exposure potential than the surrounding area. In all cases, a discrete area where the concentration of OHM is greater than 100 times the concentration in the surrounding area is considered a hot spot. In addition, a hot spot cannot be created as a result of a remedial action (MassDEP, 2009), such as soil excavation.

Data from the affected areas of the Site were evaluated to identify potential "hot spots;" none were identified.

Soil

The MCP (310 CMR 40.924(2)(b)(4)) specifies that for current and potential soil exposures, the following depths should be considered, with any applicable Site-specific information, when determining EPCs:

1. 0 to three (3) feet for exposures associated with surficial activity;
2. 0 to six (6) feet for exposures associated with utility installation and repair;
3. 0 to fifteen (15) feet for exposures associated with excavation scenarios and building construction.

One set of soil EPCs was used to characterize potential risks to construction workers. The EPCs were developed in the Weston & Sampson Method 3 RC as presented in the

PSS, Appendix F. As presented in Section 5.4.3 of the Weston & Sampson Method 3 RC, soil EPCs were based on the 95th percentile upper confidence limit on the mean ("UCL95"). This UCL95 represents a concentration in which there is 95% confidence or certainty that the true mean of the sample population will be at or less than this value. The UCL95 was computed assuming that the data was normally distributed. One-half the reporting limit ("RL") was used for concentrations reported as non-detect.

The UCL95 for PCBs were calculated based on all data collected from the Site. The soil statistical summaries for other COCs included in Appendix F of the Weston & Sampson PSS were calculated for various depth intervals in the range of soil sample collection (i.e., 2 to 15 feet bgs).

Weston & Sampson used the EPCs calculated for the depth interval to approximately 6 feet below grade for soil exposure to construction workers. In general, the EPCs for this depth interval were the highest in comparison to EPCs for other depth intervals (e.g., 0 to 3 feet and 0 to 15 feet bgs).

The Weston & Sampson soil EPCs derived to approximately 6 feet below grade were used to calculated construction worker risks in this Method 3 RC.

2.4.4 Calculation of Average Daily Doses

The average daily dose (ADD) is a quantitative estimate of how much of each compound is taken into the receptor's body during exposure. The ADD is expressed as milligrams of OHM per kilograms of body weight per day. The general form of the dose equation is:

$$ADD = \frac{\text{(Total Amount of OHM Contacted) * (RAF)}}{\text{(Body Weight) * (Averaging Period)}}$$

ADDs are calculated differently for assessment of carcinogenic and non-carcinogenic effects. For non-carcinogenic effects, the ADD is averaged over the exposure period. The resulting ADD is an estimate of dose experienced during the actual period of exposure.

$$\text{Averaging Period}_{\text{non-carcinogenic}} = \text{Exposure Period}$$

For carcinogenic effects there is assumed to be no threshold level, and exposures are cumulative over a lifetime. The dose received is therefore averaged over a lifetime (70 years) instead of over just the exposure period. The resulting dose estimate is referred to as a lifetime average daily dose, or LADD.

$$\text{Averaging Period}_{\text{carcinogenic}} = \text{Lifetime (70 years)}$$

For air exposures, instead of an ADD, the average daily exposure (ADE) is estimated. The ADE is a quantitative estimate of the applied concentration of each compound for

the receptor during exposure.

The ADE is expressed as ug of OHM per m^3 of air and is based on the EPC and an adjustment for the amount of time the receptor spends in the area with contaminated air. The general form of the exposure equation is:

$$ADE = \frac{EPC * Exposure\ Duration * Exposure\ Frequency * Exposure\ Period * Conversion\ Factors}{Averaging\ Period}$$

As with ADDs, ADEs are calculated differently for assessment of carcinogenic and non-carcinogenic effects. For assessment of non-carcinogenic effects, the ADE is averaged over the exposure period. The resulting ADE is an estimate of dose experienced during the actual period of exposure.

$$Averaging\ Period_{non\text{-}carcinogenic} = Exposure\ Period$$

For carcinogenic effects there is assumed to be no threshold level, and exposures are cumulative over a lifetime. The exposure received is therefore averaged over a lifetime (70 years) instead of over just the exposure period. The resulting exposure estimate is referred to as a lifetime average daily exposure, or LADE.

The ADDs and ADEs for construction worker and resident soil exposure are encoded in the Shortforms *sf12cw* (Table 4).

2.5 Human Health Risk Characterization

Risk characterization is the final step in the risk assessment process. In this step, the results of the Hazard Assessment, Dose-Response Assessment, and Exposure Assessment are combined to yield quantitative estimates of incremental risk posed by potential exposures to environmental media at the Site. Separate estimates of potential cancer and non-cancer risk are made for each receptor and are discussed below.

These estimates are compared to applicable MCP risk management criteria to establish whether a Condition of No Significant Risk is present.

2.5.1 Non-Cancer Risk

The indicator used to describe the potential for non-carcinogenic health effects is the Hazard Quotient (HQ). For a given constituent, an HQ is the ratio of a receptor's exposure level (or dose) to the level of exposure considered safe.

In this Risk Characterization, a safe level of exposure is represented by the RfD or RfC for each compound. An HQ that does not exceed 1 indicates the receptor's exposure to that compound is without risk of adverse health effect.

$$Hazard\ Quotient = ADD/RfD\ or$$

Hazard Quotient = ADE/RfC

When the Hazard Quotients for each of the compounds of concern and exposure pathway at the Site are summed for each receptor, the result is a total Site Hazard Index (HI). The total Site HI is referred to as a screening HI because it does not segregate different compounds of concern based on their mode of toxicological activity. Thus, when used as an indicator of total Site non-carcinogenic risk, the screening HI is likely to overstate the actual level of non-carcinogenic risk. If the screening level HI is not greater than the noncancer risk limit of an HI of 1, this indicates there is no significant non-carcinogenic health risk associated with Site exposures. If the screening level HI is greater than 1, this HI is segregated by toxicity endpoint.

The hazard index for construction worker soil exposure is presented in Table 4. The cumulative screening hazard index value for construction workers does exceed 1. The exceedance is attributed to PCBs and lead. This finding indicates that a Condition of No Significant Risk for non-cancer effects does not exist for these receptors and medium.

2.5.2 Cancer Risk

The potential for carcinogenic health effects is estimated as the Incremental Excess Lifetime Cancer Risk (ELCR). The ELCR represents the incremental probability of an exposed individual developing cancer over a lifetime as a result of exposure. For each chemical, the ELCR is the product of the Lifetime Average Daily Dose (LADD) or Lifetime Average Daily Exposure (LADE) and that compound's carcinogenic potency.

The indicator of carcinogenic potency used in this risk characterization is the USEPA Cancer Slope Factor (CSF) or Unit Risk.

ELCR = LADD x CSF or
ELCR = LADE x Unit Risk

As in the case of non-cancer risk, the ELCRs for each of the different compounds and pathways are summed to produce a receptor-specific cumulative ELCR. This cumulative ELCR is compared to the cancer risk limit of 1×10^{-5} (one in one hundred thousand). A cumulative ELCR that does not exceed 1×10^{-5} indicates that no significant carcinogenic risk is present due to OHM at the Site. A cumulative ELCR greater than 1×10^{-5} indicates a potential for significant cancer risk is present as defined by the MCP.

The ELCRs for construction worker soil exposure is presented in Table 4. The calculated ELCRs for construction workers does not exceed 1×10^{-5}. This finding indicates a Condition of No Significant Risk for cancer effects exists for these receptors and medium.

2.5.3 Summary of Findings

1. The cumulative HI for construction workers from soil exposure is greater than MassDEP's risk management criteria of 1, indicating a Condition of No Significant Risk for non-cancer risks does not exist for this receptor and medium. Therefore, the HASP specified in the AUL and AEG RAM plan is required to achieve and maintain a Condition of No Significant Risk.

2. The cumulative ELCR for construction workers from soil exposure is not greater than MassDEP's risk management criteria of $1x10^{-5}$, indicating a Condition of No Significant Risk for cancer risks does exist for this receptor and medium.

3. The implemented AUL and current Site conditions of a geotextile liner at 1.5 feet bgs and pavement, and future Site conditions of an additional Site building serve as controls for the elimination of potential soil exposure for other receptors. These receptors include commercial workers, utility workers, landscapers, residents, school employees and students, patrons, and trespassers.

4. There were no current or foreseeable future complete exposure pathways identified related to Site groundwater.

5. There are no exceedances of applicable or suitably analogous standards.

Based on these findings, a Condition of No Significant Risk exists for current and future Site uses with the implementation of the AUL, including cap of liner, pavement, and building.

2.6 Uncertainty Analysis

The risk assessment process uses information from a variety of sources, such as analytical data from the Site investigation and toxicity data from published research. This information is combined with assumptions regarding potential receptors and Site use. Uncertainties may be present in each of these assumptions, and may affect the outcome of the risk assessment.

The risk assessment was developed to be a conservative estimate of potential adverse health effects. Its results should not be interpreted as definitive quantitative values. Uncertainties in the various portions of this risk assessment are discussed below.

A. Hazard Identification

The identification of constituents present in soil and groundwater and their distribution across the Site are dependent upon the sampling and analytical program conducted.

Conservative assumptions were made in developing soil and groundwater EPCs that are likely to lead to overestimates of actual exposure point concentrations.

EPCs were based on detected concentrations in samples collected from higher concentration areas. Sampling programs tend to focus on areas of higher concentration, resulting in a high-end estimate of the EPC.

B. Exposure Assessment

There is uncertainty associated with exposure assessment because the range of potential human activity is broad. Variability is associated with differences between individual receptors such as body weight, skin surface area, and rates of soil or water ingestion.

Conservative assumptions that are consistent with those recommended by MassDEP risk guidance documents have been used in developing pathway exposure factors that are anticipated to err on the side of the protection of human health.

C. Dose-Response Assessment

Toxicity information for many of the constituents detected at the Site is associated with varying degrees of uncertainty. Sources of uncertainty for toxicity values (USEPA, 1989) may include:

- Using dose-response information from effects observed at high doses to predict the health effects that may occur following exposure to low levels expected from human contact with the agent in the environment;
- Using dose-response information from short-term exposure to predict the effects of long-term exposures, and vice-versa;
- Using dose-response information from animal studies to predict effects in humans;
- Using dose-response information from homogeneous animal populations or healthy human populations to predict the effects likely to be observed in the general population, which will include individuals with a wide range of sensitivities.

Most of the toxicity values used in this Risk Characterization are USEPA-verified RfDs/RfCs and slope factors. These values, as presented in IRIS, are derived using a number of safety factors and are accompanied by a statement of confidence in the value itself, the critical study, and the overall data base for RfDs/RfCs, and the weight-of-evidence classifications for slope factors.

D. Risk Calculations

The risk calculations were performed using a deterministic methodology as required

under MCP guidance. In a deterministic methodology, a single value (point estimate) is used for exposure parameters and exposure point concentrations. The result is that a single risk value is calculated for each scenario and receptor of concern.

However, the use of a mix of mid-range and conservative exposure assumptions is intended to produce realistic upper-end exposure estimates, which will be protective of public health and produce risk estimates that will be valid for comparison to MCP Cumulative Risk Limits (MassDEP, 1995).

3.0 CHARACTERIZATION OF RISK OF HARM TO PUBLIC WELFARE

The MCP (310 CMR 40.0994) defines two (2) purposes for conducting a characterization of risk to public welfare: (a) to identify and evaluate nuisance conditions which may be localized, and (b) to identify and evaluate significant community effects. The characterization of risk to public welfare considers effects that exist or may result from the presence of residual contamination or the implementation of a proposed remedial alternative.

The characterization of the risk of harm to public welfare considers Site, receptor, and exposure information, as well as data collected pursuant to the response action(s) being performed.

The characterization of risk of harm to public welfare also considers such factors as the existence of nuisance conditions, loss of active or passive property use(s), and any non-pecuniary effects not otherwise considered in the characterization of risk of harm to health, safety, and the environment, but which may accrue due to the degradation of public resources directly attributed to the release or threat of release of OHM or the remedial alternative (310 CMR 40.0994(2)).

The risk of harm to public welfare is also characterized by comparing OHM concentrations to the UCLs in soil and groundwater, as defined in 310 CMR 40.0996. In addition, a level of No Significant Risk of harm to public welfare exists or has been achieved if no nuisance conditions, such as noxious odors, persist in the breathing zone of ambient and indoor air in the reasonably foreseeable future.

The EPCs for soil do not exceed the numerical UCLs.

The Site has been shown to contain soils that have been affected by constituents that may possess an odor at close range. Intermittent odors may occur if these soils are disturbed.

MassDEP guidance (MassDEP, 2002b) has suggested rules of thumb for determining when an odor condition would generally not be considered a nuisance condition. The rules of thumb that would be applicable to potential intermittent odors are:

1. Odors observed in the subsurface during excavation or boring advancement would generally not be considered a nuisance condition, as long as such odors are not detectable in ambient or indoor air, and as long as there are no plans to excavate or disturb such areas.
2. Odors observed in the breathing zone of the ambient air, or indoor air of an impacted structure, would generally not be considered a nuisance condition, if such odors do not persist for more than three (3) months.
3. Odors observed in the breathing zone of the ambient air would generally not be considered a nuisance condition if they are discernable less than ten (10) days a year.
4. Odors observed in the ambient air or indoor air of an affected structure would generally not be considered a nuisance condition if the occupants of such a structure do not believe such odors significantly affect or degrade their quality of life.

Potential odors are not believed to pose a significant risk to public welfare based on these rules of thumb being met and the infrequent occurrence and low potential for human exposures.

In addition, a vapor barrier is proposed to be installed under the Site building.

Therefore, there is No Significant Risk to public welfare for soils and groundwater at the Site under current and foreseeable future conditions.

4.0 CHARACTERIZATION OF RISK OF HARM TO SAFETY

The risk of harm to safety, as described in 310 CMR 40.0960, was evaluated for the Site. The following observations concerning the Site apply to release-related conditions at the Site and the relevant criteria set forth in Section 40.0960 of the MCP:

1. There are no rusted or corroded drums or containers, open pits, lagoons, or other dangerous structures at the Site;
2. There is no threat of fire or explosion from the presence of explosive vapors resulting from a release of OHM at the Site; and
3. There are no uncontained materials at the Site exhibiting the characteristics of corrosivity, reactivity, or flammability as described at 310 CMR 40.0347.

Therefore, there is not a risk of harm to safety due to release-related conditions at the Site.

5.0 ENVIRONMENTAL RISK CHARACTERIZATION

In accordance with Section 40.0995 of the MCP, this section of the risk assessment evaluates possible ecological risks due to OHM in soil and groundwater at the Site. For the Site, a Stage I Environmental Risk Screening was conducted.

The Stage I Screening steps are:

1. Identify complete Exposure Pathways [310 CMR 40.0995 (3)(a)],
2. Determine whether Readily Apparent Harm Exists [310 CMR 40.0995 (3)(b)], and
3. Establish if Potentially Significant Exposures Exist [310 CMR 40.0995 (3)(c)].

If there are complete exposure pathways then, for each complete exposure, Site conditions are evaluated to determine when significant environmental harm is "readily apparent" and if the potential exposure is significant.

If there are no complete exposure pathways, no further action to assess readily apparent harm and potentially significant exposures is required (MassDEP, 1996a). The Stage I screening for potential ecological receptors has been conducted separately for aquatic and terrestrial habitats.

5.1 Site Environmental Setting

There are no surface water bodies located on the Site. The nearest surface water body to the Site is the North River Canal located approximately 530 feet north of the Site. The North River Canal connects to the North River on the eastern side of the North Street Bridge. The North River discharges into the Danvers River approximately 1 mile east of the Site. Both the North River and the Danvers River are tidal rivers, which are connected to Beverly Harbor, which is located northeast of the Site. It is noted that the Site is located within the FEMA 100-year Floodplain.

5.2 Aquatic Habitat Screening

Complete Exposure Pathways - Four criteria are identified in the MCP (310 CMR 40.0995(3)(a)(1)) to assess whether there is evidence of current or potential future exposures to aquatic environmental receptors:

a. Evidence that OHM have come to be located in a surface water body or wetland,
b. Evidence that OHM have had an adverse impact on aquatic biota,
c. The presence of OHM in a surface water body or wetland as indicated by analytical data, and
d. The potential for transport of OHM in groundwater or surface runoff to aquatic receptors.

No significant impact to groundwater has been identified. Therefore, there is no evidence that OHM has had an adverse effect on aquatic biota or in the future a potential for adverse environmental impacts.

Therefore, conditions associated with the Site release do not pose a current or future potential for significant risk to ecological aquatic receptors in surface waters. Therefore, no current or potential future exposure is identified and a Condition of "No Significant

Risk of Harm" to Site aquatic biota and habitats exists or has been achieved.

5.3 Terrestrial Habitat Screening

Natural vegetation on the Site is limited due to the current development of the Site. Wildlife is limited to suburban biota, such as birds and small mammals. Their presence at the Site is likely to be of a transient and/or seasonal nature. Impacted soils are located a depth or under the Site building. Therefore, wildlife are not likely to access these soils.

A further evaluation of the presence of potentially significant exposure pathways was completed. Since no soil screening criteria are available, the terrestrial habitat has been screened on the basis of its size. For the purposes of this screening, the size of undeveloped/open land at the Site determines the specific evaluation of terrestrial environments.

MassDEP (1996a) states that, for the purposes of the screening process, undeveloped/open land is characterized by the presence of native vegetation, and does not include landscaped residential and commercial parcels, landscaped parks, or golf courses.

Based on this MassDEP definition, the open space at the Site is less than two (2) acres in size. Therefore, pursuant to the MCP, no further action to characterize ecological risk is required for such a site unless:

1. Contaminant transport from surface soil to an ACEC is possible, or
2. State-listed threatened or endangered species, or other species of special concern are present.

According to the MassDEP Site Scoring Map, the Site is not:

1. Within an ACEC, nor is contaminant transport from surface soil to an ACEC possible, or
2. The location of state-listed threatened or endangered species, or other species of special concern.

Based on the above information, potentially significant exposures do not exist for terrestrial ecological receptors potentially exposed to soils at the Site.

As a result, in summary, there is currently No Significant Risk to the environment from OHM detected in soil and groundwater at the Site.

6.0 CONCLUSIONS

In accordance with the Massachusetts Contingency Plan, 310 CMR 40.0990, we have conducted an updated Method 3 Risk Characterization related to the releases of OHM at 297 - 305 Bridge Street in Salem, Massachusetts, and referenced under RTN 3-11726.

To assess whether reported concentrations of OHM represent a Condition of NSR, this Method 3 Risk Characterization was completed. In accordance with the MCP, the Method 3 Risk Characterization included the following components:

1. Assessment of risks to human health,
2. Assessment of risks to public welfare,
3. Assessment of risk of harm to safety, and
4. Assessment of environmental risks.

The updated Method 3 RC indicates that the MassDEP and EPA risk-based cleanup achieved a Condition of NSR for current Site use with the construction of a pavement cap and future construction of a building, as well as implementation of an AUL to mitigate and control the future direct exposure to residual PCB soil impacts at the Site.

In general, the AUL was placed on the entire parcel to restrict future residential use and other Site activities that would result in greater exposure to residual contaminated soil at the Site. The AUL allows for industrial and commercial uses; and landscaping above the geotextile liner at 1.5 feet bgs; and underground utility and/or construction activities below the geotextile liner at depths greater than 1.5 feet bgs or more provided that a SMP and a HASP are implemented. The AUL also specifies maintenance of the existing asphalt, and no disturbance and direct contact with soil under the geotextile liner, except for underground utility and/or construction activities as described previously.

There were no complete direct exposure pathways identified for groundwater. The potential vapor intrusion pathway into the proposed Site building will be mitigated through the installation of a vapor barrier under the building slab.

The risk characterization also concluded that a Condition of No Significant Risk exists for public welfare, safety, and the environment at the Site based on available Site data and exposures evaluated for current and foreseeable future Site activities and uses.

Based on the conclusion of this Method 3 Risk Characterization, a Condition of No Significant Risk for current and future Site use has been achieved with presence of a cap (e.g., geotextile liner, pavement and proposed building) and implementation of the AUL. Therefore, a Permanent Solution with Conditions is applicable to the Site.

REFERENCES

Massachusetts Department of Environmental Protection (MassDEP). 1995. *Guidance for Disposal Site Risk Characterization - In Support of the Massachusetts Contingency Plan*. July 1995.

Massachusetts Department of Environmental Protection (MassDEP). 1996a. *Guidance for Disposal Site Risk Characterization In Support of the Massachusetts Contingency Plan *** Chapter 9 *** Method 3 Environmental Risk Characterization.* Bureau of Waste Site Cleanup and Office of Research and Standards. Interim Final Policy. April 1996.

Massachusetts Department of Environmental Protection (MassDEP). 1996b. *Draft Commercial/Industrial Shortform Exposure Scenario*. December 1996.

Massachusetts Department of Environmental Protection (MassDEP). 2002a. *Background Levels of Polycyclic Aromatic Hydrocarbons and Metals in Soil. Technical Update.* May 2002.

Massachusetts Department of Environmental Protection (MassDEP). 2002b. *Characterizing Risks Posed by Petroleum Contaminated Sites: Implementation of VPH/EPH Approach and Background/Support Documentation.* Final Policy. October 31, 2002. Policy #WSC-02-411.

Massachusetts Department of Environmental Protection (MassDEP). 2009. *Bureau of Waste Site Cleanup Master MCP Q&A: 1993-2009.*

Massachusetts Department of Environmental Protection (MassDEP). 2010. *Guidance on Implementing Activity and Use Limitations Policy #WSC 11-300*. December 2010.

Massachusetts Department of Environmental Protection (MassDEP). 2014a. *310 CMR 40.0000, the Massachusetts Contingency Plan.* Effective June 20, 2014.

Massachusetts Department of Environmental Protection (MassDEP). 2014b. Public *Hearing Draft Vapor Intrusion Guidance*. WSC#-14-435. October 2014.

Massachusetts Department of Environmental Protection (MassDEP). 2015. *ShortForms for Human Health Risk Assessment under the MCP.* Vlookup Version v0315. March 2015.

United States Environmental Protection Agency (USEPA). 2015. *Integrated Risk Information System*. Office of Health and Environmental Assessment. On-line database. October 2015.

Weston & Sampson. 2015. Phase III Remedial Action Plan (RAP) and Permanent Solution with Conditions Statement. January 2015.

TABLES

Table 1
Toxicity Values for Site Constituents of Concern (COCs)

Vlookup Version v0315																				
OIL OR HAZARDOUS MATERIAL	CAS	CHRONIC ORAL REFERENCE DOSE (OR SUBSTITUTE) mg/kg/day	REF	SUBCHRONIC ORAL REFERENCE DOSE (OR SUBSTITUTE) mg/kg/day	REF	Chronic Inhalation Reference Concentration (or substitute) mg/m3	REF	Subchronic Inhalation Reference Concentration (or substitute) mg/m3	REF	Oral Cancer Slope Factor 1/(mg/kg/day)	CLASS	REF	Inhalation Unit Risk 1/(µg/m3)	REF	Subchronic Inhalation Reference Dose (or Substitute) mg/kg/day	REF	Inhalation Cancer Slope Factor $(mg/kg/day)^{-1}$	REF	Chronic Inhalation Reference Dose (or Substitute) mg/kg/day	REF
ACENAPHTHENE	83-32-9	6.0E-02	1	2.0E-01	6	5.0E-02	5d	5.0E-01	5d						1.4E-01	5f			1.4E-02	5f
ACENAPHTHYLENE	208-96-8	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d		D	1			1.4E-01	5f			1.4E-02	5f
ACETONE	67-64-1	9.0E-01	1	2.7E+00	1i	8.0E-01	3	8.0E-01	7c		D	1			2.3E-01	5f			2.3E-01	5f
ANTHRACENE	120-12-7	3.0E-01	1	1.0E+00	6	5.0E-02	5d	5.0E-01	5d		D	1			1.4E-01	5f			1.4E-02	5f
ARSENIC	7440-38-2	3.0E-04	1	3.0E-04	2	2.0E-05	3a	2.0E-05	7c	1.5E+00	A	1	3.0E-03	3a	5.7E-06	5f	1.1E+01	5g	5.7E-06	5f
BARIUM	7440-39-3	2.0E-01	1	7.00E-02	1d	5.0E-04	2e	5.0E-03	2b						1.4E-03	5f			1.4E-04	5f
BENZO(a)ANTHRACENE	56-55-3	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E-01	B2	1e	2.1E-04	7a	1.4E-01	5f	7.3E-01	5g	1.4E-02	5f
BENZO(a)PYRENE	50-32-8	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E+00	B2	1	2.1E-03	7a	1.4E-01	5f	7.3E+00	5g	1.4E-02	5f
BENZO(b)FLUORANTHENE	205-99-2	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E-01	B2	1e	2.1E-04	7a	1.4E-01	5f	7.3E-01	5g	1.4E-02	5f
BENZO(g,h,i)PERYLENE	191-24-2	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d						1.4E-01	5f			1.4E-02	5f
BENZO(k)FLUORANTHENE	207-08-9	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E-02	B2	1e	2.1E-05	7a	1.4E-01	5f	7.3E-02	5g	1.4E-02	5f
CADMIUM	7440-43-9	5.0E-04	1c	5.0E-04	1d	2.0E-05	3a	2.0E-05	7c		B1	1	1.8E-03	3	5.7E-06	5f	6.3E+00	5g	5.7E-06	5f
CHROMIUM (TOTAL)	7440-47-3	3.0E-03	1	2.0E-02	2	1.0E-04	1	3.0E-04	1k				1.2E-02	1	8.6E-05	5f	4.2E+01	5g	2.9E-05	5f
CHRYSENE	218-01-9	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E-02	B2	1e	2.1E-05	7a	1.4E-01	5f	7.3E-02	5g	1.4E-02	5f
DIBENZO(a,h)ANTHRACENE	53-70-3	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E+00	B2	1e	2.1E-03	7a	1.4E-01	5f	7.3E+00	5g	1.4E-02	5f
DICHLOROBENZENE, 1,2- (o-DCB)	95-50-1	9.0E-02	1	9.0E-01	2d	8.0E-01	1m	2.4E+00	1m		D	1			6.9E-01	5f			2.3E-01	5f
DICHLOROETHYLENE, CIS-1,2-	156-59-2	2.0E-03	1	2.0E-02	6	6.0E-03	7b	6.0E-02	7b		D	1			1.7E-02	5f			1.7E-03	5f
FLUORANTHENE	206-44-0	4.0E-02	1	1.0E-01	6	5.0E-02	5d	5.0E-01	5d		D	1			1.4E-01	5f			1.4E-02	5f
FLUORENE	86-73-7	4.0E-02	1	4.0E-01	2	5.0E-02	5d	5.0E-01	5d						1.4E-01	5f			1.4E-02	5f
INDENO(1,2,3-cd)PYRENE	193-39-5	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d	7.3E-01	B2	1e	2.1E-04	7a	1.4E-01	5f	7.3E-01	5g	1.4E-02	5f
LEAD	7439-92-1	7.5E-04	4	7.5E-04	4	1.0E-03	3	1.0E-03	7c		B2	1			2.9E-04	5f			2.9E-04	5f
MERCURY	7439-97-6	3.0E-04	2d	3.0E-04	2d	3.0E-04	1	3.0E-04	7c		D	1			8.6E-05	5f			8.6E-05	5f
METHYLNAPHTHALENE, 2-	91-57-6	4.0E-03	1	4.0E-03	6	5.0E-02	5d	5.0E-01	5d						1.4E-01	5f			1.4E-02	5f
NAPHTHALENE	91-20-3	2.0E-02	1	2.0E-01	1i	3.0E-03	1	3.0E-03	7c						8.6E-04	5f			8.6E-04	5f
PETROLEUM HYDROCARBONS	NA																			
ALIPHATICS C5 to C8	NA	4.0E-02	5c	4.0E-01	5c	2.0E-01	5c	2.0E-01	7c						5.7E-02	5f			5.7E-02	5f
ALIPHATICS C9 to C12	NA	1.0E-01	5c	1.0E+00	5c	2.0E-01	5c	6.0E-01	5c						1.7E-01	5f			5.7E-02	5f
ALIPHATICS C9 to C18	NA	1.0E-01	5c	1.0E+00	5c	2.0E-01	5c	6.0E-01	5c						1.7E-01	5f			5.7E-02	5f
ALIPHATICS C19 to C36	NA	2.0E+00	5c	6.0E+00	5c															
AROMATICS C9 to C10	NA	3.0E-02	5c	3.0E-01	5c	5.0E-02	5c	5.0E-01	5c						1.4E-01	5f			1.4E-02	5f
AROMATICS C11 to C22	NA	3.0E-02	5c	3.0E-01	5c	5.0E-02	5c	5.0E-01	5c						1.4E-01	5f			1.4E-02	5f
PHENANTHRENE	85-01-8	3.0E-02	5d	3.0E-01	5d	5.0E-02	5d	5.0E-01	5d		D	1			1.4E-01	5f			1.4E-02	5f
POLYCHLORINATED BIPHENYLS (PCBs)	1336-36-3	2.0E-05	1	5.0E-05	2	2.0E-05	3	2.0E-05	7c	2.0E+00	B2	1	1.0E-04	1	5.7E-06	5f	3.5E-01	5g	5.7E-06	5f
PYRENE	129-00-0	3.0E-02	1	3.0E-01	6	5.0E-02	5d	5.0E-01	5d		D	1			1.4E-01	5f			1.4E-02	5f
SILVER	7440-22-4	5.0E-03	1	5.0E-03	2	1.4E-04	5b	1.4E-04	7c		D	1			4.0E-05	5f			4.0E-05	5f
TETRACHLOROETHYLENE	127-18-4	6.0E-03	1	6.0E-03	1d	4.0E-02	1	4.0E-02	7c	2.0E-02		5h	3.0E-06	5h	1.1E-02	5f	1.1E-02	5g	1.1E-02	5f
TRICHLOROBENZENE, 1,2,4-	120-82-1	1.0E-02	1	9.0E-02	6	2.0E-03	6	2.0E-02	6		D	1			5.7E-03	5f			5.7E-04	5f
TRICHLOROETHANE, 1,1,1-	71-55-6	2.0E+00	1	7.0E+00	1	5.0E+00	1	5.0E+00	1		D	1			1.4E+00	5f			1.4E+00	5f
TRICHLOROETHYLENE	79-01-6	5.0E-04	1	5.0E-04	1d	2.0E-03	1	2.0E-03	1j	5.0E-02	C-B2	1	5.0E-06	1	5.7E-04	5f	1.8E-02	5g	5.7E-04	5f
XYLENES (Mixed Isomers)	1330-20-7	2.0E-01	1	4.0E-01	6	1.0E-01	1	4.0E-01	6		D	1			1.1E-01	5f			2.9E-02	5f

Table 1
Toxicity Values for Site Constituents of Concern (COCs)

References used in calculating Method 3 Risk

Reference #	Description
	Toxicity Values
1	USEPA, Integrated Risk Information System (IRIS). Current as of May 2012.
1c	IRIS lists two oral RfDs for cadmium, one for food and one for water exposure. The more conservative is used.
1d	The chronic oral RfD (from IRIS) has been used here as a subchronic oral RfD equivalent.
1e	The IRIS Oral Cancer Slope Factor for benzo(a)pyrene is the basis for the Oral Cancer Slope Factor applied to the seven PAH compounds which are designated as category A, B1, B2 or C carcinogens.
1i	The subchronic RfD is based upon the subchronic toxicity data that is the basis of the chronic RfD presented in the IRIS file.
1j	The subchronic RfC is set equal to the chronic RfC based on information in the IRIS file.
1k	The subchronic RfC is based upon the subchronic toxicity data that is the basis of the chronic RfC presented in the IRIS file.
1m	The chronic and subchronic RfCs for 1,4-Dichlorobenze are used for 1,2- and 1,3- Dichlorobenzene.
2	USEPA Health Effects Assessment Summary Tables (HEAST), Annual FY-1996.
2b	The subchronic RfC is based upon the subchronic toxicity data that is the basis of the chronic RfC presented in HEAST.
2d	This value has been withdrawn from HEAST, MassDEP continues to use it pending new information.
2e	From Table 2 of HEAST. Values in Table 2 were calculated by an alternative method.
3	MassDEP Chemical Health Effects Assessment Methodology and Method to Derive Allowable Ambient Limits (CHEM/AAL) http://www.mass.gov/dep/toxics/stypes/telaal.htm
3a	MassDEP Methodology for Updating Air Guidelines: Allowable Ambient Limits (AALs) and Threshold Effects Exposure Limits (TELs) (MassDEP 2011). More info on the MassDEP Amibient Air Toxics Guidelines webpage. (http://www.mass.gov/eea/agencies/massdep/toxics/sources/air-guideline-values.html)
4	Developed for the Risk Assessment ShortForm - Residential Scenario (MassDEP, 1992) by MassDEP staff. Documentation of this value may be found in Appendix D of that document.
5c	Final Updated Petroleum Hydrocarbon Fraction Toxicity Values for the VPH/EPH/APH Methodology. See: http://www.mass.gov/dep/cleanup/laws/tphtox03.doc
5d	Toxicity values for PAHs are consistent with the approach presented in "Updated Petroleum Hydrocarbon Fraction Toxicity Values for the VPH/EPH/APH Methodology" MassDEP 2003 and Characterizing Risks Posed by Petroleum Contaminated Sites MassDEP 2002.
5f	Conversion of the chronic or subchronic Reference Concentration to an inhalation Reference Dose using the equation: RfC x Ventilation Rate/ Body Weight (RfC x V) / BW = (RfC x 20 m3/day) / 70 kg
5g	Conversion of the Inhalation Unit Risk Factor to an inhalation Cancer Slope Factor using the equation: URF x Conversion Factor x Body Weight / Ventilation Rate (URF x CF x V) / BW = (URF x 1000 x 20 m^3/day) / 70 kg
5h	Developed by MassDEP ORS in 2013, adopted in by MassDEP in January 2014.
6	PPRTVs
7a	Conversion of the oral Cancer Slope Factor to the inhalation Unit Risk, using the equation: Slope Factor x Ventilation Rate x Constant / Body Weight (CSF x V x C)/BW = (CSF x 20 m3/day x 0.001 mg/µg) / 70 kg
7b	Conversion of the oral Reference Dose to a Reference Concentration, using the equation: RfD x BW / Ventilation Rate RfC= (RfD x 70 kg) / 20 m3/day
7c	The Subchronic Inhalation Reference Concentration for this chemical is taken to be equal to the chronic value, absent clear chemical-specific information justifying a higher value..

Table 2
Relative Absorption Factors (RAFs) for Site Constituents of Concern (COCs)

Vlookup Version v0315 Relative Absorption Factors (RAFs)		Soil												Air[1]	
OIL OR HAZARDOUS MATERIAL	CAS	Chronic Ingestion	Ref	Chronic Dermal	Ref	Subchronic Ingestion	Ref	Subchronic Dermal	Ref	Cancer Ingestion	Ref	Cancer Dermal	Ref	NonCancer Inhalation	Cancer
ACENAPHTHENE	83-32-9	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
ACENAPHTHYLENE	208-96-8	0.3	9d	0.1	9d	0.3	9e	0.1	9d	NC		NC		1	
ACETONE	67-64-1	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	
ANTHRACENE	120-12-7	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
ARSENIC	7440-38-2	0.5	9e	0.03	9f	0.5	9e	0.03	9f	0.5	9e	0.03	9e	1	1
BARIUM	7440-39-3	1	9e	0.1	9e	1	9e	0.1	9e	NC		NC		1	
BENZO(a)ANTHRACENE	56-55-3	0.3	9d	0.02	9d	0.3	9d	0.02	9d	0.3	9d	0.02	9d	1	1
BENZO(a)PYRENE	50-32-8	0.3	9d	0.02	9d	0.3	9d	0.02	9d	0.3	9d	0.02	9d	1	1
BENZO(b)FLUORANTHENE	205-99-2	0.3	9d	0.02	9d	0.3	9d	0.02	9d	0.3	9d	0.02	9d	1	1
BENZO(g,h,i)PERYLENE	191-24-2	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
BENZO(k)FLUORANTHENE	207-08-9	0.3	9d	0.02	9d	0.3	9e	0.02	9d	0.3	9d	0.02	9d	1	1
CADMIUM	7440-43-9	0.5	9g	0.01	9e	0.5	9g	0.01	9e	NC		NC		1	1
CHROMIUM (TOTAL)	7440-47-3	1	9e	0.1	9e	1	9e	0.1	9e	NC		NC		1	1
CHRYSENE	218-01-9	0.3	9d	0.02	9d	0.3	9e	0.02	9d	0.3	9d	0.02	9d	1	1
DIBENZO(a,h)ANTHRACENE	53-70-3	0.3	9d	0.02	9d	0.3	9d	0.02	9d	0.3	9d	0.02	9d	1	1
DICHLOROBENZENE, 1,2- (o-DCB)	95-50-1	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	
DICHLOROETHYLENE, CIS-1,2-	156-59-2	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	
FLUORANTHENE	206-44-0	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
FLUORENE	86-73-7	0.3	9d	0.1	9d	0.3	9e	0.1	9d	NC		NC		1	
INDENO(1,2,3-cd)PYRENE	193-39-5	0.3	9d	0.02	9d	0.3	9d	0.02	9d	0.3	9d	0.02	9d	1	1
LEAD	7439-92-1	0.5	9	0.006	9	0.5	9	0.006	9	NC		NC		1	
MERCURY	7439-97-6	0.5	9e	0.1	9e	0.5	9e	0.1	9e	NC		NC		1	
METHYLNAPHTHALENE, 2-	91-57-6	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
NAPHTHALENE	91-20-3	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
PETROLEUM HYDROCARBONS	NA														
ALIPHATICS C5 to C8	NA	1	9e	0.2	9e	1	9e	0.2	9e	NC		NC		1	
ALIPHATICS C9 to C12	NA	1	9e	0.2	9e	1	9e	0.2	9e	NC		NC		1	
ALIPHATICS C9 to C18	NA	1	9e	0.2	9e	1	9e	0.2	9e	NC		NC		1	
ALIPHATICS C19 to C36	NA	1	9e	0.2	9e	1	9e	0.2	9e	NC		NC			
AROMATICS C11 to C22	NA	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
PHENANTHRENE	85-01-8	0.3	9d	0.1	9d	0.3	9e	0.1	9d	NC		NC		1	
POLYCHLORINATED BIPHENYLS (PCBs)	1336-36-3	1	9e	0.1	9a	1	9e	0.1	9a	1	9e	0.1	9a	1	1
PYRENE	129-00-0	0.3	9d	0.1	9d	0.3	9d	0.1	9d	NC		NC		1	
SILVER	7440-22-4	1	9e	0.3	9e	1	9e	0.3	9e	NC		NC		1	
TETRACHLOROETHYLENE	127-18-4	1	9e	0.03	9e	1	9e	0.03	9e	1	9e	0.03	9e	1	1
TRICHLOROBENZENE, 1,2,4-	120-82-1	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	
TRICHLOROETHANE, 1,1,1-	71-55-6	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	
TRICHLOROETHYLENE	79-01-6	1	9e	0.03	9e	1	9e	0.03	9e	1	9e	0.03	9e	1	1
XYLENES (Mixed Isomers)	1330-20-7	1	9e	0.03	9e	1	9e	0.03	9e	NC		NC		1	

Table 2
Relative Absorption Factors (RAFs) for Site Constituents of Concern (COCs)

References used in calculating Method 3 Risk

Reference # Description

RAFs

1 Default values. If there is no Reference Concentration, a non-cancer inhalation RAF was not implied. If there is no Inhalation Unit Risk, a cancer inhalation RAF was not implied.

9 MassDEP 2012 RAF Review. Unless specified otherwise, due to data limitations and consistent with the approach in Ontario Ministry of the Environment (2011 - for full reference see note 48e), a default RAF of 1 was chosen for all organic compounds for oral ingestion of contaminated soil and water.

9a MassDEP 2012 RAF Review - Dermal RAFs for dioxins, furans, and PCBs consider data presented in: Brewster DW, Banks YB, Clark AM and Birbaum LS. (1998). Comparative Dermal Absorption of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Three Polychlorinated Dibenzofurans. Toxicol Appl Pharacol 97(1):156-166.
Mayes BA, Brown GL, Mondello FJ, Holtzclaw KW, Hamilton SB, Ramsey AA. (2002).Dermal Absorption in Rhesus Monkeys of Polychlorinated Biphenyls from Soil Contaminated With Aroclor 1260. Regul Toxicol Pharmacol 35(3):289-295.
Roy TA, Hammerstron AK and Schaum J. (2008). Percutaneous Absorption of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) from Soil. J. Toxicol Environ Health, Part A: Current Issues: 1509-1515.
Wester RC, Maibach HI, Sedik L, Melendres J, and Wade M. (1993). Percutaneous Absorption of PCBs from Soil: In-vivo Rhesus Monkey, In-vitro Human Skin, and Binding to Powered Human Stratum Corneum. J. Toxicol. Environ. Health 39:375-382.
Absorption of these compounds from soil with high to low organic content has been reported to range from less than 1% to over 10%. In light of the variability in the reported dermal absorption values and study characteristics, a default value of 0.1 was selected, which is toward the high end of the reported values.

9d MassDEP 2012 RAF Review - Based on Magee B, Andersen P and Burmaster. (1996). Absorption Adjustment Factor (AAF) Distributions for Polycyclic Aromatic Hydrocarbons (PAHs). Human and Ecological Risk Assessment 2:841-873.

9e MassDEP 2012 RAF Review - Based on Ontario Ministry of the Environment (2011). Rationale for the Development of Soil and Ground Water Standards for Use at Contaminated Sites in Ontario (April 15, 2011, Standards Development Branch, Ontario Ministry of the Environment (see Section 2.6, Development of Relative Absorption Factors, pp 61-67 and Table 2.35b Estimation of Dermal Relative Absorption Factors (RAFs) PP 120- 141) http://www.ene.gov.on.ca/environment/en/resources/STDPROD_081485.html; Accessed March 22, 2012.

9e MassDEP 2012 RAF Review - Based on USEPA (2004). Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual Part E, Supplemental Guidance for Dermal Risk Assessment.

Table 3
Exposure Assumptions and Equations for Construction Worker - Incidental Ingestion of and Dermal Contact with Soil, and Inhalation of Particulates

Construction Worker - Soil: Table CW-2
Equations to Calculate Cancer Risk for Construction Worker

Vlookup Version v0315

Cancer Risk from Ingestion

$$ELCR_{ing} = LADD_{ing} * CSF_{oral}$$

$$LADD_{ing} = \frac{EPC * IR * RAF_{c\text{-}ing} * EF * ED_{ing} * EP * C1}{BW * AP_{lifetime}}$$

Cancer Risk from Dermal Absorption

$$ELCR_{derm} = LADD_{derm} * CSF_{oral}$$

$$LADD_{derm} = \frac{EPC * SA * AF * RAF_{c\text{-}derm} * EF * ED_{derm} * EP * C1}{BW * AP_{lifetime}}$$

Cancer Risk from Particulate Inhalation - Gastrointestinal Absorption

$$ELCR_{inh\text{-}GI} = LADD_{inh\text{-}GI} * CSF_{oral}$$

$$LADD_{inh\text{-}GI} = \frac{EPC * RCAF_{inh\text{-}gi} * PM_{10} * VR_{work} * RAF_{c\text{-}ing} * EF * ED_{inh} * EP * C2 * C3 * C4}{BW * AP_{lifetime}}$$

Cancer Risk from Particulate Inhalation - Pulmonary Absorption

$$ELCR_{inh} = LADD_{inh} * CSF_{inhalation}$$

$$LADD = \frac{EPC * RCAF_{inh} * PM_{10} * VR_{work} * RAF_{c\text{-}inh} * EF * ED_{inh} * EP * C2 * C3 * C4}{BW * AP_{lifetime}}$$

Parameter	Value	Units
CSF	OHM-specific	$(mg/kg\text{-}day)^{-1}$
LADD	age/OHM-specific	mg/kg-day
EPC	OHM-specific	mg/kg
IR	100	mg/day
$RAF_{c\text{-}ing}$	OHM-specific	dimensionless
$RAF_{c\text{-}derm}$	OHM-specific	dimensionless
$RAF_{c\text{-}inh}$	OHM-specific	dimensionless
EF	0.714	event/day
$ED_{ing\ \&\ derm}$	1	day/event
ED_{inh}	0.333	day/event
EP	182	days
C1	1.0E-06	kg/mg
C2	1.0E-09	kg/µg
C3	1440	min/days
C4	1.0E-03	m^3/L
BW	58.0	kg
$AP_{(lifetime)}$	25,550	days
VR_{work}	60	L/min
AF	0.29	mg/cm^2
SA	3473	cm^2/day
$RCAF_{inh\text{-}gi}$	1.5	dimensionless
$RCAF_{inh}$	0.5	dimensionless
PM_{10}	60	$\mu g/m^3$

Contact: Lydia Thompson
Lydia.Thompson@state.ma.us
617-556-1165

Table 3 (cont'd.)
Exposure Assumptions and Equations for Construction Worker - Incidental Ingestion of and Dermal Contact with Soil, and Inhalation of Particulates

Construction Worker - Soil: Table CW-3
Equations to Calculate Noncancer Risk for Construction Worker

Vlookup Version v0315

Noncancer Risk from Ingestion

$$HQ_{ing} = \frac{ADD_{ing}}{RfD_{oral\text{-}subchronic}}$$

$$ADD_{ing} = \frac{EPC * IR * RAF_{nc\text{-}ing} * EF * ED_{ing} * EP * C1}{BW * AP_{noncancer}}$$

Noncancer Risk from Dermal Absorption

$$HQ_{derm} = \frac{ADD_{derm}}{RfD_{oral\text{-}subchronic}}$$

$$ADD_{dermal} = \frac{EPC * SA * AF * RAF_{nc\text{-}derm} * EF * ED_{dermal} * EP * C1}{BW * AP_{noncancer}}$$

Noncancer Risk from Particulate Inhalation - Gastrointestinal Absorption

$$HQ_{inh\text{-}GI} = \frac{ADD_{inh\text{-}GI}}{RfD_{oral\text{-}subchronic}}$$

$$ADD_{inh\text{-}GI} = \frac{EPC * RCAF_{inh\text{-}gi} * PM_{10} * VR_{work} * RAF_{nc\text{-}ing} * EF * ED_{inh} * EP * C2 * C3 * C4}{BW * AP_{noncancer}}$$

Noncancer Risk from Particulate Inhalation - Pulmonary Absorption

$$HQ_{inh} = \frac{ADD}{RfD_{inhalation\text{-}subchronic}}$$

$$ADD_{inh} = \frac{EPC_{soil} * RCAF_{inh} * PM_{10} * VR_{work} * RAF_{nc\text{-}inh} * EF * ED_{inh} * EP * C2 * C3 * C4}{BW * AP_{noncancer}}$$

Parameter	Value	Units
RfD	OHM-specific	mg/kg-day
ADD	OHM-specific	mg/kg-day
EPC	OHM-specific	mg/kg
IR	100	mg/day
$RAF_{nc\text{-}ing}$	OHM-specific	dimensionless
$RAF_{nc\text{-}derm}$	OHM-specific	dimensionless
$RAF_{nc\text{-}inh}$	OHM-specific	dimensionless
EF	0.714	event/day
$ED_{ing\ \&\ derm}$	1	day/event
ED_{inh}	0.333	day/event
EP	182	days
C1	1.0E-06	kg/mg
C2	1.0E-09	kg/µg
C3	1440	min/days
C4	1.0E-03	m^3/L
BW	58.0	kg
$AP_{noncancer}$	182	days
VR_{work}	60	L/min
AF	0.29	mg/cm^2
SA	3473	cm^2/day
$RCAF_{inh\text{-}gi}$	1.5	dimensionless
$RCAF_{inh}$	0.5	dimensionless
PM10	60	$µg/m^3$

MassDEP ORS
Contact: Lydia Thompson
Lydia.Thompson@state.ma.us
617-556-1165

Table 3 (cont'd.)
Exposure Assumptions and Equations for Construction Worker - Incidental Ingestion of and Dermal Contact with Soil, and Inhalation of Particulates

Construction Worker - Soil: Table CW-4
Definitions and Exposure Factors

Vlookup Version v0315

Parameter	Value	Units	Notes
ELCR - Excess Lifetime Cancer Risk	chemical specific	dimensionless	Pathway specific (ing =ingestion, derm=dermal, inh=inhalation)
HI - Hazard Index	chemical specific	dimensionless	Pathway specific (ing =ingestion, derm=dermal, inh=inhalation)
CSF - Cancer Slope Factor	chemical specific	$(mg/kg\text{-}day)^{-1}$	see Table CW-5.
RfD - Reference Dose	chemical specific	mg/kg-day	see Table CW-5.
LADD - Lifetime Average Daily Dose	chemical specific	mg/kg-day	Pathway specific. See Table CW-2.
ADD - Average Daily Dose	chemical specific	mg/kg-day	Pathway specific. See Table CW-3.
EPC - Exposure Point Concentration	chemical specific	mg/kg	see Table CW-1.
IR - Soil Ingestion Rate	100	mg/day	MADEP. 2002. Technical Update: Calculation of an Enhanced Soil Ingestion Rate. (http://www.mass.gov/dep/ors/orspubs.htm).
RAF_c - Relative Absorption Factor for Cancer Effects	chemical specific	dimensionless	Pathway specific - see Table CW-5.
RAF_{nc} - Relative Absorption Factor for Noncancer Effects	chemical specific	dimensionless	Pathway specific - see Table CW-5.
EF - Exposure Frequency	0.714	event/day	5 events (days) / 7 events (days) in a week; MADEP 1995 Guidance for Disposal Site Risk Characterization pg B-38.
$ED_{ing,derm}$ - Exposure Duration for ingestion or dermal exposure	1	day/event	
ED_{inh} - Exposure Duration for inhalation exposure	0.333	day/event	Represents 8 hours / event.
EP - Exposure Period	182	days	6 months; MADEP 1995 Guidance for Disposal Site Risk Characterization.
BW - Body Weight	58.0	kg	U.S. EPA. 1997. Exposure Factors Handbook. Table 7-7, Females, ages 18 - 25.
$AP_{(lifetime)}$ - Averaging Period for lifetime	25,550	days	Represents 70 years
$AP_{(noncancer)}$ - Averaging Period for noncancer	182	days	6 months; MADEP 1995 Guidance for Disposal Site Risk Characterization.
AF - Adherence Factor	0.29	mg/cm^2	MA DEP. 2002 Technical Update: Weighted Skin-Soil Adherence Factors. (http://www.mass.gov/dep/ors/orspubs.htm)
VR_{work} - Ventilation Rate during work (heavy exertion)	60	L/min	Table B-4 MADEP 1995 Guidance for Disposal Site Risk Characterization.
SA - Surface Area	3473	cm^2/day	MADEP. 1995. Guidance for Disposal Site Risk Characterization. 50th percentile for females. Appendix Table B-2.
$IFAF_{inh\text{-}gi}$ - Ingestion Fraction Adjustment Factor, gastrointestinal	1.5	dimensionless	MADEP 2007. Characterization of Risks Due to Inhalation of Particulates by Construction Workers
$IFAF_{inh}$ - Inhalation Fraction Adjustment Factor, inhalation	0.5	dimensionless	MADEP 2002. Characterization of Risks Due to Inhalation of Particulates by Construction Workers
PM10 - Concentration of PM_{10}	60	$\mu g/m^3$	MADEP 1995 Guidance for Disposal Site Risk Characterization pg B-11

MassDEP ORS
Contact: Lydia Thompson
Lydia.Thompson@state.ma.us
617-556-1165

Table 4
Calculation of Risk Estimates for Construction Worker - Incidental Ingestion of and Dermal Contact with Soil, and Inhalation of Particulates

Construction Worker - Soil: Table CW-1
Exposure Point Concentration (EPC) and Risk
Based on Construction Worker 18-25 years of age

ShortForm Version 10-12
Vlookup Version v0315

ELCR (all chemicals) = 2.3E-06
HI (all chemicals) = 2.8E+00

****Do not insert or delete any rows****
Click on empty cell below and select OHM using arrow.

Oil or Hazardous Material (OHM)	EPC (mg/kg)	ELCR ingestion	ELCR dermal	ELCR inhalation GI	ELCR inhalation pulmonary	$ELCR_{total}$	Subchronic HQ_{ing}	Subchronic HQ_{derm}	Subchronic HQ_{inh-GI}	Subchronic HQ_{inh}	HQ_{total}
POLYCHLORINATED BIPHENYLS (PCBs)	2.6E+01	4.6E-07	4.6E-07	1.2E-08	6.9E-10	9.3E-07	6.4E-01	6.5E-01	1.7E-02	4.8E-02	1.4E+00
ALIPHATICS C9 to C12	1.3E+01						1.6E-05	3.2E-05	4.1E-07	8.1E-07	4.9E-05
AROMATICS C9 to C10	1.0E+01						4.1E-05	8.3E-05	1.1E-06	7.4E-07	1.3E-04
ACETONE	4.9E-02						2.2E-08	6.8E-09	5.8E-10	2.3E-09	3.2E-08
DICHLOROBENZENE, 1,2- (o-DCB)	1.8E-03						2.5E-09	7.4E-10	6.4E-11	2.8E-11	3.3E-09
DICHLOROETHYLENE, CIS-1,2-	6.7E-02						4.1E-06	1.2E-06	1.1E-07	4.2E-08	5.5E-06
TETRACHLOROETHYLENE	1.9E-02	3.3E-12	1.0E-12	8.6E-14	1.5E-14	4.4E-12	3.9E-06	1.2E-06	1.0E-07	1.8E-08	5.2E-06
TRICHLOROBENZENE, 1,2,4-	1.8E-03						2.5E-08	7.4E-09	6.4E-10	3.4E-09	3.6E-08
TRICHLOROETHANE, 1,1,1-	2.4E-03						4.2E-10	1.3E-10	1.1E-11	1.8E-11	5.8E-10
TRICHLOROETHYLENE	1.2E-01	5.3E-11	1.6E-11	1.4E-12	1.6E-13	7.0E-11	3.0E-04	8.9E-05	7.7E-06	2.2E-06	3.9E-04
XYLENES (Mixed Isomers)	1.8E-03						5.5E-09	1.7E-09	1.4E-10	1.7E-10	7.5E-09
ALIPHATICS C9 to C18	1.2E+02						1.5E-04	3.0E-04	3.8E-06	7.4E-06	4.6E-04
ALIPHATICS C19 to C36	6.3E+02						1.3E-04	2.6E-04	3.4E-06		3.9E-04
AROMATICS C11 to C22	1.2E+02						1.5E-04	5.0E-04	3.8E-06	8.9E-06	6.6E-04
ACENAPHTHENE	3.1E+00						5.7E-06	1.9E-05	1.5E-07	2.3E-07	2.5E-05
ACENAPHTHYLENE	3.8E-01						4.7E-07	1.6E-06	1.2E-08	2.8E-08	2.1E-06
ANTHRACENE	7.5E+00						2.8E-06	9.3E-06	7.2E-08	5.6E-07	1.3E-05
BENZO(a)ANTHRACENE	1.1E+01	2.1E-08	1.4E-08	5.5E-10	6.1E-10	3.6E-08	1.4E-05	9.1E-06	3.5E-07	8.2E-07	2.4E-05
BENZO(a)PYRENE	9.3E+00	1.8E-07	1.2E-07	4.6E-09	5.1E-09	3.1E-07	1.1E-05	7.7E-06	3.0E-07	6.9E-07	2.0E-05
BENZO(b)FLUORANTHENE	1.1E+01	2.1E-08	1.4E-08	5.5E-10	6.1E-10	3.6E-08	1.4E-05	9.1E-06	3.5E-07	8.2E-07	2.4E-05
BENZO(g,h,i)PERYLENE	4.5E+00						5.5E-06	1.9E-05	1.4E-07	3.4E-07	2.5E-05
BENZO(k)FLUORANTHENE	4.9E+00	9.4E-10	6.3E-10	2.4E-11	2.7E-11	1.6E-09	6.0E-06	4.1E-06	1.6E-07	3.6E-07	1.1E-05
CHRYSENE	1.0E+01	1.9E-09	1.3E-09	5.0E-11	5.5E-11	3.3E-09	1.2E-05	8.3E-06	3.2E-07	7.4E-07	2.2E-05
DIBENZO(a,h)ANTHRACENE	4.1E-01	7.9E-09	5.3E-09	2.0E-10	2.3E-10	1.4E-08	5.0E-07	3.4E-07	1.3E-08	3.1E-08	8.9E-07
FLUORANTHENE	2.4E+01						8.9E-05	3.0E-04	2.3E-06	1.8E-06	3.9E-04
FLUORENE	3.3E+00						3.0E-06	1.0E-05	7.9E-08	2.5E-07	1.4E-05
INDENO(1,2,3-cd)PYRENE	5.3E+00	1.0E-08	6.8E-09	2.6E-10	2.9E-10	1.8E-08	6.5E-06	4.4E-06	1.7E-07	3.9E-07	1.1E-05
METHYLNAPHTHALENE, 2-	1.1E+00						1.0E-04	3.4E-04	2.6E-06	8.2E-08	4.5E-04
NAPHTHALENE	2.9E+00						5.4E-06	1.8E-05	1.4E-07	3.6E-05	5.9E-05
PHENANTHRENE	2.4E+01						3.0E-05	9.9E-05	7.7E-07	1.8E-06	1.3E-04
PYRENE	2.1E+01						2.6E-05	8.7E-05	6.7E-07	1.6E-06	1.1E-04
ARSENIC	1.4E+01	9.2E-08	5.6E-08	2.4E-09	1.1E-08	1.6E-07	2.9E-02	1.7E-02	7.4E-04	2.6E-02	7.3E-02
BARIUM	3.0E+02						5.3E-03	5.3E-03	1.4E-04	2.2E-03	1.3E-02
CADMIUM	5.9E+00				2.8E-09	2.8E-09	7.3E-03	1.5E-03	1.9E-04	1.1E-02	2.0E-02
CHROMIUM (TOTAL)	2.5E+02				8.0E-07	8.0E-07	1.5E-02	1.6E-02	4.0E-04	3.1E-02	6.2E-02
LEAD	1.3E+03						1.1E+00	1.3E-01	2.8E-02	4.8E-02	1.3E+00
MERCURY	2.8E+00						5.7E-03	1.2E-02	1.5E-04	3.5E-04	1.8E-02
SILVER	7.8E-01						1.9E-04	5.8E-04	5.0E-06	2.1E-04	9.9E-04

Note! Cr(VI) limit is 200 mg/kg due to contact dermitis.

MassDEP ORS
Contact: Lydia Thompson
Lydia.Thompson@state.ma.us
617-556-1165

APPENDIX A
TOXICITY PROFILES

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

ACETONE
CAS # 67-64-1

This fact sheet answers the most frequently asked health questions (FAQs) about acetone. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to acetone results mostly from breathing air, drinking water, or coming in contact with products or soil that contain acetone. Exposure to moderate-to-high amounts of acetone can irritate your eyes and respiratory system, and make you dizzy. Very high exposure may cause you to lose consciousness. This chemical has been found in at least 572 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is acetone?

(Pronounced ăs′ĭ-tōn′)

Acetone is a manufactured chemical that is also found naturally in the environment. It is a colorless liquid with a distinct smell and taste. It evaporates easily, is flammable, and dissolves in water. It is also called dimethyl ketone, 2-propanone, and beta-ketopropane.

Acetone is used to make plastic, fibers, drugs, and other chemicals. It is also used to dissolve other substances.

It occurs naturally in plants, trees, volcanic gases, forest fires, and as a product of the breakdown of body fat. It is present in vehicle exhaust, tobacco smoke, and landfill sites. Industrial processes contribute more acetone to the environment than natural processes.

What happens to acetone when it enters the environment?

- A large percentage (97%) of the acetone released during its manufacture or use goes into the air.
- In air, about one-half of the total amount breaks down from sunlight or other chemicals every 22 days.
- It moves from the atmosphere into the water and soil by rain and snow. It also moves quickly from soil and water back to air.
- Acetone doesn't bind to soil or build up in animals.
- It's broken down by microorganisms in soil and water.
- It can move into groundwater from spills or landfills.
- Acetone is broken down in water and soil, but the time required for this to happen varies.

How might I be exposed to acetone?

- Breathing low background levels in the environment.
- Breathing higher levels of contaminated air in the workplace or from using products that contain acetone (for example, household chemicals, nail polish, and paint).
- Drinking water or eating food containing acetone.
- Touching products containing acetone.
- For children, eating soil at landfills or hazardous waste sites that contain acetone.
- Smoking or breathing secondhand smoke.

How can acetone affect my health?

If you are exposed to acetone, it goes into your blood which then carries it to all the organs in your body. If it is a small amount, the liver breaks it down to chemicals that are not harmful and uses these chemicals to make energy for normal body functions. Breathing moderate- to-high levels

of acetone for short periods of time, however, can cause nose, throat, lung, and eye irritation; headaches; light-headedness; confusion; increased pulse rate; effects on blood; nausea; vomiting; unconsciousness and possibly coma; and shortening of the menstrual cycle in women.

Swallowing very high levels of acetone can result in unconsciousness and damage to the skin in your mouth. Skin contact can result in irritation and damage to your skin.

The smell and respiratory irritation or burning eyes that occur from moderate levels are excellent warning signs that can help you avoid breathing damaging levels of acetone.

Health effects from long-term exposures are known mostly from animal studies. Kidney, liver, and nerve damage, increased birth defects, and lowered ability to reproduce (males only) occurred in animals exposed long-term. It is not known if people would have these same effects.

How likely is acetone to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified acetone for carcinogenicity.

Acetone does not cause skin cancer in animals when applied to the skin. We don't know if breathing or swallowing acetone for long periods will cause cancer. Studies of workers exposed to it found no significant risk of death from cancer.

Is there a medical test to show whether I've been exposed to acetone?

Methods are available to measure the amount of acetone in your breath, blood, and urine. The test can tell you how much acetone you were exposed to, although the amount that people have naturally in their bodies varies with each person. The tests can't tell you if you will experience any health effects from the exposure.

The test must be performed within 2-3 days after exposure because acetone leaves your body within a few days. These tests are not routinely performed at your doctor's office, but your doctor can take blood or urine samples and send them to a testing laboratory.

Has the federal government made recommendations to protect human health?

The EPA requires that spills of 5,000 pounds or more of acetone be reported.

The Occupational Safety and Health Administration (OSHA) has set a maximum concentration limit in workplace air of 1,000 parts of acetone per million parts of air (1,000 ppm) for an 8-hour workday over a 40-hour week to protect workers. The National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit of 250 ppm in workplace air for up to a 10-hour workday over a 40-hour workweek.

Glossary

Carcinogenicity: Ability to cause cancer.
Evaporate: To change into a vapor or a gas.
Ingesting: Taking food or drink into your body.
Long-term: Lasting one year or longer.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1994. Toxicological profile for acetone. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone:1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

ARSENIC

CAS # 7440-38-2

Division of Toxicology and Environmental Medicine ToxFAQs™ **August 2007**

This fact sheet answers the most frequently asked health questions (FAQs) about arsenic. For more information, call the ATSDR Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to higher than average levels of arsenic occur mostly in the workplace, near hazardous waste sites, or in areas with high natural levels. At high levels, inorganic arsenic can cause death. Exposure to lower levels for a long time can cause a discoloration of the skin and the appearance of small corns or warts. Arsenic has been found in at least 1,149 of the 1,684 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is arsenic?

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Copper chromated arsenate (CCA) is used to make "pressure-treated" lumber. CCA is no longer used in the U.S. for residential uses; it is still used in industrial applications. Organic arsenic compounds are used as pesticides, primarily on cotton fields and orchards.

What happens to arsenic when it enters the environment?

❑ Arsenic occurs naturally in soil and minerals and may enter the air, water, and land from wind-blown dust and may get into water from runoff and leaching.
❑ Arsenic cannot be destroyed in the environment. It can only change its form.
❑ Rain and snow remove arsenic dust particles from the air.
❑ Many common arsenic compounds can dissolve in water. Most of the arsenic in water will ultimately end up in soil or sediment.
❑ Fish and shellfish can accumulate arsenic; most of this arsenic is in an organic form called arsenobetaine that is much less harmful.

How might I be exposed to arsenic?

❑ Ingesting small amounts present in your food and water or breathing air containing arsenic.
❑ Breathing sawdust or burning smoke from wood treated with arsenic.
❑ Living in areas with unusually high natural levels of arsenic in rock.
❑ Working in a job that involves arsenic production or use, such as copper or lead smelting, wood treating, or pesticide application.

How can arsenic affect my health?

Breathing high levels of inorganic arsenic can give you a sore throat or irritated lungs.

Ingesting very high levels of arsenic can result in death. Exposure to lower levels can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso.

Skin contact with inorganic arsenic may cause redness and swelling.

ToxFAQs™ Internet address is http://www.atsdr.cdc.gov/toxfaq.html

Almost nothing is known regarding health effects of organic arsenic compounds in humans. Studies in animals show that some simple organic arsenic compounds are less toxic than inorganic forms. Ingestion of methyl and dimethyl compounds can cause diarrhea and damage to the kidneys

How likely is arsenic to cause cancer?

Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. Inhalation of inorganic arsenic can cause increased risk of lung cancer. The Department of Health and Human Services (DHHS) and the EPA have determined that inorganic arsenic is a known human carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans.

How can arsenic affect children?

There is some evidence that long-term exposure to arsenic in children may result in lower IQ scores. There is also some evidence that exposure to arsenic in the womb and early childhood may increase mortality in young adults.

There is some evidence that inhaled or ingested arsenic can injure pregnant women or their unborn babies, although the studies are not definitive. Studies in animals show that large doses of arsenic that cause illness in pregnant females, can also cause low birth weight, fetal malformations, and even fetal death. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is found at low levels in breast milk.

How can families reduce the risks of exposure to arsenic?

❑ If you use arsenic-treated wood in home projects, you should wear dust masks, gloves, and protective clothing to decrease exposure to sawdust.

❑ If you live in an area with high levels of arsenic in water or soil, you should use cleaner sources of water and limit contact with soil.

❑ If you work in a job that may expose you to arsenic, be aware that you may carry arsenic home on your clothing, skin, hair, or tools. Be sure to shower and change clothes before going home.

Is there a medical test to determine whether I've been exposed to arsenic?

There are tests available to measure arsenic in your blood, urine, hair, and fingernails. The urine test is the most reliable test for arsenic exposure within the last few days. Tests on hair and fingernails can measure exposure to high levels of arsenic over the past 6-12 months. These tests can determine if you have been exposed to above-average levels of arsenic. They cannot predict whether the arsenic levels in your body will affect your health.

Has the federal government made recommendations to protect human health?

The EPA has set limits on the amount of arsenic that industrial sources can release to the environment and has restricted or cancelled many of the uses of arsenic in pesticides. EPA has set a limit of 0.01 parts per million (ppm) for arsenic in drinking water.

The Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit (PEL) of 10 micrograms of arsenic per cubic meter of workplace air (10 $\mu g/m^3$) for 8 hour shifts and 40 hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Arsenic (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-800-232-4636, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

BARIUM AND COMPOUNDS

CAS # 7440-39-3

Division of Toxicology and Environmental Medicine ToxFAQs™ **August 2007**

This fact sheet answers the most frequently asked health questions (FAQs) about barium and barium compounds. For more information, call the ATSDR Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because these substances may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to barium occurs mostly in the workplace or from drinking contaminated water. Ingesting drinking water containing levels of barium above the EPA drinking water guidelines for relatively short periods of time can cause gastrointestinal disturbances and muscle weakness. Ingesting high levels for a long time can damage the kidneys. Barium and barium compounds have been found in at least 798 of the 1,684 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is barium?

Barium is a silvery-white metal which exists in nature only in ores containing mixtures of elements. It combines with other chemicals such as sulfur or carbon and oxygen to form barium compounds.

Barium compounds are used by the oil and gas industries to make drilling muds. Drilling muds make it easier to drill through rock by keeping the drill bit lubricated. They are also used to make paint, bricks, ceramics, glass, and rubber.

Barium sulfate is sometimes used by doctors to perform medical tests and to take x-rays of the gastrointestinal tract.

What happens to barium when it enters the environment?

❏ Barium gets into the air during the mining, refining, and production of barium compounds, and from the burning of coal and oil.
❏ The length of time that barium will last in air, land, water, or sediments depends on the form of barium released.
❏ Barium compounds, such as barium sulfate and barium carbonate, which do not dissolve well in water, can last a long time in the environment.
❏ Barium compounds, such as barium chloride, barium nitrate, or barium hydroxide, that dissolve easily in water usually do not last in these forms for a long time in the environment. The barium in these compounds that is dissolved in water quickly combines with sulfate or carbonate that are naturally found in water and become the longer lasting forms (barium sulfate and barium carbonate).
❏ Fish and aquatic organisms can accumulate barium.

How might I be exposed to barium?

❏ Ingesting small amounts present in your food and water or breathing air containing very low levels of barium.
❏ Living in areas with unusually high natural levels of barium in the drinking water.
❏ Working in a job that involves barium production or use.
❏ Living or working near waste sites where barium has been disposed of.

How can barium affect my health?

The health effects of the different barium compounds depend on how well the compound dissolves in water or in the stomach contents. Barium compounds that do not dissolve well, such as barium sulfate, are not generally harmful.

BARIUM AND COMPOUNDS
CAS # 7440-39-3

Barium has been found to potentially cause gastrointestinal disturbances and muscular weakness when people are exposed to it at levels above the EPA drinking water standards for relatively short periods of time. Some people who eat or drink amounts of barium above background levels found in food and water for a short period may experience vomiting, abdominal cramps, diarrhea, difficulties in breathing, increased or decreased blood pressure, numbness around the face, and muscle weakness. Eating or drinking very large amounts of barium compounds that easily dissolve can cause changes in heart rhythm or paralysis and possibly death. Animals that drank barium over long periods had damage to the kidneys, decreases in body weight, and some died.

How likely is barium to cause cancer?

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified barium as to its carcinogenicity. The EPA has determined that barium is not likely to be carcinogenic to humans following ingestion and that there is insufficient information to determine whether it will be carcinogenic to humans following inhalation exposure.

How can barium affect children?

We do not know whether children will be more or less sensitive than adults to barium toxicity. A study in rats that swallowed barium found a decrease in newborn body weight; we do not know if a similar effect would be seen in humans.

How can families reduce the risks of exposure to barium?

The greatest potential source of barium exposure is through food and drinking water. However, the amount of barium in foods and drinking water are typically too low to be of concern.

Is there a medical test to determine whether I've been exposed to barium?

There is no routine medical test to determine whether you have been exposed to barium. Doctors can measure barium in body tissues and fluids, such as bones, blood, urine, and feces, using very complex instruments. These tests cannot be used to predict the extent of the exposure or potential health effects.

The geometric mean barium level measured in the U.S. general population aged 6 and older is reported by the Centers for Disease Control and Prevention (CDC) as 1.44 μg/g creatinine (measured in urine).

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 2.0 milligrams of barium per liter of drinking water (2.0 mg/L), which is the same as 2 ppm.

The Occupational Safety and Health Administration (OSHA) has set Permissible Exposure Limits (PELs) of 0.5 milligrams of soluble barium compounds per cubic meter of workplace air (0.5 mg/m^3) for 8 hour shifts and 40 hour work weeks. The OSHA limits for barium sulfate dust are 15 mg/m^3 of total dust and 5 mg/m^3 for respirable fraction.

The National Institute for Occupational Safety and Health (NIOSH) has set Recommended Exposure Limits (RELs) of 0.5 mg/m^3 for soluble barium compounds. The NIOSH has set RELs of 10 mg/m^3 (total dust) for barium sulfate and 5 mg/m^3 (respirable fraction).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Barium and Compounds (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-800-232-4636, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Cadmium- ToxFAQs™

CAS # 7440-43-9

This fact sheet answers the most frequently asked health questions (FAQs) about cadmium. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to cadmium happens mostly in the workplace where cadmium products are made. The general population is exposed from breathing cigarette smoke or eating cadmium contaminated foods. Cadmium damages the kidneys, lungs, and bones. Cadmium has been found in at least 1,014 of the 1,669 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What is cadmium?

Cadmium is a natural element in the earth's crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide).

All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics.

What happens to cadmium when it enters the environment?

- Cadmium enters soil, water, and air from mining, industry, and burning coal and household wastes.
- Cadmium does not break down in the environment, but can change forms.
- Cadmium particles in air can travel long distances before falling to the ground or water.
- Some forms of cadmium dissolve in water.
- Cadmium binds strongly to soil particles.
- Fish, plants, and animals take up cadmium from the environment.

How might I be exposed to cadmium?

- Eating foods containing cadmium; low levels are found in all foods (highest levels are found in leafy vegetables, grains, legumes, and kidney meat).
- Smoking cigarettes or breathing cigarette smoke.
- Breathing contaminated workplace air.
- Drinking contaminated water.
- Living near industrial facilities which release cadmium into the air.

How can cadmium affect my health?

Breathing high levels of cadmium can severely damage the lungs. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea.

Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones.

How likely is cadmium to cause cancer?

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have determined that cadmium and cadmium compounds are human carcinogens. The EPA determined that cadmium is a probable human carcinogen (group B1).

Cadmium

CAS # 7440-43-9

How can cadmium affect children?

The health effects in children are expected to be similar to the effects seen in adults (kidney and lung damage depending on the route of exposure).

A few studies in animals indicate that younger animals absorb more cadmium than adults. Animal studies also indicate that the young are more susceptible than adults to a loss of bone and decreased bone strength from exposure to cadmium.

We don't know if cadmium causes birth defects in people. Studies in animals exposed to high levels of cadmium during pregnancy have resulted in harmful effects to the young. Young animals exposed to cadmium before birth have shown effects on behavior and learning. There is also some information from animal studies that high enough exposures to cadmium before birth can reduce body weights and affect the skeleton in the developing young.

How can families reduce the risk of exposure to cadmium?

- Do not allow children to play with batteries. Dispose of nickel-cadmium batteries properly.
- Cadmium is a component of tobacco smoke. Avoid smoking and smoking in enclosed spaces like inside the home or car in order to limit exposure to children and other family members.
- If you work with cadmium, use all safety precautions to avoid carrying cadmium-containing dust home from work on your clothing, skin, hair, or tools.
- A balanced diet can reduce the amount of cadmium taken into the body from food and drink.

Is there a medical test to determine whether I've been exposed to cadmium?

Cadmium can be measured in blood, urine, hair, or nails. Urinary cadmium has been shown to accurately reflect the amount of cadmium in the body.

The amount of cadmium in your blood shows your recent exposure to cadmium. The amount of cadmium in your urine shows both your recent and your past exposure.

Has the federal government made recommendations to protect human health?

The EPA has determined that exposure to cadmium in drinking water at concentrations of 0.04 milligrams per liter (0.04 mg/L) for up to 10 days is not expected to cause any adverse effects in a child.

The EPA has determined that lifetime exposure to 0.005 mg/L cadmium is not expected to cause any adverse effects.

The Food and Drug Administration (FDA) has determined that the cadmium concentration in bottled drinking water should not exceed 0.005 mg/L.

The Occupational Health and Safety Administration (OSHA) has limited workers' exposure to an average of 5 $\mu g/m^3$ for an 8-hour workday, 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological Profile for Cadmium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Chromium - ToxFAQs™

CAS # 7440-47-3

This fact sheet answers the most frequently asked health questions (FAQs) about chromium. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to chromium occurs from ingesting contaminated food or drinking water or breathing contaminated workplace air. Chromium(VI) at high levels can damage the nose and cause cancer. Ingesting high levels of chromium(VI) may result in anemia or damage to the stomach or intestines. Chromium(III) is an essential nutrient. Chromium has been found in at least 1,127 of the 1,669 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What is chromium?

Chromium is a naturally occurring element found in rocks, animals, plants, and soil. It can exist in several different forms. Depending on the form it takes, it can be a liquid, solid, or gas. The most common forms are chromium(0), chromium(III), and chromium(VI). No taste or odor is associated with chromium compounds.

The metal chromium, which is the chromium(0) form, is used for making steel. Chromium(VI) and chromium(III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

What happens to chromium when it enters the environment?

- Chromium can be found in air, soil, and water after release from the manufacture, use, and disposal of chromium-based products, and during the manufacturing process.
- Chromium does not usually remain in the atmosphere, but is deposited into the soil and water.
- Chromium can easily change from one form to another in water and soil, depending on the conditions present.
- Fish do not accumulate much chromium in their bodies from water.

How might I be exposed to chromium?

- Eating food containing chromium(III).
- Breathing contaminated workplace air or skin contact during use in the workplace.
- Drinking contaminated well water.
- Living near uncontrolled hazardous waste sites containing chromium or industries that use chromium.

How can chromium affect my health?

Chromium(III) is an essential nutrient that helps the body use sugar, protein, and fat.

Breathing high levels of chromium(VI) can cause irritation to the lining of the nose, nose ulcers, runny nose, and breathing problems, such as asthma, cough, shortness of breath, or wheezing. The concentrations of chromium in air that can cause these effects may be different for different types of chromium compounds, with effects occurring at much lower concentrations for chromium(VI) compared to chromium(III).

The main health problems seen in animals following ingestion of chromium(VI) compounds are irritation and ulcers in the stomach and small intestine and anemia. Chromium(III) compounds are much less toxic and do not appear to cause these problems.

Sperm damage and damage to the male reproductive system have also been seen in laboratory animals exposed to chromium(VI).

Skin contact with certain chromium(VI) compounds can cause skin ulcers. Some people are extremely sensitive tochromium(VI) or chromium(III). Allergic reactions consisting of severe redness and swelling of the skin have been noted.

Chromium

CAS # 7440-47-3

How likely is chromium to cause cancer?

The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have determined that chromium(VI) compounds are known human carcinogens.

In workers, inhalation of chromium(VI) has been shown to cause lung cancer. Chromium(VI) also causes lung cancer in animals. An increase in stomach tumors was observed in humans and animals exposed to chromium(VI) in drinking water.

How can chromium affect children?

It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults.

We do not know if exposure to chromium will result in birth defects or other developmental effects in people. Some developmental effects have been observed in animals exposed to chromium(VI).

How can families reduce the risk of exposure to chromium?

- Children should avoid playing in soils near uncontrolled hazardous waste sites where chromium may have been discarded.
- Chromium is a component of tobacco smoke. Avoid smoking in enclosed spaces like inside the home or car in order to limit exposure to children and other family members.
- Although chromium(III) is an essential nutrient, you should avoid excessive use of dietary supplements containing chromium.

Is there a medical test to determine whether I've been exposed to chromium?

Since chromium(III) is an essential element and naturally occurs in food, there will always be some level of chromium in your body. Chromium can be measured in hair, urine, and blood.

Higher than normal levels of chromium in blood or urine may indicate that a person has been exposed to chromium. However, increases in blood and urine chromium levels cannot be used to predict the kind of health effects that might develop from that exposure.

Has the federal government made recommendations to protect human health?

The EPA has established a maximum contaminant level of 0.1 mg/L for total chromium in drinking water.

The FDA has determined that the chromium concentration in bottled drinking water should not exceed 0.1 mg/L.

The Occupational Health and Safety Administration (OSHA) has limited workers' exposure to an average of 0.005 mg/m^3 chromium(VI), 0.5 mg/m^3 chromium(III), and 1.0 mg/m^3 chromium(0) for an 8-hour workday, 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological Profile for Chromium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

DICHLOROBENZENES

1,2-Dichlorobenzene CAS# 95-50-1
1,3-Dichlorobenzene CAS# 541-73-1
1,4-Dichlorobenzene CAS# 106-46-7

Division of Toxicology and Environmental Medicine ToxFAQs™ **August 2006**

This fact sheet answers the most frequently asked health questions (FAQs) about dichlorobenzenes. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because these substances may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to dichlorobenzenes mostly occurs from breathing indoor air or workplace air. Exposure to high levels of 1,2- or 1,4-dichlorobenzene may be very irritating to your eyes and nose and cause difficult breathing, and an upset stomach. Extremely high exposures to 1,4-dichlorobenzene can result in dizziness, headaches, and liver problems. 1,2-, 1,3-, and 1,4-Dichlorobenzenes have been identified in at least 281, 175, and 330, respectively, of the 1,662 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are dichlorobenzenes?

There are three dichlorobenzene isomers- 1,2-dichlorobenzene, 1,3-dichlorobenzene, and 1,4-dichlorobenzene. Dichlorobenzenes do not occur naturally. 1,2-Dichlorobenzene is a colorless to pale yellow liquid used to make herbicides. 1,3-Dichlorobenzene is a colorless liquid used to make herbicides, insecticides, medicine, and dyes. 1,4-Dichlorobenzene, the most important of the three chemicals, is a colorless to white solid with a strong, pungent odor. When exposed to air, it slowly changes from a solid to a vapor. Most people can smell 1,4-dichlorobenzene in the air at very low levels.

What happens to dichlorobenzenes when they enter the environment?

❑ 1,4-Dichlorobenzene enters the environment when it is used in mothballs and in toilet-deodorizer blocks. Very little enters the environment from hazardous waste sites.

❑ Some 1,2- and 1,3-dichlorobenzenes are released into the environment when used to make herbicides and when people use products that contain these chemicals.

❑ Dichlorobenzenes do not dissolve easily in water, the small amounts that enter water quickly evaporate into the air.

❑ Sometimes, dichlorobenzenes bind to soil and sediment. Dichlorobenzenes in soil usually are not easily broken down by soil organisms. Evidence suggests that plants and fish absorb dichlorobenzenes.

How might I be exposed to dichlorobenzenes?

❑ You may be exposed to 1,4-dichlorobenzene by breathing vapors from products used in the home or in buildings, such as air fresheners, mothballs, and toilet-deodorizer blocks. 1,2-dichlorobenzene and 1,3-dichlorobenzene are not found frequently in the air of homes and buildings because these chemicals are not used in household products.

❑ You may be exposed to very low levels of dichlorobenzenes in drinking water. You are not likely to be exposed to dichlorobenzenes in soil.

❑ You may also be exposed to low levels of dichlorobenzenes in beef, pork, chicken, eggs, baked goods, soft drinks, butter, peanut butter, fruits, vegetables, and fish.

How can dichlorobenzenes affect my health?

Very little is known about the health effects of 1,3-dichlorobenzene, especially in humans, but they are likely to be similar to those of 1,2- and 1,4-dichlorobenzene.

Inhaling the vapor or dusts of 1,2-dichlorobenzene and 1,4-dichlorobenzene at very high concentrations could be very irritating to your eyes and nose and cause burning and tearing

of the eyes, coughing, difficult breathing, and an upset stomach. Dizziness, headaches, and liver problems have also been observed in people exposed to very high levels of 1,4-dichlorobenzene. There is limited evidence that inhaling 1,4-dichlorobenzene may decrease lung function.

People who have eaten 1,4-dichlorobenzene products regularly for long periods (months to years) developed skin blotches and anemia. 1,4-Dichlorobenzene might cause a burning feeling in your skin if you hold mothballs or toilet-deodorizer blocks against your skin for a long time.

Breathing or eating any of the dichlorobenzenes caused harmful effects in the liver of laboratory animals. Animal studies also found that 1,2- and 1,4-dichlorobenzene caused effects in the kidneys and blood, and that 1,3-dichlorobenzene caused thyroid and pituitary effects.

How likely are dichlorobenzenes to cause cancer?

The Department of Health and Human Services (DHHS) has determined that 1,4-dichlorobenzene may reasonably be anticipated to be a carcinogen. There is no direct evidence that 1,4-dichlorobenzene can cause cancer in humans. However, animals given very high levels in water developed liver tumors. 1,2-Dichlorobenzene was not carcinogenic in laboratory animals and 1,3-dichlorobenzene has not been tested for its potential to cause cancer. Both the International Agency for Research on Cancer (IARC) and the EPA concluded that 1,2- and 1,3-dichlorobenzene are not classifiable as to human carcinogenicity.

How can dichlorobenzenes affect children?

Children who are exposed to dichlorobenzenes are likely to exhibit the same effects as adults, although this is not known for certain. Children can also be exposed to dichlorobenzenes prenatally, because all three isomers have been detected in placenta samples, as well as through breast feeding. There is no reliable evidence suggesting that dichlorobenzenes cause birth defects, although animal data raise concern for effects of 1,4-dichlorobenzene on postnatal development of the nervous system.

How can families reduce the risk of exposure to dichlorobenzenes?

Exposure of children to 1,4-dichlorobenzene can be minimized by discouraging them from playing with, swallowing, or having skin contact with products containing 1,4-dichlorobenzene. These items should be stored out of reach of young children and kept in their original containers to prevent accidental poisonings. Keep your Poison Control Center's number by the phone.

Is there a medical test to show whether I've been exposed to dichlorobenzenes?

Several tests can be used to show if you have been exposed to dichlorobenzenes. The most commonly used tests measure their dichlorophenol breakdown products in urine and blood. The presence of the dichlorophenol breakdown products in the urine indicates a person has been exposed to dichlorobenzenes within the previous day or two. Another test measures the levels of dichlorobenzenes in your blood, but this is used less often. These tests require special equipment that is not routinely available in a doctor's office, but they can be performed in a special laboratory. Neither of these tests can be used to show how high the level of dichlorobenzene exposure was or to predict whether harmful health effects will follow.

Has the federal government made recommendations to protect human health?

EPA regulates the levels of dichlorobenzenes that are allowable in drinking water. The highest level of 1,4-dichlorobenzene allowed in drinking water is 0.075 parts 1,4-dichlorobenzene per 1 million parts of water (0.075 ppm).

The Occupational Safety and Health Administration (OSHA) has set a limit for 1,4-dichlorobenzene of 75 parts 1,4-dichlorobenzene per 1 million parts of air (75 ppm) in the workplace.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological Profile for Dichlorobenzenes (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

1,2-DICHLOROETHENE
CAS # 540-59-0, 156-59-2, and 156-60-5

This fact sheet answers the most frequently asked health questions (FAQs) about 1,2-dichloroethene. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to 1,2-dichloroethene occurs mainly in workplaces where it is made or used. Breathing high levels of 1,2-dichloroethene can make you feel nauseous, drowsy, and tired. *cis*-1,2-Dichloroethene has been found in at least 146 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA). *trans*-1,2-Dichloroethene was found in at least 563 NPL sites. 1,2-Dichloroethene was found at 336 sites, but the isomer (*cis*- or *trans*-) was not specified.

What is 1,2-dichloroethene?

(Pronounced 1,2-dī-klôrʹ ō-ĕthʹēn)

1,2-Dichloroethene, also called 1,2-dichloroethylene, is a highly flammable, colorless liquid with a sharp, harsh odor. It is used to produce solvents and in chemical mixtures. You can smell very small amounts of 1,2-dichloroethene in air (about 17 parts of 1,2-dichloroethene per million parts of air [17 ppm]).

There are two forms of 1,2-dichloroethene; one is called *cis*-1,2-dichloroethene and the other is called *trans*-1,2-dichloroethene. Sometimes both forms are present as a mixture.

What happens to 1,2-dichloroethene when it enters the environment?

- 1,2-Dichloroethene evaporates rapidly into air.
- In the air, it takes about 5-12 days for half of it to break down.
- Most 1,2-dichloroethene in the soil surface or bodies of water will evaporate into air.
- 1,2-Dichloroethene can travel through soil or dissolve in water in the soil. It is possible that it can contaminate groundwater.
- In groundwater, it takes about 13-48 weeks to break down.
- There is a slight chance that 1,2-dichloroethene will break down into vinyl chloride, a different chemical which is believed to be more toxic than 1,2-dichloroethene.

How might I be exposed to 1,2-dichloroethene?

- Breathing 1,2-dichloroethene that has leaked from hazardous waste sites and landfills.
- Drinking contaminated tap water or breathing vapors from contaminated water while cooking, bathing, or washing dishes.
- Breathing 1,2-dichloroethene, touching it, or touching contaminated materials in the workplace.

How can 1,2-dichloroethene affect my health?

Breathing high levels of 1,2-dichloroethene can make you feel nauseous, drowsy, and tired; breathing very high levels can kill you.

When animals breathed high levels of *trans*-1,2-dichloroethene for short or longer periods of time, their livers and lungs were damaged and the effects were more severe with longer exposure times. Animals that breathed very high

levels of *trans*-1,2-dichloroethene had damaged hearts.

Animals that ingested extremely high doses of *cis*- or *trans*-1,2-dichloroethene died.

Lower doses of *cis*-1,2-dichloroethene caused effects on the blood, such as decreased numbers of red blood cells, and also effects on the liver.

The long-term (365 days or longer) human health effects after exposure to low concentrations of 1,2-dichloroethene aren't known. One animal study suggested that an exposed fetus may not grow as quickly as one that hasn't been exposed.

Exposure to 1,2-dichloroethene hasn't been shown to affect fertility in people or animals.

How likely is 1,2-dichloroethene to cause cancer?

The EPA has determined that *cis*-1,2-dichloroethene is not classifiable as to its human carcinogenicity.

No EPA cancer classification is available for *trans*-1,2-dichloroethene.

Is there a medical test to show whether I've been exposed to 1,2-dichloroethene?

Tests are available to measure concentrations of the breakdown products of 1,2-dichloroethene in blood, urine, and tissues. However, these tests aren't used routinely to determine whether a person has been exposed to this compound. This is because after you are exposed to 1,2-dichloroethene, the breakdown products in your body that are detected with these tests may be the same as those that come from exposure to other chemicals. These tests aren't available in most doctors' offices, but can be done at special laboratories that have the right equipment.

Has the federal government made recommendations to protect human health?

The EPA has set the maximum allowable level of *cis*-1,2-dichloroethene in drinking water at 0.07 milligrams per liter of water (0.07 mg/L) and *trans*-1,2-dichloroethene at 0.1 mg/L.

The EPA requires that any spills or accidental release of 1,000 pounds or more of 1,2-dichloroethene must be reported to the EPA.

The Occupational Health Safety and Health Administration (OSHA) has set the maximum allowable amount of 1,2-dichloroethene in workroom air during an 8-hour workday in a 40-hour workweek at 200 parts of 1,2-dichloroethene per million parts of air (200 ppm).

Glossary

Carcinogenicity: Ability of a substance to cause cancer.

CAS: Chemical Abstracts Service.

Fertility: Ability to reproduce.

Ingest: To eat or drink something.

Milligram (mg): One thousandth of a gram.

ppm: Parts per million.

Solvent: A chemical that can dissolve other substances.

References

This ToxFAQs information is taken from the 1996 Toxicological Profile for 1,2-Dichloroethene produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Public Health Service in Atlanta, GA.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Lead – ToxFAQs™

CAS # 7439-92-1

This fact sheet answers the most frequently asked health questions (FAQs) about lead. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system. Lead has been found in at least 1,272 of the 1,684 National Priority List (NPL) sites identified by the Environmental Protection Agency (EPA).

What is lead?

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing.

Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. The use of lead as an additive to gasoline was banned in 1996 in the United States.

What happens to lead when it enters the environment?

- Lead itself does not break down, but lead compounds are changed by sunlight, air, and water.
- When lead is released to the air, it may travel long distances before settling to the ground.
- Once lead falls onto soil, it usually sticks to soil particles.
- Movement of lead from soil into groundwater will depend on the type of lead compound and the characteristics of the soil.

How might I be exposed to lead?

- Eating food or drinking water that contains lead. Water pipes in some older homes may contain lead solder. Lead can leach out into the water.
- Spending time in areas where lead-based paints have been used and are deteriorating. Deteriorating lead paint can contribute to lead dust.
- Working in a job where lead is used or engaging in certain hobbies in which lead is used, such as making stained glass.
- Using health-care products or folk remedies that contain lead.

How can lead affect my health?

The effects of lead are the same whether it enters the body through breathing or swallowing. Lead can affect almost every organ and system in your body. The main target for lead toxicity is the nervous system, both in adults and children. Long-term exposure of adults can result in decreased performance in some tests that measure functions of the nervous system. It may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people and can cause anemia. Exposure to high lead levels can severely damage the brain and kidneys in adults or children and ultimately cause death. In pregnant women, high-levels of exposure to lead may cause miscarriage. High-level exposure in men can damage the organs responsible for sperm production.

How likely is lead to cause cancer?

We have no conclusive proof that lead causes cancer in humans. Kidney tumors have developed in rats and mice that had been given large doses of some kind of lead compounds. The Department of Health and Human Services (DHHS) has determined that lead and lead compounds are reasonably anticipated to be human carcinogens and the EPA has determined that lead is a probable human carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic lead is probably carcinogenic to humans and that there is insufficient information to determine whether organic lead compounds will cause cancer in humans.

Lead

CAS # 7439-92-1

How can lead affect children?

Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint, or swallowing house dust or soil that contains lead.

Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth.

Exposure to lead is more dangerous for young and unborn children. Unborn children can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead. Some of these effects may persist beyond childhood.

How can families reduce the risks of exposure to lead?

- Avoid exposure to sources of lead.
- Do not allow children to chew or mouth surfaces that may have been painted with lead-based paint.
- If you have a water lead problem, run or flush water that has been standing overnight before drinking or cooking with it.
- Some types of paints and pigments that are used as make-up or hair coloring contain lead. Keep these kinds of products away from children.
- If your home contains lead-based paint or you live in an area contaminated with lead, wash children's hands and faces often to remove lead dusts and soil, and regularly clean the house of dust and tracked in soil.

Is there a medical test to determine whether I've been exposed to lead?

A blood test is available to measure the amount of lead in your blood and to estimate the amount of your recent exposure to lead. Blood tests are commonly used to screen children for lead poisoning. Lead in teeth or bones can be measured by X-ray techniques, but these methods are not widely available. Exposure to lead also can be evaluated by measuring erythrocyte protoporphyrin (EP) in blood samples. EP is a part of red blood cells known to increase when the amount of lead in the blood is high. However, the EP level is not sensitive enough to identify children with elevated blood lead levels below about 25 micrograms per deciliter (µg/dL). These tests usually require special analytical equipment that is not available in a doctor's office. However, your doctor can draw blood samples and send them to appropriate laboratories for analysis.

Has the federal government made recommendations to protect human health?

The Centers for Disease Control and Prevention (CDC) recommends that states test children at ages 1 and 2 years. Children should be tested at ages 3–6 years if they have never been tested for lead, if they receive services from public assistance programs for the poor such as Medicaid or the Supplemental Food Program for Women, Infants, and Children, if they live in a building or frequently visit a house built before 1950; if they visit a home (house or apartment) built before 1978 that has been recently remodeled; and/or if they have a brother, sister, or playmate who has had lead poisoning. CDC has updated its recommendations on children's blood lead levels. Experts now use an upper reference level value of 97.5% of the population distribution for children's blood lead. In 2012-2015, the value to identify children with blood lead levels that are much higher than most children have, is 5 micrograms per deciliter (µg/dL). EPA limits lead in drinking water to 15 µg per liter.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for lead (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636.

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Mercury - ToxFAQs™

CAS # 7439-97-6

This fact sheet answers the most frequently asked health questions (FAQs) about mercury. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to mercury occurs from breathing contaminated air, ingesting contaminated water and food, and having dental and medical treatments. Mercury, at high levels, may damage the brain, kidneys, and developing fetus. This chemical has been found in at least 714 of 1,467 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What is mercury?

Mercury is a naturally occurring metal which has several forms. The metallic mercury is a shiny, silver-white, odorless liquid. If heated, it is a colorless, odorless gas.

Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts," which are usually white powders or crystals. Mercury also combines with carbon to make organic mercury compounds. The most common one, methylmercury, is produced mainly by microscopic organisms in the water and soil. More mercury in the environment can increase the amounts of methylmercury that these small organisms make.

Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, some dental fillings, and batteries. Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments.

What happens to mercury when it enters the environment?

- Inorganic mercury (metallic mercury and inorganic mercury compounds) enters the air from mining ore deposits, burning coal and waste, and from manufacturing plants.
- It enters the water or soil from natural deposits, disposal of wastes, and volcanic activity.
- Methylmercury may be formed in water and soil by small organisms called bacteria.
- Methylmercury builds up in the tissues of fish. Larger and older fish tend to have the highest levels of mercury.

How might I be exposed to mercury?

- Eating fish or shellfish contaminated with methylmercury.
- Breathing vapors in air from spills, incinerators, and industries that burn mercury-containing fossil fuels.
- Release of mercury from dental work and medical treatments.
- Breathing contaminated workplace air or skin contact during use in the workplace.
- Practicing rituals that include mercury.

How can mercury affect my health?

The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems.

Short-term exposure to high levels of metallic mercury vapors may cause effects including lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

Mercury

CAS # 7439-97-6

How likely is mercury to cause cancer?

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has caused kidney tumors in male mice. The EPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

How can mercury affect children?

Very young children are more sensitive to mercury than adults. Mercury in the mother's body passes to the fetus and may accumulate there, possibly causing damage to the developing nervous system. It can also pass to a nursing infant through breast milk. However, the benefits of breast feeding may be greater than the possible adverse effects of mercury in breast milk.

Mercury's harmful effects that may affect the fetus include brain damage, mental retardation, incoordination, blindness, seizures, and inability to speak. Children poisoned by mercury may develop problems of their nervous and digestive systems, and kidney damage.

How can families reduce the risk of exposure to mercury?

Carefully handle and dispose of products that contain mercury, such as thermometers or fluorescent light bulbs. Do not vacuum up spilled mercury, because it will vaporize and increase exposure. If a large amount of mercury has been spilled, contact your health department. Teach children not to play with shiny, silver liquids.

Properly dispose of older medicines that contain mercury. Keep all mercury-containing medicines away from children.

Pregnant women and children should keep away from rooms where liquid mercury has been used.

Learn about wildlife and fish advisories in your area from your public health or natural resources department.

Is there a medical test to determine whether I've been exposed to mercury?

Tests are available to measure mercury levels in the body. Blood or urine samples are used to test for exposure to metallic mercury and to inorganic forms of mercury. Mercury in whole blood or in scalp hair is measured to determine exposure to methylmercury. Your doctor can take samples and send them to a testing laboratory.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 2 parts of mercury per billion parts of drinking water (2 ppb).

The Food and Drug Administration (FDA) has set a maximum permissible level of 1 part of methylmercury in a million parts of seafood (1 ppm).

The Occupational Safety and Health Administration (OSHA) has set limits of 0.1 milligram of organic mercury per cubic meter of workplace air (0.1 mg/m^3) and 0.05 mg/m^3 of metallic mercury vapor for 8-hour shifts and 40-hour work weeks.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636.

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Polychlorinated Biphenyls - ToxFAQs™

This fact sheet answers the most frequently asked health questions (FAQs) about polychlorinated biphenyls. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Polychlorinated biphenyls (PCBs) are a mixture of individual chemicals which are no longer produced in the United States, but are still found in the environment. Health effects that have been associated with exposure to PCBs include acne-like skin conditions in adults and neurobehavioral and immunological changes in children. PCBs are known to cause cancer in animals. PCBs have been found in at least 500 of the 1,598 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What are polychlorinated biphenyls?

Polychlorinated biphenyls are mixtures of up to 209 individual chlorinated compounds (known as congeners). There are no known natural sources of PCBs. PCBs are either oily liquids or solids that are colorless to light yellow. Some PCBs can exist as a vapor in air. PCBs have no known smell or taste. Many commercial PCB mixtures are known in the U.S. by the trade name Aroclor.

PCBs have been used as coolants and lubricants in transformers, capacitors, and other electrical equipment because they don't burn easily and are good insulators. The manufacture of PCBs was stopped in the U.S. in 1977 because of evidence they build up in the environment and can cause harmful health effects. Products made before 1977 that may contain PCBs include old fluorescent lighting fixtures and electrical devices containing PCB capacitors, and old microscope and hydraulic oils.

What happens to PCBs when they enter the environment?

- PCBs entered the air, water, and soil during their manufacture, use, and disposal; from accidental spills and leaks during their transport; and from leaks or fires in products containing PCBs.
- PCBs can still be released to the environment from hazardous waste sites; illegal or improper disposal of industrial wastes and consumer products; leaks from old electrical transformers containing PCBs; and burning of some wastes in incinerators.
- PCBs do not readily break down in the environment and thus may remain there for very long periods of time. PCBs can travel long distances in the air and be deposited in areas far away from where they were released. In water, a small amount of PCBs may remain dissolved, but most stick to organic particles and bottom sediments. PCBs also bind strongly to soil.
- PCBs are taken up by small organisms and fish in water. They are also taken up by other animals that eat these aquatic animals as food. PCBs accumulate in fish and marine mammals, reaching levels that may be many thousands of times higher than in water.

How might I be exposed to PCBs?

- Using old fluorescent lighting fixtures and electrical devices and appliances, such as television sets and refrigerators, that were made 30 or more years ago. These items may leak small amounts of PCBs into the air when they get hot during operation, and could be a source of skin exposure.
- Eating contaminated food. The main dietary sources of PCBs are fish (especially sportfish caught in contaminated lakes or rivers), meat, and dairy products.
- Breathing air near hazardous waste sites and drinking contaminated well water.
- In the workplace during repair and maintenance of PCB transformers; accidents, fires or spills involving transformers, fluorescent lights, and other old electrical devices; and disposal of PCB materials.

How can PCBs affect my health?

The most commonly observed health effects in people exposed to large amounts of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage. PCB exposures in the general population are not likely to result in skin and liver effects. Most of the studies of health effects of PCBs in the general population examined children of mothers who were exposed to PCBs.

Animals that ate food containing large amounts of PCBs for short periods of time had mild liver damage and some died. Animals that ate smaller amounts of PCBs in food over

several weeks or months developed various kinds of health effects, including anemia; acne-like skin conditions; and liver, stomach, and thyroid gland injuries. Other effects of PCBs in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects.

How likely are PCBs to cause cancer?

Few studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. The Department of Health and Human Services (DHHS) has concluded that PCBs may reasonably be anticipated to be carcinogens. PCBs have been classified as probably carcinogenic, and carcinogenic to humans (group 1) by the Environmental Protection Agency (EPA) and International Agency for Research on Cancer (IARC), respectively.

How can PCBs affect children?

Women who were exposed to relatively high levels of PCBs in the workplace or ate large amounts of fish contaminated with PCBs had babies that weighed slightly less than babies from women who did not have these exposures. Babies born to women who ate PCB-contaminated fish also showed abnormal responses in tests of infant behavior. Some of these behaviors, such as problems with motor skills and a decrease in short-term memory, lasted for several years. Other studies suggest that the immune system was affected in children born to and nursed by mothers exposed to increased levels of PCBs. There are no reports of structural birth defects caused by exposure to PCBs or of health effects of PCBs in older children. The most likely way infants will be exposed to PCBs is from breast milk. Transplacental transfers of PCBs were also reported In most cases, the benefits of breast-feeding outweigh any risks from exposure to PCBs in mother's milk.

How can families reduce the risks of exposure to PCBs?

- You and your children may be exposed to PCBs by eating fish or wildlife caught from contaminated locations. Certain states, Native American tribes, and U.S. territories have issued advisories to warn people about PCB-contaminated fish and fish-eating wildlife. You can reduce your family's exposure to PCBs by obeying these advisories.
- Children should be told not play with old appliances, electrical equipment, or transformers, since they may contain PCBs.
- Children should be discouraged from playing in the dirt near hazardous waste sites and in areas where there was a transformer fire. Children should also be discouraged from eating dirt and putting dirty hands, toys or other objects in their mouths, and should wash hands frequently.
- If you are exposed to PCBs in the workplace it is possible to carry them home on your clothes, body, or tools. If this is the case, you should shower and change clothing before leaving work, and your work clothes should be kept separate from other clothes and laundered separately.

Is there a medical test to show whether I've been exposed to PCBs?

Tests exist to measure levels of PCBs in your blood, body fat, and breast milk, but these are not routinely conducted. Most people normally have low levels of PCBs in their body because nearly everyone has been environmentally exposed to PCBs. The tests can show if your PCB levels are elevated, which would indicate past exposure to above-normal levels of PCBs, but cannot determine when or how long you were exposed or whether you will develop health effects.

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 0.0005 milligrams of PCBs per liter of drinking water (0.0005 mg/L). Discharges, spills or accidental releases of 1 pound or more of PCBs into the environment must be reported to the EPA. The Food and Drug Administration (FDA) requires that infant foods, eggs, milk and other dairy products, fish and shellfish, poultry and red meat contain no more than 0.2-3 parts of PCBs per million parts (0.2-3 ppm) of food. Many states have established fish and wildlife consumption advisories for PCBs.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636.

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Polycyclic Aromatic Hydrocarbons (PAHs) - ToxFAQs™

This fact sheet answers the most frequently asked health questions (FAQs) about polycyclic aromatic hydrocarbons (PAHs). For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to polycyclic aromatic hydrocarbons usually occurs by breathing air contaminated by wild fires or coal tar, or by eating foods that have been grilled. PAHs have been found in at least 600 of the 1,430 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What are polycyclic aromatic hydrocarbons?

(Pronounced pŏl′ĭ-sī′klĭk ăr′ə-măt′ĭk hī′drə-kar′bənz)

Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot.

Some PAHs are manufactured. These pure PAHs usually exist as colorless, white, or pale yellow-green solids. PAHs are found in coal tar, crude oil, creosote, and roofing tar, but a few are used in medicines or to make dyes, plastics, and pesticides.

What happens to PAHs when they enter the environment?

- PAHs enter the air mostly as releases from volcanoes, forest fires, burning coal, and automobile exhaust.
- PAHs can occur in air attached to dust particles.
- Some PAH particles can readily evaporate into the air from soil or surface waters.
- PAHs can break down by reacting with sunlight and other chemicals in the air, over a period of days to weeks.
- PAHs enter water through discharges from industrial and wastewater treatment plants.
- Most PAHs do not dissolve easily in water. They stick to solid particles and settle to the bottoms of lakes or rivers.
- Microorganisms can break down PAHs in soil or water after a period of weeks to months.
- In soils, PAHs are most likely to stick tightly to particles; certain PAHs move through soil to contaminate underground water.
- PAH contents of plants and animals may be much higher than PAH contents of soil or water in which they live.

How might I be exposed to PAHs?

- Breathing air containing PAHs in the workplace of coking, coal-tar, and asphalt production plants; smokehouses; and municipal trash incineration facilities.
- Breathing air containing PAHs from cigarette smoke, wood smoke, vehicle exhausts, asphalt roads, or agricultural burn smoke.
- Coming in contact with air, water, or soil near hazardous waste sites.
- Eating grilled or charred meats; contaminated cereals, flour, bread, vegetables, fruits, meats; and processed or pickled foods.
- Drinking contaminated water or cow's milk.
- Nursing infants of mothers living near hazardous waste sites may be exposed to PAHs through their mother's milk.

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Human Health Sciences

ATSDR

CS249955-AD

Polycyclic Aromatic Hydrocarbons

How can PAHs affect my health?

Mice that were fed high levels of one PAH during pregnancy had difficulty reproducing and so did their offspring. These offspring also had higher rates of birth defects and lower body weights. It is not known whether these effects occur in people.

Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. But these effects have not been seen in people.

How likely are PAHs to cause cancer?

The Department of Health and Human Services (DHHS) has determined that some PAHs may reasonably be expected to be carcinogens.

Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

Is there a medical test to show whether I've been exposed to PAHs?

In the body, PAHs are changed into chemicals that can attach to substances within the body. There are special tests that can detect PAHs attached to these substances in body tissues or blood. However, these tests cannot tell whether any health effects will occur or find out the extent or source of your exposure to the PAHs. The tests aren't usually available in your doctor's office because special equipment is needed to conduct them.

Has the federal government made recommendations to protect human health?

The Occupational Safety and Health Administration (OSHA) has set a limit of 0.2 milligrams of PAHs per cubic meter of air (0.2 mg/m^3). The OSHA Permissible Exposure Limit (PEL) for mineral oil mist that contains PAHs is 5 mg/m^3 averaged over an 8-hour exposure period.

The National Institute for Occupational Safety and Health (NIOSH) recommends that the average workplace air levels for coal tar products not exceed 0.1 mg/m^3 for a 10-hour workday, within a 40-hour workweek. There are other limits for workplace exposure for things that contain PAHs, such as coal, coal tar, and mineral oil.

Glossary

Carcinogen: A substance that can cause cancer.

Ingest: Take food or drink into your body.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636.

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

SILVER
CAS # 7440-22-4

This fact sheet answers the most frequently asked health questions (FAQs) about silver. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Silver is an element found naturally in the environment. At very high levels, it may cause argyria, a blue-gray discoloration of the skin and other organs. This chemical has been found in at least 27 of the 1,177 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is silver?

(Pronounced sĭl′vər)

Silver is a naturally occurring element. It is found in the environment combined with other elements such as sulfide, chloride, and nitrate. Pure silver is "silver" colored, but silver nitrate and silver chloride are powdery white and silver sulfide and silver oxide are dark-gray to black. Silver is often found as a by-product during the retrieval of copper, lead, zinc, and gold ores.

Silver is used to make jewelry, silverware, electronic equipment, and dental fillings. It is also used to make photographs, in brazing alloys and solders, to disinfect drinking water and water in swimming pools, and as an antibacterial agent. Silver has also been used in lozenges and chewing gum to help people stop smoking.

What happens to silver when is enters the environment?

- Silver may be released into the air and water through natural processes such as the weathering of rocks.
- Human activities such as the processing of ores, cement manufacture, and the burning of fossil fuel may release silver into the air.
- It may be released into water from photographic processing.
- Rain may wash silver out of soil into the groundwater.
- Silver does not appear to concentrate to a significant extent in aquatic animals.

How might I be exposed to silver?

- Breathing low levels in air.
- Swallowing it in food or drinking water.
- Carrying out activities such as jewelry-making, soldering, and photography.
- Using anti-smoking lozenges or other medicines containing it.

How can silver affect my health?

Exposure to high levels of silver for a long period of time may result in a condition called arygria, a blue-gray discoloration of the skin and other body tissues. Lower-level exposures to silver may also cause silver to be deposited in the skin and other parts of the body; however, this is not known to be harmful. Argyria is a permanent effect, but it appears to be a cosmetic problem that may not be otherwise harmful to health.

Exposure to high levels of silver in the air has resulted in breathing problems, lung and throat irritation, and stomach pains. Skin contact with silver can cause mild allergic reactions such as rash, swelling, and inflammation in some people.

Animal studies have shown that swallowing silver results in the deposit of silver in the skin. One study in mice found that the animals exposed to silver in drinking water were less active than unexposed animals.

No studies are available on whether silver affects reproduction or causes developmental problems in people.

How likely is silver to cause cancer?

No studies are available on whether silver may cause cancer in people. The only available animal studies showed both positive and negative results when silver was implanted under the skin.

The EPA has determined that silver is not classifiable as to human carcinogenicity.

Is there a medical test to show whether I've been exposed to silver?

Silver can be measured in the blood, urine, feces, and body tissues of exposed people. Silver builds up in the body, and the best way to learn if past exposure has occurred is to look for silver in samples of skin. Tests for silver are not commonly done at a doctor's office because they require special equipment. Although doctors can find out if a person has been exposed to silver by doing these tests, they cannot tell whether any health effects will occur.

Has the federal government made recommendations to protect human health?

The EPA recommends that the concentration of silver in drinking water not exceed 0.10 milligrams per liter of water (0.10 mg/L) because of the skin discoloration that may occur.

The EPA requires that spills or accidental releases of 1,000 pounds or more of silver be reported to the EPA.

The Occupational Safety and Health Administration (OSHA) limits silver in workplace air to 0.01 milligrams per cubic meter (0.01 mg/m^3) for an 8-hour workday, 40-hour workweek. The National Institute of Occupational Safety and Health (NIOSH) also recommends that workplace air contain no more that 0.01 mg/m^3 silver.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends that workplace air contain no more than 0.1 mg/m^3 silver metal and 0.01 mg/m^3 soluble silver compounds.

The federal recommendations have been updated as of July 1999.

Glossary

Carcinogenicity: Ability to cause cancer.

CAS: Chemcial Abstracts Service.

Milligram (mg): One thousandth of a gram.

National Priorities List: A list of the nation's worst hazardous waste sites.

Soluble: Capable of being dissolved in water.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological profile for silver. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone:1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Tetrachloroethylene - ToxFAQs™

CAS # 127-18-4

This fact sheet answers the most frequently asked health questions (FAQs) about tetrachloroethylene. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Tetrachloroethylene is a manufactured chemical used for dry cleaning and metal degreasing. Exposure to very high concentrations of tetrachloroethylene can cause dizziness, headaches, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death. Tetrachloroethylene has been found in at least 771 of the 1,430 National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA).

What is tetrachloroethylene?

(Pronounced tĕt'rə-klôr' ō-ĕth'ə-lēn')

Tetrachloroethylene is a manufactured chemical that is widely used for dry cleaning of fabrics and for metal-degreasing. It is also used to make other chemicals and is used in some consumer products.

Other names for tetrachloroethylene include perchloroethylene(PERC), PCE, and tetrachloroethene. It is a nonflammable liquid at room temperature. It evaporates easily into the air and has a sharp, sweet odor. Most people can smell tetrachloroethylene when it is present in the air at a level of 1 part tetrachloroethylene per million parts of air (1 ppm) or more, although some can smell it at even lower levels.

What happens to tetrachloroethylene when it enters the environment?

- Much of the tetrachloroethylene that gets into water or soil evaporates into the air.
- Microorganisms can break down some of the tetrachloroethylene in soil or underground water.
- In the air, it is broken down by sunlight into other chemicals or brought back to the soil and water by rain.
- It does not appear to collect in fish or other animals that live in water.

How might I be exposed to tetrachloroethylene?

- When you bring clothes from the dry cleaners, they will release small amounts of tetrachloroethylene into the air.
- When you drink water containing tetrachloroethylene, you are exposed to it.

How can tetrachloroethylene affect my health?

High concentrations of tetrachloroethylene (particularly in closed, poorly ventilated areas) can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death.

Irritation may result from repeated or extended skin contact with it. These symptoms occur almost entirely in work (or hobby) environments when people have been accidentally exposed to high concentrations or have intentionally used tetrachloroethylene to get a "high."

In industry, most workers are exposed to levels lower than those causing obvious nervous system effects. The health effects of breathing in air or drinking water with low levels of tetrachloroethylene are not known.

Results from some studies suggest that women who work in dry cleaning industries where exposures to tetrachloroethylene can be quite high may have more menstrual problems and spontaneous abortions than women who are not exposed. However, it is not known if tetrachloroethylene was responsible for these problems because other possible causes were not considered.

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Human Health Sciences

ATSDR

CS249955-AH

Tetrachloroethylene

CAS # 127-18-4

Results of animal studies, conducted with amounts much higher than those that most people are exposed to, show that tetrachloroethylene can cause liver and kidney damage. Exposure to very high levels of tetrachloroethylene can be toxic to the unborn pups of pregnant rats and mice. Changes in behavior were observed in the offspring of rats that breathed high levels of the chemical while they were pregnant.

How likely is tetrachloroethylene to cause cancer?

The Department of Health and Human Services (DHHS) has determined that tetrachloroethylene may reasonably be anticipated to be a carcinogen. Tetrachloroethylene has been shown to cause liver tumors in mice and kidney tumors in male rats.

Is there a medical test to show whether I've been exposed to tetrachloroethylene?

One way of testing for tetrachloroethylene exposure is to measure the amount of the chemical in the breath, much the same way breath-alcohol measurements are used to determine the amount of alcohol in the blood.

Because it is stored in the body's fat and slowly released into the bloodstream, tetrachloroethylene can be detected in the breath for weeks following a heavy exposure.

Tetrachloroethylene and trichloroacetic acid (TCA), a breakdown product of tetrachloroethylene, can be detected in the blood. These tests are relatively simple to perform. These tests aren't available at most doctors' offices, but can be per formed at special laboratories that have the right equipment.

Because exposure to other chemicals can produce the same breakdown products in the urine and blood, the tests for breakdown products cannot determine if you have been exposed to tetrachloroethylene or the other chemicals.

Has the federal government made recommendations to protect human health?

The EPA maximum contaminant level for the amount of tetrachloroethylene that can be in drinking water is 0.005 milligrams tetrachloroethylene per liter of water (0.005 mg/L).

The Occupational Safety and Health Administration (OSHA) has set a limit of 100 ppm for an 8-hour workday over a 40-hour workweek.

The National Institute for Occupational Safety and Health (NIOSH) recommends that tetrachloroethylene be handled as a potential carcinogen and recommends that levels in workplace air should be as low as possible.

Glossary

Carcinogenicity: The ability of a substance to cause cancer.

CAS: Chemical Abstracts Service.

Milligram (mg): One thousandth of a gram.

Nonflammable: Will not burn.

References

This ToxFAQs™ information is taken from the 1997 Toxicological Profile for Tetrachloroethylene (update) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Public Health Service in Atlanta, GA.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30333.

Phone: 1-800-232-4636.

ToxFAQs™ Internet address via WWW is http://www.atsdr.cdc.gov/toxfaqs/index.asp.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Basic Information about 1,2,4-Trichlorobenzene in Drinking Water

EPA regulates 1,2,4-trichlorobenzene in drinking water to protect public health. 1,2,4-Trichlorobenzene may cause health problems if present in public or private water supplies in amounts greater than the drinking water standard set by EPA.

1,2,4-Trichlorobenzene at a Glance

Maximum Contaminant Level (MCL) = 0.07 milligrams per Liter (mg/L) or 70 parts per billion (ppb)

Maximum Contaminant Level Goal (MCLG) = 0.07 mg/L or 70 ppb

Health Effects
Some people who drink water containing 1,2,4- trichlorobenzene in excess of the MCL over many years could experience changes in their adrenal glands.

Drinking Water Health Advisories provide more information on health effects

Chemical Abstract Service Registry Number
120-82-1

Sources of Contamination
Discharge from textile fishing factories

List of all Regulated Contaminants (PDF) (6 pp, 396K, About PDF)

What is 1,2,4-trichlorobenzene?

1,2,4-Trichlorobenzene is an aromatic, colorless organic liquid.

Uses for 1,2,4-trichlorobenzene.

The greatest use of 1,2,4-trichlorobenzene is primarily as a dye carriet. It is also used to make herbicides and other organic chemicals; as a solvent; in wood preservatives; in abrasives. It was once used as a soil treatment for termite control.

If you are concerned about 1,2,4-trichlorobenzene in a private well, please visit:

- EPA's private drinking water wells Web site
- Water Systems Council Web site EXIT Disclaimer

What are 1,2,4-trichlorobenzene's health effects?
Some people who drink water containing 1,2,4-trichlorobenzene well in excess of the maximum contaminant level (MCL) for many years could experience changes in their adrenal glands.

This health effects language is not intended to catalog all possible health effects for 1,2,4-trichlorobenzene. Rather, it is intended to inform consumers of some of the possible health effects associated with 1,2,4-trichlorobenzene in drinking water when the rule was finalized.

Top of page

How is 1,2,4-trichlorobenzene regulated?
In 1974, Congress passed the Safe Drinking Water Act. This law requires EPA to determine safe levels of contaminants in drinking water which do or may cause health problems. These non-enforceable health goals, based solely on possible health risks and exposure over a lifetime, are called maximum contaminant level goals (MCLG). Contaminants are any physical, chemical, biological or radiological substances or matter in water.

The MCLG for 1,2,4-trichlorobenzene is 0.07 mg/L or 70 ppb. EPA has set this level of protection based on the best available science to prevent potential health problems.

Top of page

What are EPA's drinking water regulations for 1,2,4-trichlorobenzene?
EPA has set an enforceable regulation for 1,2,4-trichlorobenzene, called a maximum contaminant level (MCL), at 0.07 mg/L or 70 ppb. MCLs are set as close to the health goals as possible, considering cost, benefits and the ability of public water systems to detect and remove contaminants using suitable treatment technologies. In this case, the MCL equals the MCLG, because analytical methods or treatment technology do not pose any limitation.

The Phase V Rule, the regulation for 1,2,4-trichlorobenzene, became effective in 1994. The Safe Drinking Water Act requires EPA to periodically review the national primary drinking water regulation for each contaminant and revise the regulation, if appropriate. EPA reviewed 1,2,4-trichlorobenzene as part of the Six Year Review and determined that the 0.07 mg/L or 70 ppb MCLG and 0.07 mg/L or 70 ppb MCL for 1,2,4-trichlorobenzene are still protective of human health.

- More information on the Six Year Review of Drinking Water Standards.

States may set more stringent drinking water MCLGs and MCLs for 1,2,4-trichlorobenzene than EPA.

Top of page

How does 1,2,4-trichlorobenzene get into my drinking water?
The major source of 1,2,4-trichlorobenzene in drinking water is discharge from textile finishing factories.

A federal law called the Emergency Planning and Community Right to Know Act

(EPCRA) requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. For more information on the uses and releases of chemicals in your state, contact the Community Right-to-Know Hotline: (800) 424-9346.

- EPA's Toxics Release Inventory (TRI) Web site provides information about the types and amounts of toxic chemicals that are released each year to the air, water, and land.

Top of page

How will I know if 1,2,4-trichlorobenzene is in my drinking water?
When routine monitoring indicates that 1,2,4-trichlorobenzene levels are above the MCL, your water supplier must take steps to reduce the amount of 1,2,4-trichlorobenzene so that it is below that level. Water suppliers must notify their customers as soon as practical, but no later than 30 days after the system learns of the violation. Additional actions, such as providing alternative drinking water supplies, may be required to prevent serious risks to public health.

- See EPA's public notification requirements for public water systems.

If your water comes from a household well, check with your health department or local water systems that use ground water for information on contaminants of concern in your area.

- For more information on wells, go to EPA's Web site on private wells.

Top of page

How will 1,2,4-trichlorobenzene be removed from my drinking water?
The following treatment method(s) have proven to be effective for removing 1,2,4-trichlorobenzene to below 0.07 mg/L or 70 ppb: granular activated carbon in combination with packed tower aeration.

Top of page

How do I learn more about my drinking water?
EPA strongly encourages people to learn more about their drinking water, and to support local efforts to protect the supply of safe drinking water and upgrade the community water system. Your water bill or telephone book's government listings are a good starting point for local information.

Contact your water utility. EPA requires all community water systems to prepare and deliver an annual consumer confidence report (CCR) (sometimes called a water quality report) for their customers by July 1 of each year. If your water provider is not a community water system, or if you have a private water supply, request a copy from a nearby community water system.

The CCR summarizes information regarding sources used (i.e., rivers, lakes, reservoirs, or aquifers), any detected contaminants, compliance and educational information.

Some water suppliers have posted their annual reports on EPA's Web site.

Other EPA Web sites

- Find an answer or ask a question about drinking water contaminants on EPA's Question and Answer Web site or call EPA's Safe Drinking Water Hotline at (800) 426-4791
- EPA's Integrated Risk Information System
- EPA's Substance Registry System

Top of page

Safewater Home | About Our Office | Publications | Questions and Answers | Links | Office of Water | En Español

EPA Home | Privacy and Security Notice | Contact Us

This document will now print as it appears on screen when you use the File » Print command.
Use View » Refresh to return to original state.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

1,1,1-TRICHLOROETHANE

CAS # 71-55-6

Division of Toxicology and Environmental Medicine ToxFAQs™ **July 2006**

This fact sheet answers the most frequently asked health questions (FAQs) about 1,1,1-trichloroethane. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to 1,1,1-trichloroethane usually occurs by breathing contaminated air. It is found in building materials, cleaning products, paints, and metal degreasing agents. You are not likely to be exposed to large enough amounts to cause adverse health effects. Inhaling high levels of 1,1,1-trichloroethane can cause you to become dizzy and lightheaded. Exposure to much higher levels can cause unconsciousness and other effects. This substance has been found in at least 823 of the 1,662 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is 1,1,1-trichloroethane?

1,1,1-Trichloroethane is a synthetic chemical that does not occur naturally in the environment. It also is known as methylchloroform, methyltrichloromethane, trichloromethylmethane, and α–trichloromethane. Its registered trade names are chloroethene NU® and Aerothene TT®.

No 1,1,1-trichloroethane is supposed to be manufactured for domestic use in the United States after January 1, 2002 because it affects the ozone layer. 1,1,1-Trichloroethane had many industrial and household uses, including use as a solvent to dissolve other substances, such as glues and paints; to remove oil or grease from manufactured metal parts; and as an ingredient of household products such as spot cleaners, glues, and aerosol sprays.

What happens to 1,1,1-trichloroethane when it enters the environment?

❑ Most of the 1,1,1-trichloroethane released into the environment enters the air, where it lasts for about 6 years.

❑ Once in the air, it can travel to the ozone layer where sunlight can break it down into chemicals that may reduce the ozone layer.

❑ Contaminated water from landfills and hazardous waste sites can contaminate surrounding soil and nearby surface water or groundwater.

❑ From lakes and rivers, most of the 1,1,1-trichloroethane evaporates quickly into the air.

❑ Water can carry 1,1,1-trichloroethane through the soil and into the groundwater where it can evaporate and pass through the soil as a gas, then be released to the air.

❑ Organisms living in soil or water may also break down 1,1,1-trichloroethane.

❑ It will not build up in plants or animals.

How might I be exposed to 1,1,1-trichloroethane?

❑ Breathing 1,1,1-trichloroethane in contaminated outdoor and indoor air. Because 1,1,1-trichloroethane was used so frequently in home and office products, you are likely to be exposed to higher levels indoors than outdoors or near hazardous waste sites. However, since 2002, 1,1,1-trichloroethane is not expected to be commonly used, and therefore, the likelihood of being exposed to it is remote.

❑ In the workplace, you could have been exposed to 1,1,1-trichloroethane while using some metal degreasing agents, paints, glues, and cleaning products.

❑ Ingesting contaminated drinking water and food.

How can 1,1,1-trichloroethane affect my health?

If you breathe air containing high levels of 1,1,1-trichloroethane for a short time, you may become dizzy and lightheaded and possibly lose your coordination. These effects rapidly disappear after you stop breathing contaminated air. If you breathe in much higher levels, you may become unconscious, your blood pressure may decrease, and your heart may stop beating. Whether breathing low levels of 1,1,1-trichloroethane for a long

time causes harmful effects is not known. Studies in animals show that breathing air that contains very high levels of 1,1,1-trichloroethane damages the breathing passages and causes mild effects in the liver, in addition to affecting the nervous system. There are no studies in humans that determine whether eating food or drinking water contaminated with 1,1,1-trichloroethane could harm health. Placing large amounts of 1,1,1-trichloroethane in the stomachs of animals has caused effects on the nervous system, mild liver damage, unconsciousness, and even death. If your skin contacts 1,1,1-trichloroethane, you might feel some irritation. Studies in animals suggest that repeated exposure of the skin might affect the liver and that very large amounts may cause death. These effects occurred only when evaporation was prevented.

How likely is 1,1,1-trichloroethane to cause cancer?

Available information does not indicate that 1,1,1-trichloroethane causes cancer. The International Agency for Research on Cancer (IARC) and the EPA have determined that 1,1,1-trichloroethane is not classifiable as to its carcinogenicity in humans.

How can 1,1,1-trichloroethane affect children?

Children exposed to large amounts of 1,1,1-trichloroethane probably would be affected in the same manner as adults. In animals, it has been shown that 1,1,1-trichloroethane can pass from the mother's blood into a fetus. When pregnant mice were exposed to high levels of 1,1,1-trichloroethane in air, their babies developed more slowly than normal and had some behavioral problems. However, whether similar effects occur in humans has not been demonstrated.

How can families reduce the risk of exposure to 1,1,1-trichloroethane?

Children can be exposed to 1,1,1-trichloroethane in household products, such as adhesives and cleaners. Parents should store household chemicals out of reach of young children to prevent accidental poisonings or skin irritation. Always store household chemicals in their original labeled containers. Never store household chemicals in containers that children would find attractive to eat or drink from, such as old soda bottles. Keep your Poison Control Center's number near the phone.

Sometimes older children sniff household chemicals in an attempt to get high. Your children may be exposed to 1,1,1-trichloroethane by inhaling products containing it. Talk with your children about the dangers of sniffing chemicals.

Is there a medical test to show whether I've been exposed to 1,1,1-trichloroethane?

Samples of your breath, blood, and urine can be tested to determine if you have recently been exposed to 1,1,1-trichloroethane. In some cases, these tests can estimate how much 1,1,1-trichloroethane has entered your body. To be of any value, samples of your breath or blood have to be taken within hours after exposure, and samples of urine have to be taken within 2 days after exposure. However, these tests will not tell you whether your health will be affected by exposure to 1,1,1-trichloroethane. The exposure tests are not routinely available in hospitals and clinics because they require special analytical equipment.

Has the federal government made recommendations to protect human health?

EPA regulates the levels of 1,1,1-trichloroethane that are allowable in drinking water. The highest level of 1,1,1-trichloroethane allowed in drinking water is 0.2 parts 1,1,1,-trichloroethane per 1 million parts of water (0.2 ppm).

The Occupational Safety and Health Administration (OSHA) has set a limit of 350 parts 1,1,1-trichloroethane per 1 million parts of air (350 ppm) in the workplace.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological Profile for 1,1,1-Trichloroethane (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Trichloroethylene - ToxFAQs™

CAS # 79-01-6

This fact sheet answers the most frequently asked health questions (FAQs) about trichloroethylene. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Trichloroethylene is used as a solvent for cleaning metal parts. Exposure to very high concentrations of trichloroethylene can cause dizziness, headaches, sleepiness, incoordination, confusion, nausea, unconsciousness, and even death. The Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) classify trichloroethylene as a human carcinogen. Trichloroethylene has been found in at least 1,045 of the 1,699 National Priorities List sites identified by the EPA.

What is trichloroethylene?

Trichloroethylene is a colorless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is nonflammable and has a sweet odor.

The two major uses of trichloroethylene are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. Trichloroethylene was once used as an anesthetic for surgery.

What happens to trichloroethylene when it enters the environment?

- Trichloroethylene can be released to air, water, and soil at places where it is produced or used.
- Trichloroethylene is broken down quickly in air.
- Trichloroethylene breaks down very slowly in soil and water and is removed mostly through evaporation to air.
- It is expected to remain in groundwater for long time since it is not able to evaporate.
- Trichloroethylene does not build up significantly in plants or animals.

How might I be exposed to trichloroethylene?

- Breathing trichloroethylene in contaminated air.
- Drinking contaminated water.
- Workers at facilities using this substance for metal degreasing are exposed to higher levels of trichloroethylene.
- If you live near such a facility or near a hazardous waste site containing trichloroethylene, you may also have higher exposure to this substance.

How can trichloroethylene affect my health?

Exposure to moderate amounts of trichloroethylene may cause headaches, dizziness, and sleepiness; large amounts may cause coma and even death. Eating or breathing high levels of trichloro¬ethylene may damage some of the nerves in the face. Exposure to high levels can also result in changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Skin contact with concentrated solutions of trichloroethylene can cause skin rashes.

There is some evidence exposure to trichloroethylene in the work place may cause scleroderma (a systemic autoimmune disease) in some people. Some men occupationally-exposed to trichloroethylene and other chemicals showed decreases in sex drive, sperm quality, and reproductive hormone levels.

How likely is trichloroethylene to cause cancer?

There is strong evidence that trichloroethylene can cause kidney cancer in people and some evidence for trichloroethylene-induced liver cancer and malignant lymphoma. Lifetime exposure to trichloroethylene resulted in increased liver cancer in mice and increased kidney cancer and testicular cancer in rats.

The IARC and the EPA determined that there is convincing evidence that trichloroethylene exposure can cause kidney cancer. The National Toxicology Program (NTP) is recommending a change in cancer classification to "known human carcinogen" http://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf.

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Health Human Sciences

ATSDR

CS256651A

Trichloroethylene

CAS # 79-01-6

How can trichloroethylene affect children?

It is not known whether children are more susceptible than adults to the effects of trichloroethylene.

Some human studies indicate that trichloroethylene may cause developmental effects such as spontaneous abortion, congenital heart defects, central nervous system defects, and small birth weight. However, these people were exposed to other chemicals as well.

In some animal studies, exposure to trichloroethylene during development caused decreases in body weight, increases in heart defects, changes to the developing nervous system, and effects on the immune system.

How can families reduce the risk of exposure to trichloroethylene?

- Avoid drinking water from sources that are known to be contaminated with trichloroethylene. Use bottled water if you have concerns about the presence of chemicals in your tap water. You may also contact local drinking water authorities and follow their advice.
- Discourage your children from putting objects in their mouths. Make sure that they wash their hands frequently and before eating.
- Prevent children from playing in dirt or eating dirt if you live near a waste site that has trichloroethylene.
- Trichloroethylene is used in many industrial products. Follow instructions on product labels to minimize exposure to trichloroethylene.

Is there a medical test to show whether I've been exposed to trichloroethylene?

Trichloroethylene and its breakdown products (metabolites) can be measured in blood and urine. However, the detection of trichloroethylene or its metabolites cannot predict the kind of health effects that might develop from that exposure. Because trichloroethylene and its metabolites leave the body fairly rapidly, the tests need to be conducted within days after exposure.

Has the federal government made recommendations to protect human health?

The EPA set a maximum contaminant goal (MCL) of 0.005 milligrams per liter (mg/L; 5 ppb) as a national primary drinking standard for trichloroethylene.

The Occupational Safety and Health Administration (OSHA) set a permissible exposure limit (PEL) of 100 ppm for trichloroethylene in air averaged over an 8-hour work day, an acceptable ceiling concentration of 200 ppm provided the 8 hour PEL is not exceeded, and an acceptable maximum peak of 300 ppm for a maximum duration of 5 minutes in any 2 hours.

The National Institute for Occupational Safety and Health (NIOSH) considers trichloroethylene to be a potential occupational carcinogen and established a recommended exposure limit (REL) of 2 ppm (as a 60-minute ceiling) during its use as an anesthetic agent and 25 ppm (as a 10-hour TWA) during all other exposures.

References

This ToxFAQs™ information is taken from the 2014 Toxicological Profile for Trichloroethylene (Draft for Public Comment) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30329-4027.

Phone: 1-800-232-4636.

ToxFAQs™ Ion the web: www.atsdr.cdc.gov/toxFAQs.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

VOLATILE/EXTRACTABLE PETROLEUM HYDROCARBON FRACTIONS - VPH/EPH

The following toxicity profile was compiled using information taken directly from MA DEP documents (MA DEP, 1994, 2002, and 2003). Secondary references are cited to the MA DEP document where the information was presented.

GENERAL BACKGROUND INFORMATION

Petroleum products are a complex and highly variable mixture of hundreds of individual hydrocarbon compounds. Industry specifications for refined products, such as gasoline and diesel fuel, are based upon physical and performance-based criteria rather than on a specific chemical formulation. The compositions of petroleum products released to the environment are therefore complex and variable, and are a function of the origin and chemistry of the parent crude oil, the refining and blending processes, and the use of performance-enhancing additives. Once released to the environment, the chemistry of a petroleum product is further altered by contaminant fate and transport processes, such as leaching, volatilization, and biodegradation (MA DEP, 2002).

Although little toxicological data are available for the vast majority of petroleum constituents, it is possible to make some broad observations and conclusions:

- Petroleum products are comprised mainly of aliphatic and aromatic hydrocarbon compounds
- Aromatic hydrocarbons appear to be more toxic than aliphatic compounds; and
- The toxicity of aliphatic compounds appears to be related to their carbon number/molecular weights.

These three precepts are the foundation of the VPH/EPH approach. Under this approach, the non-cancer toxicity of petroleum-contaminated media is established by determining the collective concentrations of specified ranges of aliphatic and aromatic hydrocarbons and assigning a toxicity value (e.g., reference dose) to each range. Well-characterized compounds within specified ranges are selected as "surrogate" indicators to define the toxicity of the entire range (e.g., every aliphatic compound having between 5 and 8 carbon atoms (C_5-C_8 aliphatic hydrocarbons) are assumed to be as toxic as n-hexane). Cancer effects are evaluated separately by the identification and quantitation of those specific hydrocarbons, such as benzene and certain polycyclic aromatic hydrocarbons (PAHs), which are designated carcinogens. (MA DEP, 2002).

HUMAN TOXICOLOGICAL PROFILE - General

Inhaled or ingested volatile hydrocarbons have both general and specific effects. Many organic solvents, including petroleum hydrocarbons, have the potential on acute high-level vapor exposure to cause central nervous system (CNS) disturbances like disorientation, euphoria, giddiness, and confusion; progressing to unconsciousness, paralysis, convulsion, and death from respiratory or cardiac arrest (Browning, 1965 in MA DEP, 2003). These effects have been observed with aliphatic and aromatic compounds found within the C_5 - C_9 (aliphatics) and C_6 - C_{10} (aromatics) carbon ranges.

The acute narcotic effects of the volatile hydrocarbons result from direct chemical action. The similarity of CNS disruption produced by hydrocarbons of diverse structures suggests that these effects result from a common process, which is physical interaction of the solvents with the cells of the CNS (Andrews and Snyder, 1991 in MA DEP, 2003). For example, interaction of the lipid-soluble hydrocarbons with the synaptosomal membranes causes CNS toxicities. The potency of the CNS effects depends on the structure of the individual hydrocarbon molecule.

Other non-specific effects of hydrocarbons are exhibited after prolonged exposure to these agents. The nonspecific effects observed in animals and humans are neurobehavioral toxicities. The neurobehavioral effects are manifested as sensory, cognitive, affective, and motor abnormalities. There is some evidence suggesting that the mechanism of the behavioral effects is alterations in the utilization and turnover of biogenic amines in the brain. These effects occur at lower hydrocarbon concentrations than those producing morphological changes.

Recent animal studies indicate that both aromatic (Korsak and Rydzynski, 1996; Gralewicz et al., 1997 in MA DEP, 2003) and aliphatic (Lund et al., 1995 in MA DEP, 2003) volatile hydrocarbons may cause nonspecific neurobehavioral toxicities with differing intensities depending on the structure of the hydrocarbon.

Distinct from the general CNS effects of hydrocarbons are their associated specific organ toxicities. Examples of such effects include the hematopoietic toxicity of benzene and the neurodegenerative toxicity of n-hexane. The specific toxicities of hydrocarbons may be directly related to their metabolites, as is the case with benzene and n-hexane (Andrews and Snyder, 1991 in MA DEP, 2003).

HUMAN TOXICOLOGICAL PROFILE – Volatile/Extractable Petroleum Hydrocarbon Fractions

Aliphatic Fractions

C_5-C_8 Aliphatics

n-Hexane was originally selected by MA DEP as the indicator for this range because its toxicity has been well investigated and also because of some evidence demonstrating that the other alkanes in the group may have similar neurotoxic capacities. The peripheral neurotoxicity of n-hexane is of particular human health concern, although respiratory and irritation effects have also been observed. Several epidemiological studies have demonstrated that human inhalation exposure to n-hexane resulted in polyneuropathy. In the epidemiological studies, exposure was to n-hexane, commercial grade hexane, or other mixtures within the specified carbon ranges for this fraction. The mixtures contained n-hexane at levels ranging from 12.3 to 60%. The data do not allow comparison of the severity of the neuropathy induced by pure n-hexane or the aliphatic mixtures in the series (MA DEP, 2003).

C_9-C_{18} Aliphatics

Inhalation exposure of painters to white spirit resulted in early disability work status due to neuropsychological disorders. In most of the studies, workers were exposed to mixtures of organic solvents, with the principal component being white spirit. The effects were mainly functional disturbances in the CNS including memory and learning impairments (Lund et al. (1995) and references therein in MA DEP, 2003).

C_{19}-C_{32} Aliphatics

No human toxicological data are available. However, emerging studies suggest that exposures to petroleum distillates appear to increase the risk of autoimmune diseases (e.g., undifferentiated connective tissue diseases (Lacey et al., 1999 in MA DEP, 2003)).

Aromatic Fractions

C_6-C_8 Aromatics

In the MA DEP fractions approach (MA DEP, 1994), aromatic hydrocarbons with fewer than nine carbon atoms (e.g., benzene, toluene) are evaluated on a compound-specific basis. No toxicity data were identified on mixtures in this carbon range. However, the MA DEP has selected a representative oral reference dose for the carbon range considering the availability of good toxicity data for styrene, ethylbenzene, and xylenes and compositional information for this fraction. The human health concerns for these compounds include CNS effects, mucous membrane irritations, and developmental and reproductive effects.

C_9-C_{32} Aromatics

The MA DEP grouped the entire range of C_9-C_{32} aromatic hydrocarbon compounds as a single fraction for the purposes of deriving the oral RfD. For the purposes of evaluating inhalation toxicity, MA DEP evaluated the C_9-C_{18} and C_{19}-C_{32} carbon ranges.

The C_9-C_{18} fraction includes two and three ring PAHs, such as naphthalene, 2-methylnaphthalene, and fluorene; and alkylated benzenes, such as 1,2,4-trimethylbenzene and isopropylbenzene. The critical effects for these compounds from inhalation exposure are pulmonary, hepatic, renal, CNS, and developmental/reproductive effects.

C_{19}-C_{32} Aromatics

No appropriate data were identified to support development of inhalation RfCs for the individual components or mixtures in this carbon range. The compounds in this carbon range are not very volatile and inhalation of gaseous compounds is not a likely route of exposure.

ANIMAL TOXICOLOGICAL PROFILE – Volatile/Extractable Petroleum Hydrocarbon Fractions

Aliphatic Fractions

C_5-C_8 Aliphatics

Overall, the chronic commercial hexane studies used by the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) demonstrated that an inhalation exposure to a hexane mixture containing 53% n-hexane produced no toxicity in rodents. However, and most importantly, other chronic human and animal studies showed that commercial hexane causes peripheral neuropathy. In addition, many potential diketone metabolites of n-alkanes produce peripheral neurotoxicity.

C_9-C_{18} Aliphatics

New oral gavage studies on various petroleum streams covering C_9 - C_{17} carbon ranges observed adverse effects in the treated animals such as body weight, organ weight, and blood chemistry changes. A recent neurotoxicity study revealed that exposure of rats to dearomatized white spirit for six months induced long-lasting and possibly irreversible effects in the nervous system.

In acute animal studies, white spirit with low aromatic content produced significant reductions in animal response to learned performances. Increased levels in brain noradrenaline, dopamine, and 5-hydroxytryptamine were observed in rats exposed to various levels of white spirit. Changes in indices of oxidative stress in the synaptosomes were also reported in animals exposed to white spirit for 3 weeks.

C_{19}-C_{32} Aliphatics

Based on a rat subchronic feeding study of several different highly refined white mineral oil samples representing various mineral hydrocarbon (MHC) sizes, the low molecular weight (average molecular weight 320-420) MHC caused mesenteric lymph node histiocytosis and liver granulomas, while the high molecular weight MHC demonstrated minimal effect (Smith et al., 1996 in MA DEP, 2003). No inhalation toxicity data are available.

Aromatic Fractions

C_6-C_8 Aromatics

See discussion under human toxicological profile.

C_9-C_{32} Aromatics

The C_9-C_{18} fraction includes low molecular weight PAHs and alkylated benzenes. Naphthalene is a hematopoietic and pulmonary toxicant. Inhalation exposure to naphthalene produced severe pulmonary damage and lesions in mice (NTP, 1992 in MA DEP, 2003). A recent oral study of a structurally related compound, 1-methylnaphthalene, suggested that the target site might be the pulmonary tissues. Chronic oral administration of 1-methylnaphthalene was associated with significantly increased nodular alveolar proteinosis in mice. A significant increase in pulmonary adenoma was observed in males (Murata et al., 1993 in MA DEP, 2003). In some short-term, high dose experiments, animals exposed to isopropylbenzene exhibited damage to the spleen and fatty changes to the liver. The critical treatment-related effects were to the kidney (Sandmeyer, 1981 in MA DEP, 2003). Rats exposed to trimethylbenzene demonstrated significant changes in CNS function by the behavioral tests in the higher dose groups (Gralewicz et al., 1997 in MA DEP, 2003). Systemic, reproductive, and developmental toxicities have also been demonstrated in animals exposed to C_9 aromatic mixtures (MA DEP, 2003).

For C_{19}-C_{32} aromatics, see discussion under human toxicological profile.

GENOTOXICITY

There are no studies relating TPH-VPH/EPH exposure in humans to genotoxicity. An inhalation oncogenicity study of commercial hexane in rats and mice (API, 1995, Part II in MA DEP, 2003) demonstrated liver tumors in female mice in the highest dose group.

REFERENCES

Andrews, L. S. and Synder, R. 1991. In: MA DEP, 2003. Toxic effects of solvents and vapors. In: Casarett and Doull's Toxicology: The Basic Science of Poisons (M.O. Amadur, J. Doull and C.D., Klaassen, Eds). pp. 681-722. Pergamon Press, New York.

API. 1995. In: MA DEP, 2003. An Inhalation Oncogenicity Study of Commercial Hexane in Rats and Mice. Part I and Part II. Toxicology Report No. 40:CAIS No. 41-33.231 and 41-33.232. American Petroleum Institute, Washington, D.C.

Browning, E. 1965. In: MA DEP, 2003. Toxicity and Metabolism of Industrial Solvents. Elsevier Publishing Co. New York. pp. 739.

Gralewicz, S., Wiaderna, D., Tomas and Rydzynski, K. 1997. In: MA DEP, 2003. Behavioral changes following 4-week inhalation exposure to pseudocumene (1,2,4-trimethylbenzene) in rat. Neurotox. Teratol. 19:327-333.

Korsak, Z., Rydzynski, K. 1996. In: MA DEP, 2003. Neurotoxic effects of acute and subchronic inhalation exposure to trimethylbenzene isomers (pseudocumene, mesitylene, hemmimellitene) in rats. Int. J. Occup. Med. Environ. Health 9:341-349.

Lacey, J.V., Jr., Garabrant, D.H., Laing, T., Gillespie, B.W., Mayes, M.D., Cooper, B.C., and Schottenfeld, D. 1999. In: MA DEP, 2003. Petroleum distillate solvents as risk factors for undifferentiated connective tissue disease (UCTD). Am. J. Epidemiol. 149:761-770.

Lund, S.P., Simonsen, L., Hass, U., Ladefoged, O., Lam, H.R., and Ostergaard, G. 1995. In: MA DEP, 2003. Dearomatized white spirit inhalation exposure causes long-lasting neurophysiological changes in rats. Neurotox. Terat.18:67-76.

MA DEP, 1994. Interim Final Petroleum Report: Development of Health-Based Alternative to the Total Petroleum Hydrocarbon (TPH) Parameter. Office of Research and Standards, Boston. August, 1994.

REFERENCES (Cont'd.)

MA DEP, 2002. Characterizing Risks posed by Petroleum Contaminated Sites: Implementation of the MA DEP VPH/EPH Approach. Final Approach. Office of Research and Standards. October 31, 2002.

MA DEP, 2003. Updated Petroleum Hydrocarbon Fraction Toxicity Values for the VPH/EPH/APH Methodology. Final. Office of Research and Standards. November 2003.

Murata, T., Denda, A., Maruyama, H., et al., 1993. In: MA DEP, 2003. Chronic toxicity and carcinogenicity studies of 1-methylnaphthalene in B6C3F1 mice. Fundam. Appl. Toxicol. 21:44-51.

NTP 1992. National Toxicology Program. In: MA DEP, 2003. Technical report series No. 410. Toxicology and carcinogenesis studies of naphthalene (CAS No. 91-20-3) in B6C3F$_1$ mice (inhalation studies). Research Triangle Park, NC: U. S. Department of Health and Human Services, US Public Health Service, National Institutes of Health. NIH Publication No. 92-3141.

Sandmeyer, E.E. 1981. In: MA DEP, 2003. Aliphatic Hydrocarbons, In: Patty's Industrial Hygiene and Toxicology, 3rd Rev. Ed., Vol. 2B. Toxicology, G.D. Clayton and F.E. Clayton, Eds. pp. 3175-3194. John Wiley and Sons, New York.

Smith, J.H., Mallett, A.k, Priston, R.A.J., Brantom, P.G., Worell, N.R., Sexsmith, C, and Simpson, B.J. 1996. In: MA DEP, 2003. Ninety-day feeding study in Fischer 344 rats of highly refined petroleum-derived food grade white oils and waxes. Toxicol. Pathol. 24:214-230.