ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Evaluation of Exposure to Groundwater, Surface Water, Soil, and Sediment

AMERICAN CREOSOTE WORKS, INCORPORATED
LOUISVILLE, WINSTON COUNTY, MISSISSIPPI

EPA FACILITY ID: MSD004006995

JUNE 28, 2016

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

Comment Period Ends:

JULY 28, 2016

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment-Public Comment Release was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate. This document represents the agency's best efforts, based on currently available information, to fulfill the statutory criteria set out in CERCLA section 104 (i)(6) within a limited time frame. To the extent possible, it presents an assessment of potential risks to human health. Actions authorized by CERCLA section 104 (i)(11), or otherwise authorized by CERCLA, may be undertaken to prevent or mitigate human exposure or risks to human health. In addition, ATSDR will utilize this document to determine if follow-up health actions are appropriate at this time.

This document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. Where necessary, it has been revised in response to comments or additional relevant information provided by them to ATSDR. This revised document has now been released for a 30-day public comment period. Subsequent to the public comment period, ATSDR will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This will conclude the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

Use of trade names is for identification only and does not constitute endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Please address comments regarding this report to:

Agency for Toxic Substances and Disease Registry
Attn: Records Center
1600 Clifton Road, N.E., MS F-09
Atlanta, Georgia 30333

You May Contact ATSDR Toll Free at
1-800-CDC-INFO
or
Visit our Home Page at: http://www.atsdr.cdc.gov

PUBLIC HEALTH ASSESSMENT

Evaluation of Exposure to Groundwater, Surface Water, Soil, and Sediment

AMERICAN CREOSOTE WORKS, INCORPORATED
LOUISVILLE, WINSTON COUNTY, MISSISSIPPI

EPA FACILITY ID: MSD004006995

Prepared by:

Eastern Branch
Division of Community Health Investigations
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

SUMMARY

Introduction

ATSDR's top priority is to ensure that the community surrounding the American Creosote Works, Incorporated, (ACW) site in Louisville, Mississippi, has the best information possible to safeguard their health.

The ACW site is located on a 120-acre tract of land in the southwestern portion of Winston County. The facility operated from about 1912 to 1998. The facility pressure treated wood products (poles, piling lumber, bridge timber, crossties, and posts) with coal-tar solution and creosote oil. The treatment activity and storage practices have contaminated on-site soil, sediment, and water with polycyclic aromatic hydrocarbons (PAHs) and other compounds and elements. The United States Environmental Protection Agency (EPA) proposed and listed the ACW site on the National Priorities List (NPL) in 2001. Congress requires ATSDR to conduct public health activities on all sites proposed for the NPL. ATSDR released a draft (initial) Public Health Assessment (PHA) for the site in 2002. Since then, additional environmental sampling to further define the nature and extent of contamination on and near the site was conducted by EPA. EPA has completed all site work including the slurry wall, the waste containment cell, and the deep-soil wall under the site. These actions should eliminate on-site current exposures and prevent any future on-site exposures.

The purpose of this PHA is to determine whether exposure to contaminants from the ACW site harmed the community, and what public health actions need to be taken to reduce harmful exposures.

Conclusions Analytical results indicate that PAHs, dioxins, and dibenzofuran are the primary chemicals of potential concern at the site. After evaluating the available data, ATSDR reached six conclusions in this PHA.

Conclusion 1 Past exposure to PAHs, dioxins and dibenzofuran from on-site Surface Soil: ATSDR concluded that incidental ingestion of contaminants in surface soil on site by trespassers was not expected to harm people's health.

Basis for Conclusion Surface-soil samples collected on site from 1999 to 2009 revealed that PAHs concentrations ranged from non-detect to 612.4 milligram per kilogram (mg/kg), dioxin levels ranged from 2.7 to 2,331 nanogram per kilogram (ng/kg), and dibenzofuran levels ranged from non-detect to 1,400 microgram per kilogram (µg/kg). Conservative exposure dose calculations indicated that PAHs, dioxin and dibenzofuran levels were below levels known to result in non-cancer harmful health effects. The estimated cancer risks (PAHs and dioxin combined) ranged from 6.0E-05 to 4.9E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 6 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants in surface soil on site by trespassers was not expected to harm people's health.

Conclusion 2 Current exposure to PAHs, dioxin and dibenzofuran from off-site surface soil: ATSDR concluded that incidental ingestion of contaminated surface soil by residents in their yards is not expected to harm people's health.

Basis for Conclusion Surface soil samples collected off site near the facility from 1999 to 2009 revealed PAH levels ranging from non-detect to 0.158 milligram per kilogram (mg/kg). Dioxin levels ranged from 4.9 to 7.8 ng/kg. One sample contained dibenzofuran at a concentration of 78 µg/kg. Using conservative residential exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful health effects. The range of excess cancer risks (PAHs and dioxin combined) for residents is from 6.2E-05 to 4.6E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 6 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion

of contaminants in surface soil off site by residents is not expected to harm people's health.

Conclusion 3

Current exposure to PAHs, dioxin and dibenzofuran from off-site Hughes Creek sediments: ATSDR concluded that incidental ingestion of contaminated sediment in Hughes Creek by residents during occasional recreational activities is not expected to harm their health.

Basis for Conclusion

PAH sediment levels from the Hughes Creek ranged from non-detect to 215.7 mg/kg. Concentrations of dioxin ranged from 0.474 to 44 ng/kg. Concentrations of dibenzofuran range from negligible to 440 mg/kg. ATSDR assumed that adults and children (aged 6 to 21 years) were exposed for 105 days (every other day for 7 months) per year for 5 to 33 years. The exposure period is based upon the assumption that adults or children played in the creek in the warmer months of the year (from April to October) every other day. Using conservative exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful effects. The estimated cancer risks (PAHs and dioxin combined) ranged from 8.6E-05 to 4.8E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 9 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants in sediments during occasional recreational activities is not expected to harm people's health.

Conclusion 4

Current exposure to PAHs and dibenzofuran from off-site Hughes Creek surface water: ATSDR concluded that incidental ingestion of contaminated surface water in Hughes Creek by residents during occasional recreational activities is not expected to harm people's health.

Basis for Conclusion

PAH levels in surface water from the off-site Hughes Creek ranged from non-detect to 1.64 micrograms per liter (µg/L). Dibenzofuran was found in only one sample at the concentration of 3µg/L. ATSDR assumed that adults and children (aged 6 to 21 years) were exposed for 105 days (7 months and every other day) per year for 5 to 33 years. The exposure period is based upon the assumption that adults or children played in the creek in the warmer months of the year (from April to October) every other day. Using conservative exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful effects. The estimated cancer risks for PAHs ranged from 5.4E-05 to 1.6E-06. Stated another way, out of 100,000 people exposed to the same level

over the same amount of time, we estimate that less than 2 to 5 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants in off-site surface water during occasional recreational activities is not expected to harm people's health.

Conclusion 5 ATSDR cannot conclude whether eating fish from Railroad Lake or breathing outdoor air in the past could harm people's health because the information we need to make a decision is not available.

Basis for Conclusion Railroad Lake was used for fishing in the past and the lake was drained during remediation. There was one fish sample collected and tested from Railroad Lake and not enough information to evaluate the past exposure. Residents complained about strong odors while the facility was in operation. Inhalation of creosote components was a potential past exposure pathway. No data were collected in the past for evaluation.

Conclusion 6 ATSDR concluded that any remaining contamination in groundwater, indoor air, and subsurface soil at and near the site are not expected to harm people's health because those exposure pathways are eliminated.

Basis for Conclusion Residents are not drinking groundwater but use public water in the area, and municipal wells located near the ACW site are not contaminated. No private wells are used in this area. A deep clay layer in the aquifer prevents the contaminants from moving into the municipal wells. The potential for migration of vapors into indoor structures is low because contaminated soil was removed from the potential future building area. For off-site residents, the levels of VOCs that were present in the groundwater were too low to cause a concern for vapor intrusion. Because any remaining contamination is beneath the ground, only people engaged in earth-moving activities could be exposed to this subsurface contamination. In addition, most of the on-site contaminated soils have been removed and sent to a permitted landfill.

Next Steps EPA will continue routine environmental monitoring activities for the ACW site.

As needed, ATSDR will update this document, or prepare a new document, to reflect potential future sampling results and site remediation activities in relation to any completed or potential exposure pathways identified in this PHA.

More Information You can call ATSDR at 1-800-CDC-INFO, or go to www.cdc.gov/info for more information on the ACW site.

Table of Contents

List of Abbreviations

ACW	American Creosote Works, Incorporated
ATSDR	Agency for Toxic Substances and Disease Registry
ATV	All-terrain Vehicle
BaP	Benzo (a) pyrene
BGS	Below Ground Surface
CalEPA	California Environmental Protection Agency
CDC	Centers for Disease Control and Prevention
CREG	Cancer Risk Evaluation Guide
CSF	Cancer Slope Factor
CTE	Central Tendency Exposure
CV	Comparison Value
DTP	Direct Push Technology
EPCs	Exposure Point Concentrations
HRS	Hazard Ranking System
GAC	Granular-activated-carbon
IARC	International Agency for Research on Cancer
LOAEL	Lowest Observed Adverse Effect Levels
IRIS	Integrated Risk Assessment System
MCL	Maximum Contaminant Level
MDEQ	Mississippi Department of Environmental Quality
mg/L	Milligram per liter
mg/kg	Milligram per kilogram
MLE	Maximum Likelihood Mean
MRL	Minimal Risk Level
MWs	Monitoring Wells
NCEH	National Center for Environmental Heath
ng/kg	Nanogram per kilogram
NLM	National Library of Medicine
NPL	National Priorities List
PAHs	Polycyclic Aromatic Hydrocarbons
PBPK	Physiologically Based Pharmacokinetic
PEF	Potency Equivalent Factor
PHA	Public Health Assessment
PCB	Polychlorinated Biphenyls
PPB	Parts per Billion
PPM	Parts per Million
PPT	Parts per Trillion
QA/QC	Quality Assurance/Quality Control
RD	Remedial Design
RfD	Reference Dose

RI	Remedial Investigation
RME	Reasonable Maximum Exposure
RMEG	Reference Media Evaluation Guide
ROS	Regression on Order Statistics
RSL	Regional Screening Levels
START	Superfund Technical Assessment and Response Team
SVOC	Semivolatile Organic Compounds
TCL	Target Compound List
TEFs	Toxicity Equivalence Factors
TEQ	Toxicity Equivalence
□g/L	Microgram per liter
UCL	95% Upper Confidence Level
USDHUD	U.S. Department of Housing and Urban Development
USEPA	U.S. Environmental Protection Agency
VOC	Volatile Organic Compound

Purpose and Health Issues

The United States Environmental Protection Agency (EPA) proposed including the American Creosote Works site on the National Priorities List (NPL) in June 2001 and listed it in September 2001. Congress requires the Agency for Toxic Substances and Disease Registry (ATSDR) to conduct public health activities on all sites proposed for the NPL.

ATSDR released a draft (initial) Public Health Assessment (PHA) for the site in 2002. A PHA is a document prepared after available environmental data, community concerns, and health outcome data are evaluated to determine whether people have been, are being, or will be exposed to hazardous substances, and, if so, whether those exposures are harmful. If the exposures are deemed harmful, ATSDR makes recommendations to prevent or reduce those exposures. The 2002 draft PHA used data available at the time and concluded that the concentration of contaminants in on-site media, readily accessible to people, was not high enough to cause adverse health effects.

The EPA conducted additional environmental sampling to define further the nature and extent of contamination on and near the site after the release of the 2002 draft PHA. For this updated PHA, ATSDR reviewed available environmental data in soil, sediment, surface water, and groundwater on or near the site. ATSDR also evaluated potential exposure scenarios, exposure pathways, and community health concerns to determine whether adverse health effects are possible.

Background

Site Description and History

The ACW site is located on a 120-acre tract of land in the southwestern portion of Louisville, Winston County, Mississippi [USEPA 2000]. The site is situated at the corner of Railroad Avenue and Baremore Street. The area immediately surrounding the site is both residential and industrial, and two surface water bodies were on the site. The up-gradient Railroad Lake was located on site in the northern portion of the property. A closed surface impoundment (sludge lagoon) used to store stabilized creosote sludge was located in the southeast portion of the property. A residential community borders the facility to the north and northwest. The site is bordered on the west by Hughes Creek, east by the Illinois Central Railroad tracks, and south by woodlands and wetlands [MDEQ 1994].

The facility operated from about 1912 to 1998. During its operational years, the facility operated under various names and ownerships. In 1981, ACW's owner closed and then reopened the facility as American Creosote Works Mississippi, Inc. In 1984, the new owner (The Shannon group of Dallas Texas) changed the name of the facility to Superior Wood Treating, Inc. In 1988, Treat-All Wood Products, Inc. acquired the facility. In 1994, Worldwide Wood Treaters purchased the facility and operated it until 1998 [USEPA 2000].

The facility pressure treated wood products (poles, piling lumber, bridge timber, crossties, and posts) with coal tar solution and creosote oil. The wood-preserving process included two phases: a conditioning cycle that removed moisture from the wood, and an impregnation cycle that pressure-injected heated preservative into the wood. The conditioning cycle generated wastewater and condensate contaminated with creosote. The impregnation cycle generated spent preservatives (creosote oil and coal tar solutions). After the treated wood was removed from the treatment containers, it was placed on drip pads to allow excess preservative to drip off. At the ACW site, waste mixtures were sent to a concentration pit and then overflowed into the sludge lagoons. Wastes may have been pumped directly into Railroad Lake also [USEPA 2007].

From 1984 to 2009, several events occurred that required intervention by EPA and state agencies. From 2001 to 2005, EPA completed a Remedial Investigation (RI) and a Remedial Design (RD) for the site. In September 2011, EPA started its remedial activities for the ACW site. All EPA work at the site was completed in 2015. Below is a summary of site activities:

- In 1984, the Mississippi Bureau of Pollution Control (MBPC) discovered uncontrolled waste locations on the property. EPA conducted an emergency removal action to prevent a levy from breaking and spilling thousands of gallons of creosote sludge into Hughes Creek. Approximately 60,000 cubic yards of contaminated soils and creosote sludge was excavated from the waste lagoons and solidified with kiln dusts. The solidified materials were placed in a new unlined storage cell on site and covered with a 2–3-feet clay cap. Monitoring wells were installed up gradient and down gradient of the combined storage cell. The cap was seeded with grass and graded so that surface water would flow around the cell. Meanwhile, operations continued at the site [USEPA 2000].

- In 1999, an on-site drainage reservoir overflowed and two other reservoirs were nearing capacity. In addition, materials and buildings were left on the site after the facility closed. These included miscellaneous drums, a laboratory building with various chemicals, and nine tanks containing liquids. Other signs of environmental contamination on the site included creosote-stained soils and surface water runoff flowing toward Railroad Lake and Hughes Creek. An emergency removal action was initiated to stop the wastewater and sludge overflows. Approximately 55,000 gallons of liquid was pumped from the tanks and containment cells. The removal and disposal actions of sludge, surface soils, and subsurface soils were completed a few months later. More than 260 tons of debris was disposed off-site

and 4,000 cubic yards of solidified waste was disposed on site. Approximately 176,000 gallons of wastewater was treated [Tetra Tech 1999].

- From 2001 to 2005, EPA completed the RI through five phases of field investigations. The purposes of the RI were to determine the extent of soil and groundwater contamination of the site area. EPA collected numerous environmental samples from different media (surface water, sediment, surface and subsurface soil, and groundwater) at the site [USEPA 2007].

- In 2006 and 2007, EPA installed a line of sheet piling adjacent to Hughes Creek to stop the overflow of creosote waste from the containment area to Hughes Creek [USEPA 2009].

- In 2008 and 2009, EPA conducted an RD investigation to verify the surface and subsurface soil contamination and more accurately delineate the extent of groundwater contamination. EPA collected surface and subsurface soil, sediment, and groundwater samples during this investigation [USEPA 2009].

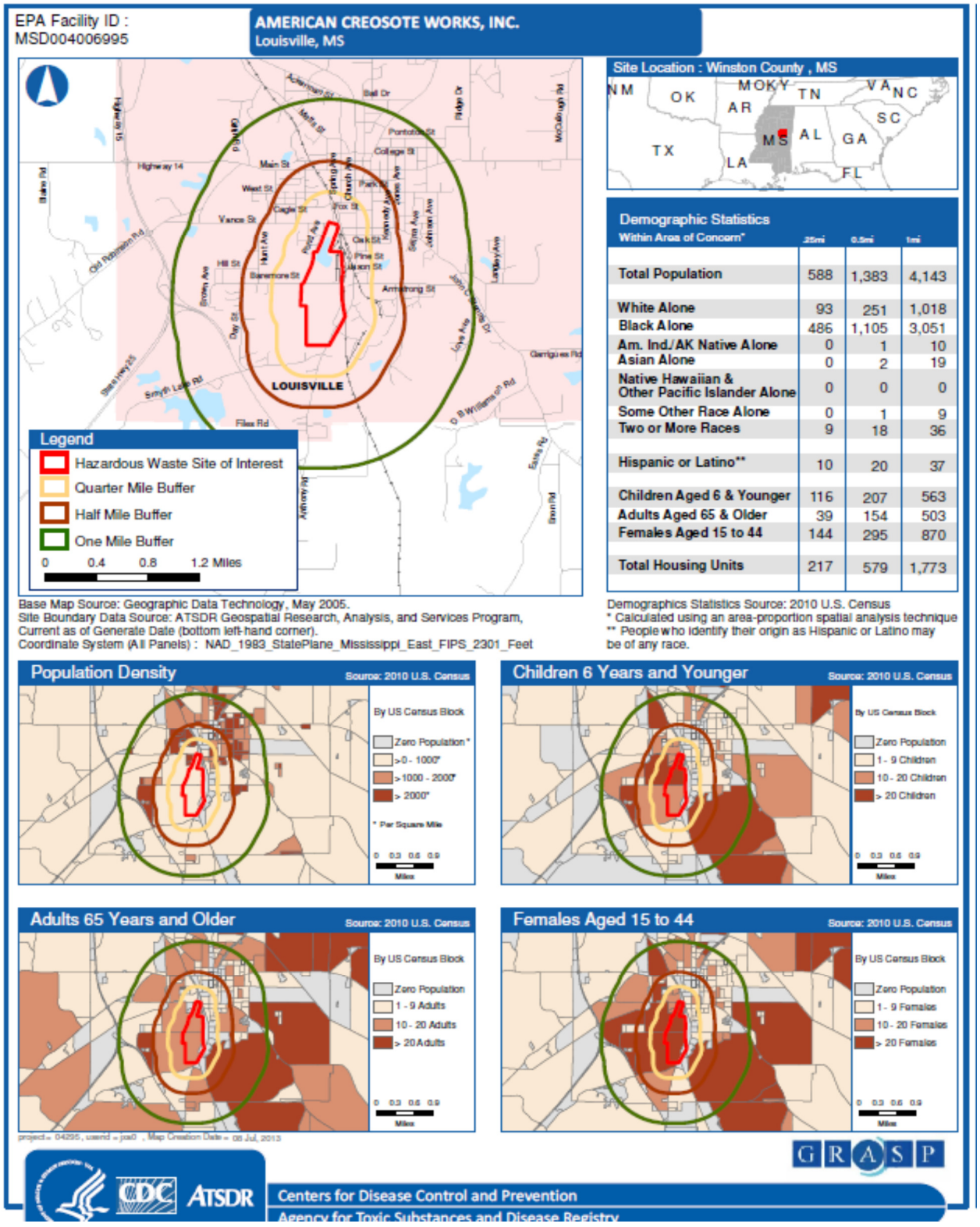
- In September 2010, EPA started the preferred remedial alternative for the ACW site. Remedial actions for soil and sediment included a combination of excavation, consolidation, and capping. For groundwater, vertical barrier walls were selected. EPA had completed all remedial site work including the slurry wall, the waste containment cell, and the deep-soil wall under the site in 2015.

- After containment cell completion, a creosote seep was observed entering a creek adjacent to the cell. It is suspected that creosote in a former creek bed that lies outside the containment cell is the cause of the creosote seeping into Hughes creek. EPA decided to install sheet piling in the area. By end of November 2015, a total of 730 feet of sheet pile was installed and contaminated soil/sediment from the seepage area were removed and placed inside the sheet pile area.

Demographics

The town of Louisville is approximately 124 miles northeast of Jackson, Mississippi. Based upon the 2010 census, approximately 4,143 people live within 1 mile of the site. Of those residents, 21% are women of reproductive age, 14% are aged 6 years or younger, and 12% are aged 65 or older (See Figure 1).

Figure 1. Map and Demographic Information for the ACW Site

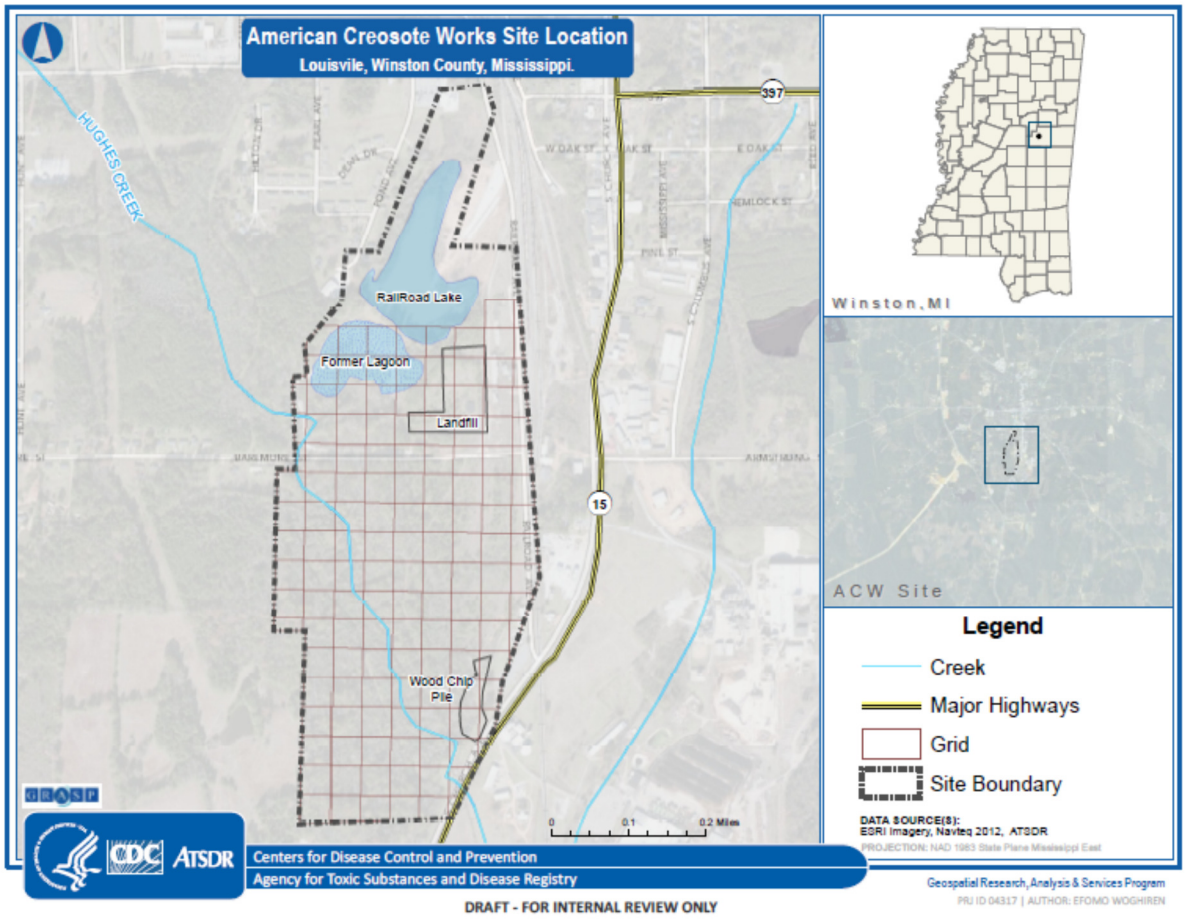


Land and Natural Resource Use

Land Use

The ACW site is situated at the corner of Railroad Avenue and Baremore Street. The area immediately surrounding the site is both residential and industrial. Railroad Lake is on site in the northern portion of the property (See Figure 2). A closed surface impoundment used to store stabilized creosote sludge is on the southeast portion of the property [MDEQ 1994]. A residential community borders the facility to the north and northwest. The west side of the site is bordered by Hughes Creek, the south side (along Baremore Street) by woodlands and wetlands, and the east (along Railroad Avenue) by the Illinois Central Railroad tracks [USEPA 2000]. The southwest portion of the site is located within the 100-year floodplain [USDHUD 1978]. Most of the buildings associated with site operations have been removed. The property is accessible from all directions. A sign that warns of the general hazards associated with the site has been posted, and access to the site from the west is a bit more difficult because of Hughes Creek and the dense trees and undergrowth there.

Figure 2. ACW Site Location



The topography of the area goes from gently rolling to hilly. In general, the materials encountered at the site include topsoil and alternating sequence of different soils (silt, silty clay, silty sand, sandy clay, clayey sand, clay, and lignite coal). The typical thickness of the topsoil is 1–6 inches [L.W. Stephenson, W.N. Logan, and G.A. Waring 1928].

Groundwater

There are two primary hydrogeologic units at the site, the unconfined surficial aquifer and the confined Middle Wilcox aquifer. The Wilcox aquifer is an important water-bearing zone for the area. The groundwater flows south-southwest towards Hughes Creek [L.W. Stephenson, W.N. Logan, and G.A. Waring 1928]. Approximately 9,500 residents obtain potable water from five municipal wells located within ½ mile of the site. The municipal wells are screened approximately 325–375 feet below ground surface (bgs) [Jewell 1999].

Surface Water and Biota

Two surface water bodies (Railroad Lake and Hughes Creek) are associated with the site. Railroad Lake was located on the ACW property. Surface water runoff from the northeast portion of the site drains into the lake. In the past (according to community members), the lake was used for recreational activities such as fishing. People stopped using the lake probably due to concerns about creosote contamination [Tetra Tech, 200]. Hughes Creek is located directly to the west of the ACW property. Surface water runoff from the southwest portion of the site flows into drainage ditches and then over land to the creek. Hughes Creek was used in the past for activities such as fishing, wading, and baptizing. Residents have stopped participating in these activities due to concerns about contamination from the site [Tetra Tech, 2000].

ATSDR Site Visits

In March 2001, ATSDR Regional Operations staff attended a public meeting held by EPA to announce upcoming activities at the ACW site. Approximately 22 residents attended the public meeting. ATSDR staff explained their role at the site and gathered the community's health-related concerns. ATSDR staff noted that children played on the site, and that access to the site should be restricted. ATSDR staff also attended an EPA-sponsored briefing for the site in December 2002. During the December visit, EPA informed us that groundwater contamination appeared to be in a confined aquifer, and that all area homes used municipal water; testing confirmed that the site-related chemicals had not contaminated the municipal water. ATSDR regional and headquarters staff held separate public availability sessions to gather additional community site-related health concerns in March 2003 and June 2004. During those activity periods, ATSDR staff also visited the site and EPA updated them on site activities. ATSDR regional and headquarters staff contacted EPA remedial project managers (RPMs) for additional environmental data and remedial activity. In June 2015, ATSDR staff and EPA RPM conducted another site visit. ATSDR staff noticed that the entrances to the site and the waste containment cell was fenced. A creosote seep adjacent to the cell is contained by absorbent materials. EPA RPM informed ATSDR that the former creek bed that lies outside the containment cell is the cause of the creosote seeping. By end of November 2015, a total of 338 feet of sheet pile was installed and contaminated soil/sediment from the seepage area were removed.

Discussion

Evaluation Process

ATSDR provides site-specific public health recommendations based on an evaluation of the toxicological literature, levels of environmental contaminants at a site compared with accepted comparison values (CV), the characteristics of the exposed population, and the frequency and duration of exposure. This section briefly describes the typical process by which ATSDR evaluates the potential for adverse health effects caused by exposure to site contaminants. See Appendix A and B for more detailed descriptions and terminology.

ATSDR evaluates ways that people may be exposed to contaminated media and subsequently to contaminants (exposure pathways). Exposure pathways consist of five elements (a contamination source, transport through an environmental medium, an exposure point, an exposure route, and an exposed population) that must be present for exposure to occur—whether that exposure occurred in the past, is occurring now, or might occur in the future.

ATSDR categorizes an exposure pathway as complete, potential, or eliminated. Completed exposure pathways are those for which the five elements are evident, and that indicate that exposure to a contaminant has occurred in the past, is now occurring, or will occur in the future. Potential exposure pathways are those for which exposure seems possible, but one or more of the elements is not clearly defined. Potential pathways indicate that exposure to a contaminant could have occurred in the past, could be occurring now, or could occur in the future. An exposure pathway can be eliminated if at least one of the five elements is missing. Exposure pathways also can be eliminated if the site characteristics make past, current, or future human exposures extremely unlikely.

Identification of an exposure pathway does not imply that health effects will occur. Exposures might be, or might not be, substantive. Therefore, even if exposure has occurred, is now occurring, or is likely to occur in the future, that exposure might not affect human health.

The following text describes in general how ATSDR further evaluated completed exposure pathways to determine whether any potential health effects were associated with exposure to contaminated media.

- When presented with results of comprehensive environmental sampling for chemicals, ATSDR reduces the number of contaminants to be evaluated by screening the maximum result for each chemical against comparison values (CVs)—concentrations of chemicals in the environment (air, water, or soil) below which no adverse human health effects would be

expected to occur. If the maximum concentration of the contaminant is present at a level higher than the corresponding CV, which does not necessarily mean adverse health effects will occur, the contaminant is retained for the next step of evaluation. In general, to select CVs, ATSDR uses the hierarchy described in the ATSDR Public Health Guidance manual. In some cases, professional judgment is used to select the most appropriate CVs for the specific site conditions [ATSDR 2005].

- The next step of evaluation focuses on identifying which chemicals and exposure situations could be health hazards. An *exposure dose* is the estimated amount of a contaminant to which a person is exposed. We calculate exposure doses under specified exposure situations. ATSDR have developed guidance to estimate reasonable exposure concentration (EPC) for exposure dose calculations. Each calculated exposure dose is compared against the corresponding *health guideline*, typically an ATSDR minimal risk level (MRL) or EPA Reference Dose (RfD), for that chemical. In general, if the calculated dose is at or below the health guideline, no adverse health effects would be expected. ATSDR also calculates and evaluates site-specific cancer risks for exposed populations.

- If the calculated exposure dose for a chemical is greater than the health guideline, the exposure dose may be refined to reflect more closely actual exposures that occurred or are occurring at the site. The exposure dose is then compared with known health effect levels identified in ATSDR's toxicological profiles or USEPA's Integrated Risk Information System (IRIS). These comparisons are the basis for stating whether the exposure presents a health hazard.

Environmental Contamination

The extent of contamination at the ACW site has been documented through numerous investigations. Maximum concentrations of chemicals in each environmental medium were compared to appropriate CVs to determine which chemicals should be selected for further evaluation. The following is a summary of all environmental data and site information available for this evaluation:

- Tetra Tech EM Inc. Site Inspection Trip Report for American Creosote Works, Inc., March 1999 [Tetra Tech 1999]
- EPA Hazard Ranking System documentation record, American Creosote Works, Inc. December 2000 [USEPA 2000]
- Tetra Tech EM Inc. Final Expanded Site Inspection Report for American Creosote Works, Inc., May 12, 2000[Tetra Tech 2000]
- USEPA Final Remedial Investigation Report, June 2007 [USEPA 2007a]
- USEPA Final Feasibility Study, July 2007 [USEPA 2007b]
- USEPA Final Remedial Design Basis of Design Report, August 2009 [USEPA 2009]

ATSDR also reviewed information on quality assurance (QA)/quality control (QC) specifications for field-data quality and laboratory-data quality to verify the acceptability and adequacy of data including chain-of-custody sheets, project narratives, and laboratory certifications. The laboratory analysis methods and the QA/QC procedures were appropriate. This evaluation included all valid results.
Following is a summary of the ACW site environmental sampling and remediation activities from 1990 to 2014.

Tetra Tech Site Inspection

In January 1999, The Tetra Tech EM, Inc., Superfund Technical Assessment and Response Team (START) conducted a site investigation that included an initial walk-through, a drum inventory, and environmental sampling. START personnel collected two surface water, three sediment, and two soil samples (sampling depth unknown) on or near the site. See Appendix E Table 1 for a summary of the data collected in this event [Tetra Tech 1999].

Analyzed chemicals include VOCs, SVOCs, PCBs, and metals. Samples analyses detected various VOCs, SVOCs, and metals. Dibenzofuran and PAHs exceeded their respective CVs. See Appendix E Table 2 for a summary of sampling results.

Final Expanded Site Inspection

In May 1999, the START team gathered information to generate a preliminary Hazard Ranking System (HRS) score for the site to determine whether the facility was a potential candidate for placement on the National Priorities List (NPL). Environmental media sampled during this event included surface and subsurface soil, sediment, and groundwater [Tetra Tech 2000]. START personnel collected 4 surface soil (0–6 inches) samples and four subsurface soil (>24 inches) to determine presence or absence of contamination. START personnel also collected 8 on-site sediment samples and 4 off-site sediment samples from Railroad Lake and Hughes Creek. Three temporary monitoring wells were installed and sampled. See Appendix E Table 1 for a summary of the data collected in this event.

The laboratories under the EPA Contract Laboratory Program (CLP) analyzed samples for EPA target compound list (TCL) VOCs, extractable SVOCs, pesticides, and PCBs. In addition, the laboratories performed dioxin and furan analysis on three sediment samples (AC-01-SD, AC-07-SD, and AC-11-SD). Analytical results indicated that dibenzofurans and PAHs exceeded their respective CVs (Appendix E. Table 3).

USEPA Final Remedial Investigation

EPA conducted this Remedial Investigation (RI) in four phases plus a supplemental RI phase from May 2001 through March 2005.

Phase I Field Investigation (May–August 2001)

The objective of this phase was to determine the horizontal and vertical extent of soil and groundwater contamination of the site area. On-site environmental samples from different media were collected as summarized in the following text.

- Soil samples from depth of 0–12 inches below land surface - A total of 113 soil samples were collected on a 200 x 200-foot grid over the 120-acre site. The depths of the samples were 0–12 inches below land surface. Twenty of the 113 samples were five-point composite samples collected from 20 grids. Seven of the 113 samples were collected from soil-boring locations for monitoring wells (MWs).

- Soil samples from depth more than 12 inches below land surface – A total of nine subsurface soil samples were collected from the monitoring well borings.

- Surface water and sediment – Nine surface water and nine sediment samples (co-located) were collected from the on-site portion of the Hughes Creek and Railroad Lake. In addition, four surface water and nine sediment samples (co-located) were collected from leachate seep/drainage locations.

- Groundwater – Eight groundwater samples were collected from the installed monitoring wells and five groundwater samples were collected from municipal wells. See Appendix E Table 1 for a summary of the sampling event.

All samples collected were analyzed for TCL VOCs, extractable SVOCs, and target analyte list metals.

Phase II Field Investigation (May 2002)

In May 2002, to further define the soil contamination on-site, EPA collected 29 five-point composite soil samples (depth 0–12 inches) from 29 of the 200 x 200-foot sampling grids. In addition, 29 grab soil samples were collected from the center of the 29 sampling grids at a depth of 5 foot below land surface. The soil samples were analyzed for SVOCs and dioxin/furans. The subsurface soil samples were analyzed for SVOCs only.

Phase III Field Investigation (2002 and 2003)

From November 2002 through February 2003, the Phase III field investigation was conducted to further define the on-site soil contamination and potential contamination of off-site surface water. A total of 95 composite soils samples (depth 0–12 inches) and 95 subsurface grab samples from the sampling grids were collected. A total of 21 surface water and 30 sediment samples were collected both on-site and off-site. The surface soil samples were analyzed for SVOCs and dioxin/furans. In addition, two fish samples were collected. The subsurface soil samples were analyzed for SVOCs only. All surface water and sediment samples were analyzed for TCL VOCs, extractable SVOCs, and target analyte list metals. See Appendix E Table 1 for a summary of the data collected in this event.

Phase IV Field Investigation (2004)

The main objective for the Phase IV investigation was to define the extent of subsurface (subsurface soil and groundwater) contaminant that had migrated off-site. A total of 54 grab subsurface soil samples were collected from the sampling grids and analyzed for SVOCs. A total of 23 groundwater samples were collected from monitoring wells and City of Louisville public wells and analyzed for SVOCs as well. One soil sample (0-12 inches) and one duplicate sample were collected from a residence southwest of the intersection of Baremore Street and Hughes Creek. The surface soil samples were analyzed for SVOCs. See Appendix E Table 1 for a summary of the data collected in this event.

Supplemental RI (2005)

In March 2005, the last RI event was conducted. During this Supplemental RI, 33 sediment samples were collected from 11 locations on-site at sampling intervals of 0–8 inches (sample ID with the Suffix "A"), 8–16 inches (sample ID with the Suffix "B"), and 16–24 inches (sample ID with the Suffix "C"). Seven off-site samples (0-6") were collected west of Hughes Creek. On-site groundwater samples were collected from the 11 on-site monitoring wells. All samples were analyzed for SVOCs. See Appendix E Table 1 for a summary of the data collected in this event.

Overall, the RI investigations collected 237 surface soil samples, 159 subsurface soil samples, 74 sediment samples, 34 surface water samples, and 47 groundwater samples. Review of the RI report indicated that: (1) the primary contaminants detected in all environmental media were PAHs and dioxin; (2) the southern half of the site (approximately 22 acres) has higher concentrations of surface soil contamination; (3) nine offsite surface soil samples were collected from private residences; (4) many of the sampling locations within the Hughes Creek and Railroad Lake onsite contained elevated level of contaminants; and (5) Hughes Creek offsite sediment was also contaminated to a distance about 1 mile beyond the property boundary.

Remedial Design Investigation (2008 and 2009)

To acquire additional data necessary to complete the remedial design, EPA's contractor collected more environmental data on site and off site from May 2008 through May 2009.

For the surface soil investigation, samples were collected at the southern half of the site (22 grids) where previous sampling indicated extensive contamination. Each of the 200 x 200-foot grids was further divided into four 100 x 100-foot grids. A total of 86 five-point composite samples were collected within the subdivided grids at a depth of 0–6 inches below land surface. All samples were analyzed for SVOCs.

The sediment investigation included collecting seven samples on-site at various depths for SVOC analysis. Off-site sediment samples were collected at a depth of 0–6 inches at 18 Hughes Creek locations about 1 mile beyond the property boundary.

A total of 138 subsurface soil samples were collected onsite at various depths from 12 to 100 feet below land surface. Samples were analyzed for SVOCs. Sampling results indicated that the contamination had migrated below and west of Hughes Creek (west of ACW property boundary).

A groundwater investigation was conducted at locations where existing groundwater data were limited. A total of 30 samples was collected from depths of 11–78 feet below land surface. See Appendix E Table 1 for a summary of the remedial design investigation.

ATSDR reviewed a total of 90 data tables that contained more than 1,200 samples. ATSDR reviewed environmental data including about 100 chemicals containing 25 dioxins/furans, 18 metals, 8 pesticides/ polychlorinated biphenyls (PCBs), and 47 volatile organic compounds (VOCs) /semi volatile organic compounds (SVOCs). Contaminant levels that did not exceed a CV were not evaluated further because these concentrations are too low to cause adverse health

effects. Analytical results indicated that concentrations of *PAHs, dioxins, and dibenzofuran* exceeded their respective CVs. The following discussions focus on these chemicals.

Polycyclic aromatic hydrocarbons (PAHs) are a group of more than 100 different chemicals that are formed during the incomplete combustion of organic substances such as coal, oil and gas, garbage, tobacco, or charbroiled meat. PAHs may occur naturally or unintentionally through manufacturing processes. Many products contain PAHs including creosote wood preservatives, roofing tar, certain medicines, dyes, and pesticides [ATSDR 1996].

Creosote is a complex mixture of many chemical compounds. At least 75 percent of creosote is made up of PAHs. The primary wood preservative used at the ACW site was coal tar creosote.

"Dioxin" is the generic name for a group of chemicals including both polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Each unique individual compound in this group is called a congener. The most studied congener, believed to be the most toxic, is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Dioxins are not intentionally produced and have no known use. They are found in very small amounts almost everywhere in the environment [ATSDR 1998].

Dibenzofuran is a combustion product. It is recovered from a wash-oil fraction of coal tar. Dibenzofuran may be released from the incomplete combustion of coal biomass, refuse, diesel fuel, residual oil, and tobacco smoke [NLM, 2000]. Dibenzofuran is found in various percentages in coal tars and coal tar creosotes. Typical wood preservative creosote is approximately 3.5% dibenzofuran. Dibenzofuran is a common component of environmental pollutants, and has been identified in air, ground water, fuel gas, fly ash from municipal incinerators, diesel exhaust gas particulates, and cigarette smoke [Watanabe, 1992].

Evaluation of Exposure Pathways

As mentioned earlier, ATSDR categorizes an exposure pathway as complete or potential, or eliminated.

Completed Exposure Pathways

On-site Surface Soil

The ACW facility was closed at the end of 1998. Although some areas of the site are covered by dense vegetation, access to the site property is not restricted. Thus, exposure to trespassers, particularly adolescents who might access the site periodically, to contaminants in on-site soil likely occurred. A completed exposure pathway for ingestion of or contact with on-site soil existed in the past for occasional trespassers from 1998 to 2014 (when EPA installed secured fence around the perimeter of the site).

Remedial actions for soil and sediment included a combination of excavation, consolidation, and capping. Currently, EPA has completed all site work including the slurry wall, the waste containment cell, and the deep-soil wall under the site. These actions should eliminate current on-site exposures and prevent any future on-site exposures to contaminants if properly maintained.

Off-site Surface Soil

ATSDR identified a completed past, current and future pathways for exposures to contaminants in surface soil in nearby off-site areas such as residential yards, playgrounds, and gardens. Residential areas are located north and about 100 feet west of the site. Potential exposures may occur primarily by accidental ingestion of soil by children or adults. Some dust particles may be breathed in, although this is not considered a primary route of exposure. Preschool children, on average, swallow more soil and dust than do people in any other age group because of their frequent hand-to-mouth activity. The amount of soil that children and adults ingest daily is approximately 30–200 mg [ATSDR 2005; EPA 2011; Calabrese 1977]. To put this amount in perspective, it is approximately equal to less than 1/32–1/8 teaspoon of soil.

Another way children are exposed to soil is by soil pica behavior. Childhood soil pica refers to children who intentionally eat large amounts of soil. Childhood pica behavior, or the eating of non-food items, is well known. Children have been observed eating paint chips, matches, paper, clay, soil, and numerous other non-food items. Soil-pica behavior most likely occurs in preschool children as part of their normal exploratory behavior. Children between the ages of 1 and 2 years have the greatest tendency for soil-pica behavior, and this tendency diminishes as they become older. The exact percentage of children who eat soil is not known. Studies have reported that soil-pica behavior occurs in as few as 4 of 100 children (i.e., 4%) or in as many as 21 of 100 children (i.e., 21%) [Barltrop 1966; Robischon 1971; Shellshear 1975; Vermeer and Frate 1979].

ATSDR and the Colorado Department of Health and Environment found that 21% of preschool children in a predominantly Hispanic population exhibited soil-pica behavior [ATSDR 2005]. Studies on children with soil-pica behavior have documented ingestion of as much as a teaspoon (or 5,000 milligrams) of dirt a day [Stanek and Calabrese 2000; Calabrese and Stanek 1993; Calabrese et al. 1989; Wong 1988]. Limited information is available concerning how often and how long soil-pica behavior occurs in children. Some preschool children might eat soil only one time during their preschool years, while others might go through a stage of eating soil several

times during a week, or over several months. Soil-pica behavior might occur for several days in a row, or a child might skip days between eating soil [Calabrese and Stanek 1998; Calabrese and Stanek 1993; Wong 1988; ATSDR 2001].

Off-site Surface Water and Sediment

Environmental sampling results indicated that some of the surface water samples collected from the Hughes Creek off-site locations contained elevated level of contaminants. Hughes Creek offsite sediment samples were contaminated to a distance about 1 mile beyond the property boundary. Local residents may fish or conduct other recreational activities in the off-site portion of Hughes Creek. People may be exposed to contaminants in Hughes Creek surface water and sediment while working outdoors or playing in nearby areas. People can accidentally swallow small amounts of contaminated surface water or sediment that cling to their hands, and they can absorb contaminants from water and sediment when it touches their skin (dermal contact). Incidental ingestion of surface soil and sediment is the primary route of contaminant exposure for the site. ATSDR identified a completed past pathway, and potential current and future pathways for exposures to surface water. Incidental ingestion is the primary route of exposure. Exposure assessment for this pathway is discussed further in this document. Table 4 is a summary of the completed exposure pathway analysis.

Table 4. Completed Exposure Pathways Identified at the American Creosote Works Site, Louisville, MS

Exposure Pathway	*Exposure Pathway Elements*					*Time Frame*	*Comments*
	Sources of Contamination	*Fate and Transport*	*Point of Exposure*	*Exposed Population*	*Route of Exposure*		
On-site surface soils	Wastes from previous industrial operations at the site	Improper disposal or spillage onto ground	On-site property	Occasional trespasser	Incidental Ingestion, Dermal Inhalation	Past	On-site soil exposure is discussed for occasional trespasser.
Off-site surface soil	Wastes from previous industrial operations at the site	Improper disposal or spillage onto ground	Nearby residences	Residents	Incidental Ingestion, Dermal Inhalation	Past, Present, Future	Residential exposure is discussed.
Off-site sediment	Wastes from previous industrial operations at the site	Improper disposal or discharge/spillage	Contaminated off-site Hughes Creek	Residents in the vicinity of the site	Incidental Ingestion, Dermal	Past, Present, Future	Incidental ingestion is the primary route of exposure.

Table 4. Completed Exposure Pathways Identified at the American Creosote Works Site, Louisville, MS

Exposure Pathway	***Exposure Pathway Elements***					***Time Frame***	***Comments***
	Sources of Contamination	***Fate and Transport***	***Point of Exposure***	***Exposed Population***	***Route of Exposure***		
Off-site surface water	Wastes from previous industrial operations at the site	Surface water runoff, waste seeps into Hughes Creek	Off-site portion of the Hughes Creek	Residents in the vicinity of the site	Incidental Ingestion, Dermal	Past, Present, Future	Incidental ingestion is the primary route of exposure.

Eliminated Exposure Pathways

On-site Surface Water and Sediments

The Railroad Lake and a portion of Hughes Creek are the two surface water bodies on site. The Railroad Lake is located on-site in the northern portion of the property; the lake is approximately 11 acres. Hughes Creek runs about one-half mile across the site's western boundary. Based upon statements made by local residents and officials, Railroad Lake is not being used for recreational purposes; recreational activities ceased at the lake more than 30 years ago [Tera Tech, 2000]. Nonetheless, there is evidence that trespassing is occurring on the site. ATSDR acknowledges that a completed exposure pathway exists for trespassers for the on-site surface water and sediments. However, trespassing events would occur infrequently. In addition, remediation activities on site drained the Railroad Lake and rerouted the Hughes Creek. Therefore, this exposure pathway is eliminated from further discussion in this document.

Groundwater exposures

Leaching of waste from the source areas contaminated the groundwater beneath and adjacent to the ACW site. However, public water is available in the area, and municipal wells located near the ACW site are not contaminated. No private wells were used in this area. Contaminated groundwater from the site likely flows to the west-southwest [USEPA, 2000]. A deep clay layer in the aquifer prevents the contaminants moving into the municipal wells. ATSDR eliminated this exposure pathway for further discussion in this document.

Indoor air exposure

Currently, there are no buildings on site. The city of Louisville may redevelop part of the site into industrial warehouses. Contaminated soil in that potential future industrial warehouse area of the site was removed. EPA and MSDEQ will develop and implement land use deed to restrict the use of the waste containment cell portion of the site. The potential for migration of vapors into indoor structures is unlikely. For off-site residents, the levels of VOCs that were present in the groundwater were too low to cause a concern for vapor intrusion. Therefore, ATSDR eliminated this exposure pathway for further discussion in this document.

Subsurface soil exposures

Only people engaged in earth-moving activities might be exposed to this subsurface contamination. In addition, most of the on-site remediation activities have been completed. Most of the on-site contaminated soils have been removed and sent to a permitted landfill. Therefore, this pathway is eliminated for further discussion in this document.

Potential Exposure Pathways

Outdoor air contaminant exposures

Residents complained about strong odors in the past while the facility was in operation. Inhalation of creosote components was a potential past exposure pathway. No data were collected when the facility was operating; therefore, no further evaluation is possible for this exposure pathway.

Biota exposures

Biota, or the plants and animals in an environment, can be sources of food, clothing, or medicines for people. If people consume contaminated biota, they can be exposed to chemicals in them. For the ACW site, Railroad Lake was used for fishing in the past and the lake was drained during remediation. One fish sample was collected from Railroad Lake and one fish sample was collected from the reference lake (off-site) during the RI. The fish tissue samples were analyzed for SVOCs, dioxins, and metals. SVOCs and metals were not detected in the lake fish tissue sample. Dioxin was detected in the fish tissue sample at very low concentrations (TEQ of 0.052ng/kg). ATSDR considers that a potential biota exposure pathway existed for the past. However, we cannot evaluate this pathway because only one fish sample was available. Table 5 is a summary of eliminated and potential exposure pathways for the ACW site.

Table 5. Eliminated and Potential Exposure Pathways Identified at the American Creosote Works Site, Louisville, MS							
Exposure Pathway	***Exposure Pathway Elements***					***Time Frame***	***Comments***
	Sources of Contamination	***Fate and Transport***	***Point of Exposure***	***Exposed Population***	***Route of Exposure***		
Ground Water (Public Water Supply and Private wells)	Past releases from wood-treating operations at the ACW facility	Infiltration of contaminants to municipal wells; infiltration of contaminants in ground through broken water pipes	Residential faucet/tap	Residents in the area who receive public drinking water; residents with broken below-ground pipes	Dermal, Ingestion, Inhalation	Past, current and future /Eliminated	No elevated levels of chemicals in tested municipal well water. A deep clay layer in the aquafer prevents the contaminants moving into the municipal wells. No wells in use per EPA RPM (May 2015). This exposure pathway is eliminated.

Table 5. Eliminated and Potential Exposure Pathways Identified at the American Creosote Works Site, Louisville, MS							
Exposure Pathway	***Exposure Pathway Elements***					***Time Frame***	***Comments***
	Sources of Contamination	***Fate and Transport***	***Point of Exposure***	***Exposed Population***	***Route of Exposure***		
Outdoor Air	Creosote components during facility operations or as a waste product	Release of creosote components (vapors) into outdoor door	On-site property, Off-site properties	Former facility workers, remedial workers, and residents in the vicinity of the site	Inhalation	Past/Potential	Residents complained about strong odors in the past while the facility was in operation. Inhalation of creosote components was a potential past completed exposure pathway. However, no data were available for evaluation.
Indoor Air	Wastes from wood-treating operations at the ACW facility	Migration of subsurface waste vapors into indoor air	Enclosed structures over contaminated soil or groundwater	People living or working in homes or buildings built over contaminated sub surfaces	Inhalation	Past, current and future /Eliminated	Because the remediation and restriction of the site, this pathway is eliminated.

Table 5. Eliminated and Potential Exposure Pathways Identified at the American Creosote Works Site, Louisville, MS

Exposure Pathway	*Exposure Pathway Elements*					*Time Frame*	*Comments*
	Sources of Contamination	*Fate and Transport*	*Point of Exposure*	*Exposed Population*	*Route of Exposure*		
Subsurface soils	Wastes from previous industrial operations at the site	Subsurface soil transported or released from site	Areas of ground excavation; above–ground seeps	Workers or others who contact contaminated subsurface soils	Ingestion, Dermal, Inhalation	Past, current and future /Eliminated	Only trained workers are coming into contact with subsurface soil and sediments. This exposure pathway is eliminated.
Biota (Fish)	Wastes from previous industrial operations at the site	Surface water to fish tissue	Railroad Lake	Residents in the vicinity of the site	Ingestion,	Past/Potential	This exposure pathway is not evaluated because of limited fish sample.

Public Health Implications

ATSDR further evaluated the completed exposure pathways to determine whether any potential health effects were associated with exposure to contaminants at the ACW site. For chemicals exceeding comparison values, ATSDR performs calculations to determine exposure doses (the amount of contaminant to which a person is exposed) and cancer risk estimates.

Assumptions

To estimate exposure doses, ATSDR made several assumptions. Assumptions are based on default values, ATSDR's Public Health Guidance Manual [ATSDR 2005], ATSDR's Exposure Dose Guidance [ATSDR 2015], EPA's Exposure Assessment Handbook [USEPA 2011], Child-Specific Exposure Factors Handbook [USEPA 2008], or professional judgment. When available, site-specific information was used. Appropriate exposure point concentrations (EPCs) were used to calculate exposure doses. EPCs are the representative contaminant concentrations within an area to which people are exposed. Each calculated exposure dose is compared against the corresponding *health guideline.* If the calculated exposure dose for a chemical is greater than the health guideline, the exposure dose may be refined to reflect more closely actual exposures that occurred or are occurring at the site. See Appendix A for a detailed discussion of ATSDR's evaluation process and Appendix D for dose calculation assumption and results.

Health Guidelines for Dioxin, PAH, and Dibenzofuran

Dioxin

"Dioxin" is the generic name for a group of chemicals including both polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Each unique individual compound in this group is called a congener. The most studied congener, which is believed to be the most toxic, is 2, 3, 7, 8-tetrachlorodibenzo-para-dioxin (TCDD). ATSDR developed a minimal risk level (MRL) for TCDD as $1x\ 10^{-9}$ mg/kg/day (or 0.000000001 mg/kg/day or 0.001 ng/kg/day; a nanogram (ng) is one millionth of a milligram). U.S. EPA has determined the RfD for TCDD as 7×10^{-10} mg/kg/day (or 0.0000000007 mg/kg/day or 0.0007 ng/kg/day). Dioxin toxicity equivalence factors (TEFs) are used to calculate TCDD's toxicity equivalence (TEQ).

EPA chose two human epidemiologic studies as the basis for deriving the RfD [Baccarelli *et al*., 2008; Mocarelli *et al*., 2008] for TCDD. Both of these studies evaluated a human population exposed to TCDD from a 1976 industrial accident in Seveso, Italy. Baccarelli *et al*. reported increased levels of thyroid stimulating hormone (TSH) in newborns who had been exposed to TCDD *in utero.* An increase in TSH in humans indicates a possible dysregulation of thyroid hormone metabolism. The study authors related TCDD concentrations in maternal plasma to newborn TSH levels using a linear regression model. Based on this regression modeling, EPA defined the Lowest Observed Adverse Effect Levels (LOAEL) to be a neonatal TSH level of 5 micro units per milliliter (µU/mL). Using the Emond human Physiologically Based Pharmacokinetic (PBPK) model, the corresponding daily oral intake at the LOAEL is calculated

to be 0.020 ng/kg/day. Adequate levels of thyroid hormone are essential in the newborn and young infant because this is a period of active brain development. Thyroid hormone disruption during pregnancy and early life can cause neurological deficiencies in newborns, particularly attention and memory deficits (EPA 2012). In another study, Mocarelli *et al*. (2008) reported decreased sperm concentrations and decreased motile sperm counts in men who were exposed to TCDD as boys (1–9 years of age) during the 1976 Seveso accident. The lowest exposure level in the Mocarelli *et al*. study (68 parts per trillion [ppt] serum TCDD) is designated as a LOAEL. Using the Emond PBPK model, EPA calculated the LOAEL over the 10-year period to be 0.02 ng/kg/day (EPA 2012). Mocarelli *et al*. (2000) also reported a lower male-to-female sex ratio in offspring of men exposed to TCDD at less than 20 ng/kg/day [EPA 2012, ATSDR 2012]. EPA divided the LOAEL of 0.02 ng/kg/day from the Baccarelli and Mocarelli studies by an uncertainty factor of 30 to arrive at the RfD of 0.0007 ng/kg/day (or 7 x 10^{-10} mg/kg/day).

In summary, exposure to TCDD *in utero* can cause neurological problems, such as memory and attention deficits, in newborns. TCDD exposure *in utero* or as a young boy can affect a man's health and cause lower sperm count and motile sperm count, and reduce the number of male sperm.

More information about the effects of TCDD and other dioxins is available at EPA's IRIS website (http://www.epa.gov/iris/subst/1024.htm) and at ATSDR's Addendum for chlorinated dibenzodioxins (http://www.atsdr.cdc.gov/toxprofiles/cdds_addendum.pdf).

Several agencies have evaluated the cancer-causing properties of dioxin. The Department of Health and Human Services (DHHS) has determined that TCDD may cause cancer in humans. The International Agency for Research on Cancer (IARC) also has determined that TCDD can cause cancer in humans. Previously, the EPA determined that TCDD and mixtures containing TCDD are probable human carcinogens; however, EPA is currently reviewing their findings about the carcinogenic effects of dioxin [ATSDR 1998, EPA 2012].

Human studies have shown that TCDD can cause liver cancer and might be associated with lung, colon, prostate, breast, lymphatic, and hematopoietic cancers [ATSDR 2012]. Rodent studies have confirmed that TCDD can cause cancer at multiple body sites, including the liver, lung, mouth, and thyroid [ATSDR 1998, 2012].

The California Environmental Protection Agency (CalEPA) has developed a CSF for dioxin, specifically 1.3E+5 $(mg/kg/day)^{-1}$ [CalEPA 2005]. Based on this CSF, ATSDR used an interim soil Cancer Risk Evaluation Guide (CREG) of 5.4 ppt for screening phase to determine if further evaluation is necessary [ATSDR 2014].

Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a group of more than 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. The most-studied PAH is benzo (a) pyrene (BaP). PAHs may occur naturally or may be manufactured. Many products contain PAHs including creosote wood preservatives, roofing tar, certain medicines, dyes, and pesticides. PAHs enter the atmosphere from vehicle exhaust, emissions from residential and industrial furnaces, tobacco

smoke, volcanoes, and forest fires [ATSDR 1996]. The PAHs at the ACW Site are residues from creosote.

Because PAHs exist in complex mixtures of different chemicals, assessing their potential health effects is difficult. No acute or chronic Minimal Risk Levels (MRLs) have been derived for PAHs because no adequate human or animal dose-response data are available that identify threshold levels for appropriate non-cancer health effects. Mice fed high concentrations of BaP during pregnancy had difficulty reproducing later, and their offspring had birth defects and low birth weights. Studies of other animals have shown that BaP causes harmful effects on skin, intestinal mucosa (enzyme alterations), and immune system deficiencies. Similar effects could occur in people but those effects have not been documented [ATSDR 2002]. PAHs generally have a low degree of acute toxicity to humans and the most significant endpoint of PAH toxicity is cancer [ATSDR 2009].

For this document, ATSDR used the potency equivalence factor (PEF) that converts the total PAH concentration in a sample to a total carcinogenic PAH (cPAH) concentration [CalEPA 2005]. Based on toxicity, this approach uses potency factors specific for each cPAH to change the concentration of that PAH to a BaP equivalent concentration. Thus, ATSDR summed the BaP equivalent concentration of various individual cPAHs in a soil sample to give the total cPAH for that sample. See Appendix C for details of PEFs each cPAH used for this evaluation. ATSDR established a CREG of 96 parts per billion (ppb) for BaP as a screening level. EPA has developed an oral cancer slope of 7.3E+00 $(mg/kg/day)^{-1}$ for BaP.

Dibenzofuran

Dibenzofuran is a white, crystal-like solid. It is made from coal tar and is used as an insecticide and to make other chemicals. Dibenzofuran may be released from the incomplete combustion of coal biomass, refuse, diesel fuel and residual oil, and tobacco smoke [NLM, 2000]. Dibenzofuran is found in various percentages in coal tars and coal tar creosotes. Typical wood-preservative creosote is approximately 3.5% dibenzofuran. Dibenzofuran is a common component of environmental pollutants, and has been identified in air, ground water, fuel gas, fly ash from municipal incinerators, diesel exhaust gas particulates, and cigarette smoke [Watanabe, 1992]. Exposure to dibenzofuran may occur from inhaling contaminated air or ingesting contaminated drinking water or food. No information is available on the acute (short-term), chronic (long-term), reproductive, developmental, or carcinogenic effects of dibenzofuran in humans or animals. EPA has established an RfD of 0.001 mg/kg/day for dibenzofuran based on an LOAEL of 12.3 mg/kg/day by applying an uncertainty factor of 10,000 [EPA 2007]. No information is available on the carcinogenic effects of dibenzofuran in humans or animals. EPA has classified dibenzofuran as a Group D contaminant, not classifiable as to human carcinogenicity [EPA IRIS]. The available guidelines are the regional screening levels (RSL) of 78 ppm for dibenzofuran in soil/sediment and 16 ppb in water [EPA RSL].

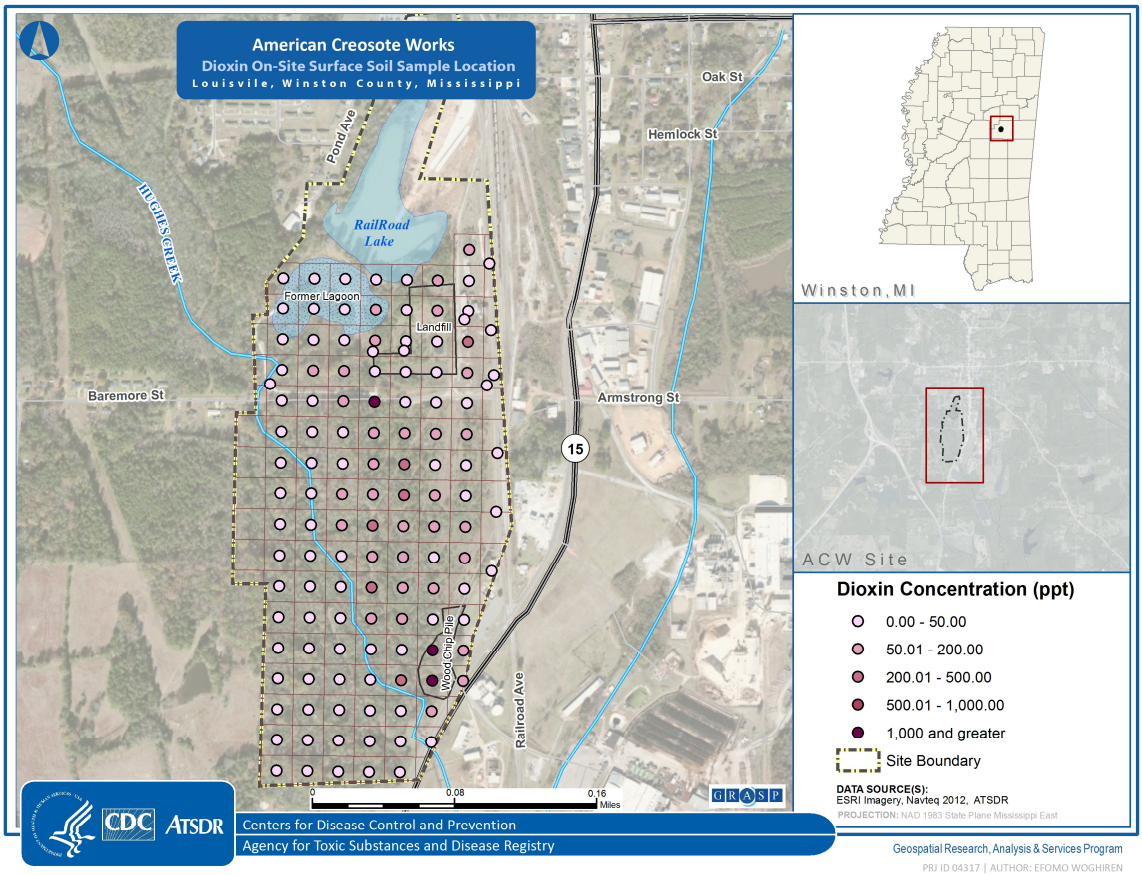
A. On-site Soil – Occasional Trespasser

As mentioned in the previous section, ATSDR identified a completed on-site surface-soil–exposure pathway for the ACW site. ATSDR used the on-site trespasser scenario to evaluate potential exposure to trespassers on the facility property from 1998 to 2014. Most of the on-site surface soil samples are collected at depth of 0-12 inches except the samples taken during the remedial design investigation. A trespasser was more likely exposed to the top few inches of soil. There were more than 80 samples collected at depth of 0-6 inches during the remedial design investigation. ATSDR compared the results of soil samples taken at the same grids at depth of 0-6 and 0-12 inches by inference tests {a parametric test (two-sample parametric T-test) and a non-parametric test (Mann-Whitney-Wilcoxon Test)}. Test results indicated that for most grids, the results from the two depths were similar. Therefore, we used results from both depths for the dose calculations. See Appendix F for details of the parametric test and a non-parametric test. The trespasser was assumed to engage in general recreational activities such as walking, hiking, riding a bike, or riding an all-terrain vehicle (ATV). ATSDR does not have default exposure factors for human trespassers, thus, we used site-specific judgment to estimate appropriate exposure inputs that would not underestimate exposures. The exposure assessment assumes that hypothetically a person trespassed on the site over time, beginning in early childhood (aged 6 years or older) and continuing into adulthood (aged 21years or older). We assumed these trespassing events occurred twice weekly, or 104 days per year, for a total of 16 years.

ATSDR evaluated analytical results for PAHs, dibenzofuran, and dioxin for ACW surface soil samples collected on site. See Appendix E Table 6 for a summary of the on-site surface soil results.

On-site soil sampling results indicated that the former process and storage areas have higher levels of contamination. For example, the following Figure 3 shows the dioxin distribution on site. It is unlikely that people were exposed to the highest levels of contaminants for the entire time of consideration. To calculate a reasonable EPC, ATSDR ran the ProUCL program first. The ProUCL program calculates a 95% upper confidence limit of the arithmetic mean (UCL95) that is a value that equals or exceeds the true (unknown) arithmetic mean of contamination concentration 95 percent of the time. Using the UCL95 provides a protective exposure estimate. However, the ProUCL program recommended method produced very large confidence intervals for cPAHs, dibenzofurans and dioxins because of the high sample variance of the on-site samples. Because of this, the recommended ProUCL method is not the best fit for the set of data. Therefore, ATSDR used another statistical method called stratified bootstrap analysis to estimate the EPC.(R 3.1.2 with packages boot 1.3-14 and NADA 1.5-6 were used to perform the bootstrapping) See Appendix D for detailed discussion on the selection of the stratified bootstrap analysis.

Figure 3. On-site Dioxin Contamination Distribution



Health Effects Evaluation for On-Site Surface Soil

(1) PAHs in On-site Soil

A total of 235 surface-soil samples collected at the site from 1999 to 2009 were available for this evaluation. PAH concentrations ranged from non-detect to 612.4 mg/kg. ATSDR used a statistical method called stratified bootstrap analysis to estimate the EPC of 24.1 mg/kg. Appendix E Table 7 shows the resulting BaP equivalent exposure cancer risks, assuming that the bodies of children and adults absorbed PAHs from incidental soil ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation).

The range of excess cancer risks for trespassers exposed to the estimated EPC of PAHs is from 5.6E-05 to 4.5E-06. Qualitatively, ATSDR does not consider this range of cancer risk to present an appreciably elevated cancer risk. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 6 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of

developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants during occasional recreational activities was not expected to harm people's health.

This evaluation has some uncertainties and limitations, which will be discussed in a separate section (Uncertainty and Limitations in Deciding Harmful Effects) of this document.

(2) Dioxin in On-site Soil

A total of 140 surface-soil samples collected from this site from 1999 to 2009 were available for this evaluation. Dioxin concentrations ranged from 2.7 to 2,331 ng/kg. Similar to the PAH contamination on site, former process and storage areas had higher levels of contamination than the rest of the onsite area. Therefore, ATSDR used a stratified bootstrap analysis to estimate the EPC of 104 ng/kg. Appendix E Table 8 shows the resulting dioxin-exposure doses and cancer risks, assuming that the bodies of children and adults absorbed dioxin by incidental soil ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation).

As mentioned previously, the RfD for TCDD is 7×10^{-10} mg/kg/day. The estimated doses for all age groups ranged from 1.8E-11 to 2.1E-10 mg/kg/day that were below the RfD. For cancer effects, the estimated excess cancer risks ranged from 3.6E-06 to 3.7E-07. Stated another way, out of 1,000,000 people exposed to the same level over the same amount of time, we estimate that less than one to 4 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, it is unlikely that exposure to dioxin-contaminated surface soil on-site will cause any adverse health effects.

(3) Dibenzofuran in On-site Soil

A total of 238 surface-soil samples collected from this site from 1999 to 2009 were available for this evaluation. Concentrations of dibenzofuran ranged from non-detect to 1,400 mg/kg. Similar to other contaminants on site, former process and storage areas had higher levels of contamination than the rest of the onsite area. Therefore, ATSDR used a stratified bootstrap analysis to estimate the EPC. The estimated EPC is 15.9 mg/kg. Using conservative exposure assumptions, calculated exposure doses for all age group are below the EPA RfD of 0.001 mg/kg-day. For example, the highest exposure dose is 0.000028 mg/kg-day for age group from 6 to 11 years old child. Therefore, ATSDR concludes that on-site exposure to dibenzofuran-contaminated soil by trespassers was not expected to harm the trespasser's health. See Appendix E Table 9 for a summary of the dose calculations.

B. Off-site Soil – Residential Exposures

A residential community borders the ACW facility to the north and northwest. Off-site soil samples were collected during three sampling events. In May 1999, four soil (0-6 inches) samples were collected to determine the presence or absence of contamination during the expanded site inspection. In the Phase IV field investigation (March 2004), one soil sample (0-12 inches) and one duplicate sample were collected from a residence southwest of the intersection of Baremore Street and Hughes Creek. During the March 2005 supplemental RI, five (0-12 inches) soil samples plus two duplicates were collected. Children and adults can be exposed to

chemicals in soil by accidentally swallowing small amounts of soil that cling to their hands when they put their hands in their mouths.

Analytical results for PAHs, dibenzofuran, and dioxin are evaluated for off-site surface soil samples. See Appendix E Table 10 for a summary of the off-site surface-soil results.

Health Effects Evaluation for Off-Site Surface Soil

(1) PAHs in Off-site Soil

Nine off-site soil samples (collected at a depth from 0 to 12 inches) were used for this evaluation. ATSDR found concentrations of PAHs ranging from negligible to 0.158 mg/kg. When the number of samples is limited, ATSDR generally use the maximum detected results to estimate the off-site EPC. In residential settings, people are usually only exposed to the top few inches of soil. Assume that all the contamination measure in these 0-12 inches samples was present in the top 3 inches, and the contaminated soil was averaged with 9 additional inches of clean soil, the surface soil contamination might actually be 4 times higher than measured. Therefore, we multiplied the maximum result of 0.158 mg/kg by 4 to represent exposure at the soil surface and the EPC used for the calculation is 0.632 mg/kg. Appendix E Table 11 is a summary of the cancer risk calculation results, assuming that the bodies of children and adults absorb PAHs by incidental soil ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation).

The range of excess cancer risks for residents exposed to the estimated EPC of PAHs is from 6.7E-07 to 5.4E-05. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 5 additional cases of cancer might occur due to the exposure. ATSDR concluded that the residential exposures to contaminated surface soil are not expected to harm people's health.

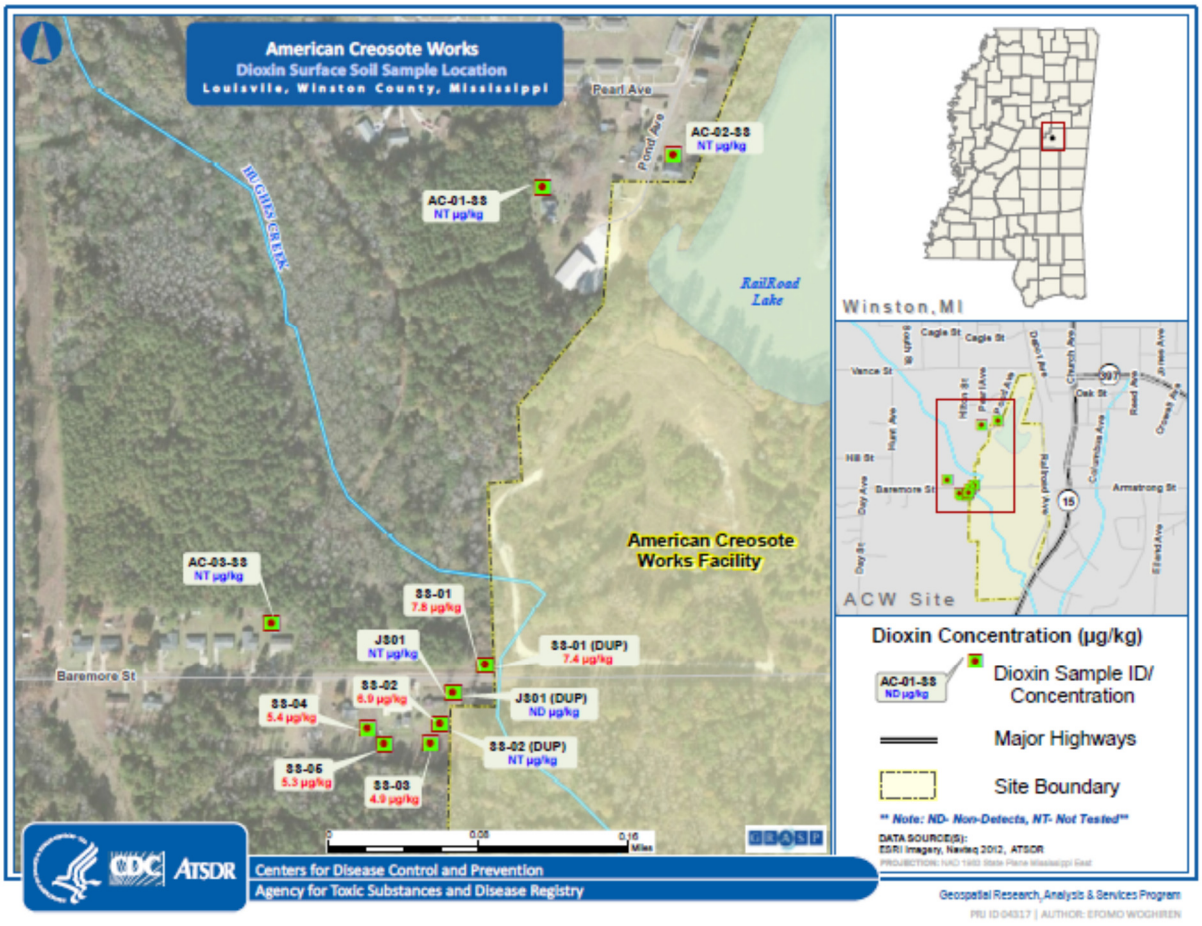
(2) Dioxin in Off-site Soil

Figure 4 shows the off-site surface soil sampling locations and the results of dioxin concentrations. Four off-site soil samples were tested for dioxin. The dioxin concentration range was 4.9–7.8 ng/kg. When the number of samples is limited, ATSDR generally use the maximum detected results to estimate the off-site EPC. Because the samples were collected at a depth from 0 to 12 inches, we multiplied the result of 7.8 ng/kg by 4 to represent exposure to the soil surface and the EPC used for the calculation is 31.2 ng/kg. Appendix E Table 12 shows the resulting dioxin exposure doses, assuming that the bodies of children and adults absorb dioxin by incidental soil ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation).

The estimated doses for all age groups are below the MRL of 1x 10^{-9} mg/kg/day and RfD of 7 x 10^{-10} mg/kg/day for TCDD for chronic exposures. For children with pica behavior, their exposure doses are below the acute oral MRL of 2E-07 mg/kg/day. For cancer effects, the range of excess cancer risks for residents is 7.9E-06 to 3.9E-06. Stated another way, out of 1,000,000 people exposed to the same level over the same amount of time, we estimate that 4 to 6

additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR concluded that the residential exposures to dioxin in contaminated surface soil are not expected to harm people's health.

Figure 4. Off-site soil–Sampling Locations and Dioxin Concentrations



(3) Dibenzofuran in Off-site Surface Soil

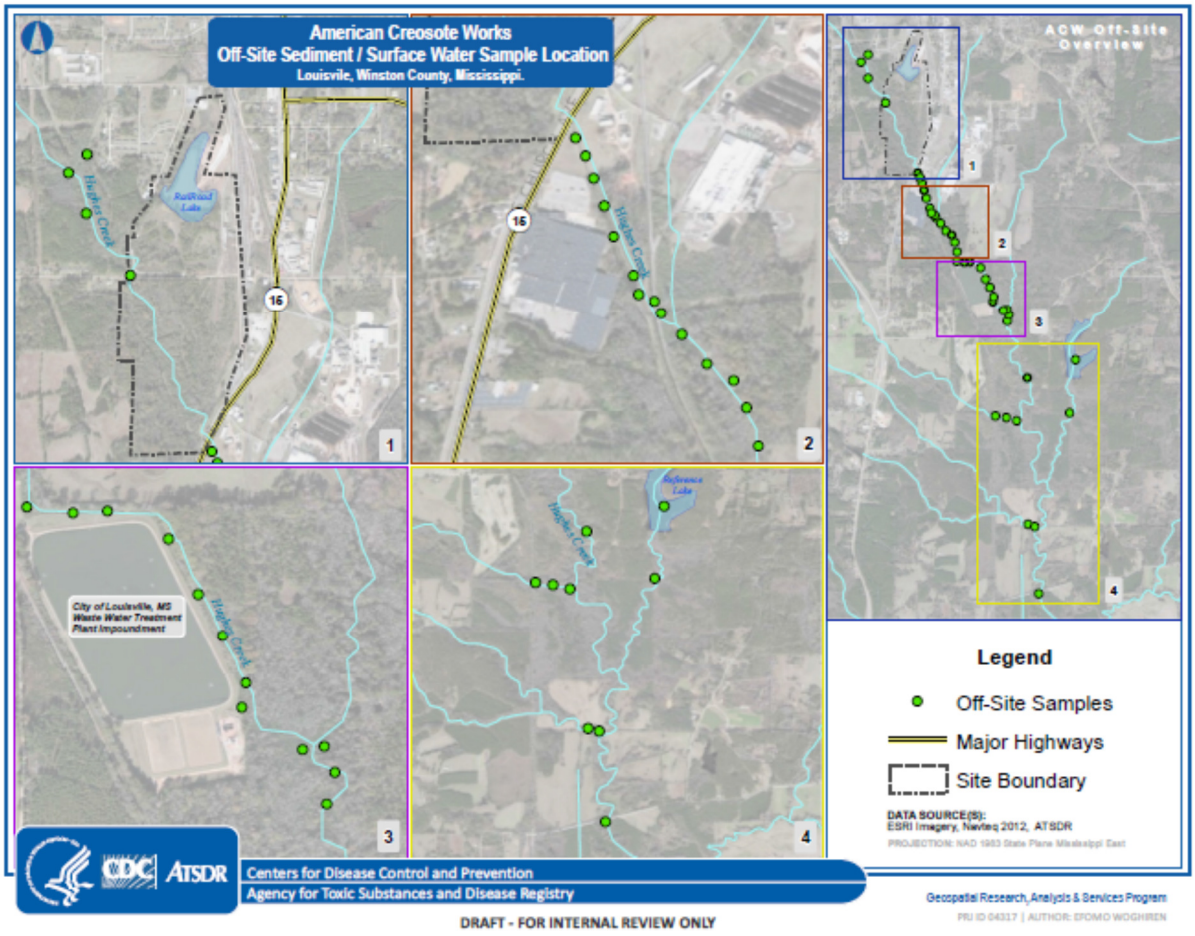
All seven off-site surface soil samples were tested for dibenzofuran; only one sample contained dibenzofuran at a concentration of 78 µg/kg (0.078 mg/kg). Because the samples were collected at a depth from 0 to 12 inches, we multiplied the result of 0.078 mg/kg by 4 to represent exposure to surface soil. The EPC used for the calculation is 0.312 mg/kg. Using conservative exposure assumptions, the highest exposure dose is 0.000028 mg/kg-day for age group from 6 to 11 years old child. This dose is below the EPA RfD of 0.001 mg/kg-day. Therefore ATSDR concluded that off-site exposure to dibenzofuran-contaminated soil likely will not harm residents' health.

C. Off-site Sediment and Surface Water

For the sediment and surface water exposures off-site, ATSDR assumes that exposure to adults and children (aged 6–21 years) would occur 105 days (7 months and every other day) per year. The exposure period is based upon the assumption that adults or children would play in the creek in the warmer months of the year (from April to October) every other day.

ATSDR evaluated the analytical results for PAHs, dibenzofuran, and dioxin in sediment and surface-water samples. Appendix E Tables 13 and 14 are summaries of the off-site sediment and the on- and off-site surface water results, respectively. Figure 5 shows the off-site surface water and sediment sampling locations.

Figure 5 Off-site Surface-water and Sediment-Sampling Locations



Health Effects Evaluation for Off-Site Sediments and Surface Water

To estimate the amount of a chemical that a person might be exposed to in contaminated sediments, ATSDR calculated exposure doses for children and adults using the formulas in Appendix D.

(1) PAHs in off-site Hughes Creek Sediment

A total of 42 off-site sediment samples collected (0 to 12 inches) from this site from 1999 to 2009 were available for this evaluation. Concentrations of PAHs ranged from non-detect to 215.7 mg/kg. Some sediment samples were collected at depth of 1 to 12 inches, to best protect public health, as described in the off-site surface soil section, we multiplied the results by 4 to represent the exposure. ATSDR calculated the doses using the ProUCL estimated EPC of 17.57 mg/kg in sediment.

As shown in Appendix E Table 15, ATSDR calculated the lifetime estimated cancer risk to be 3.5E-06 to 8.3E-05. Stated another way, out of 100,000 people exposed to the same level over

the same amount of time, we estimate that less than one to 8 additional case of cancer might occur due to the exposure. We interpret this as a low increased lifetime risk of developing cancer. ATSDR concluded that the exposures to PAHs in contaminated sediment are not expected to harm people's health.

(2) Dioxin in off-site Hughes Creek Sediment

There are 18 off-site sediment samples tested for dioxin. Concentrations of dioxin ranged from 0.474 to 44 ng/kg. Sediment samples for dioxin were collected at a depth from 0 to 12 inches, we multiplied the results of each sample by 4 to represent the exposure. Using EPA's ProUCL program, ATSDR estimated the off-site EPC as 65.79 ng/kg or 6.6E-05 mg/kg.

Appendix E Table 16 shows the resulting dioxin exposure doses, assuming that the bodies of children and adults absorb dioxin by incidental sediment ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation).

The estimated doses for all age groups are below the MRL of $1x\ 10^{-9}$ mg/kg/day and the RfD of 7×10^{-10} mg/kg/day for TCDD. For cancer effects, the estimated excess cancer risks ranged from 1.3E-06 to 2.5 E-06. We interpret this as a low increased lifetime risk of developing cancer. Therefore, it is unlikely that exposure to dioxin-contaminated sediment off-site will cause any adverse health effects.

(3) Dibenzofuran in Off-site Hughes Creek Sediment

A total of 43 off-site sediment samples collected from 1999 to 2009 were available for this evaluation. Concentrations of dibenzofuran range from negligible to 440 mg/kg. We also multiplied the results of each sample collected from 0 to 12 inches by 4 to represent the exposure. Using the USEPA ProUCL, ATSDR estimated an EPC of 41.21 mg/kg in sediment. Using conservative exposure assumptions, the highest exposure dose is 0.000074 mg/kg-day for age group from 6 to 11 years old child. This dose is below the EPA RfD of 0.001 mg/kg-day. Therefore ATSDR concludes that off-site exposure to dibenzofuran-contaminated sediment likely will not harm resident's health.

(4) PAHs and dibenzofuran in Off-site Hughes Creek Surface Water

A total of 16 off-site surface water samples from this site were available for this evaluation. Concentrations of PAHs ranged from non-detect to 1.64 µg/L. Using the USEPA ProUCL, ATSDR estimated an EPC of 1.1µg/L.

Appendix E Table 17 shows the resulting PAH exposure doses, assuming that the bodies of children and adults absorbed PAHs by incidental surface-water ingestion (see Appendix D for a detailed discussion of ATSDR's dose calculation). ATSDR used very conservative drinking water intake rates for the calculations so the actual cancer risks are likely much lower than the estimated risks.
For central tendency exposure (CTE) which refers to people who have average or typical water intake rate, the calculated lifetime estimated cancer risk is 1.6E-06. For reasonable maximum

exposure (RME) which refers to people who are at the high end of the exposure distribution (approximately the 95th percentile), the calculated lifetime estimated cancer risk is 5.4E-05. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that there may be less than one to 5 additional case of cancer might occur due to the exposure. Therefore, ATSDR considers that incidental ingestion of contaminants during occasional recreational activities was not expected to harm people's health

EPA tested 16 surface-water samples for dibenzofuran; the chemical was found in only one sample at the concentration of 3 µg/L. Using conservative exposure assumptions, the highest exposure dose is 0.000038 mg/kg-day for age group from 6 to 11 years old child. This dose is below the EPA RfD of 0.001 mg/kg-day. Therefore, ATSDR concludes that off-site exposure to dibenzofuran-contaminated surface water likely will not harm resident's health.

Health Effects Evaluation for Exposure to the Mixture of Multiple Chemicals

To evaluate the health impact of exposure to chemical mixtures at the site, we followed ATSDR protocols for evaluating exposures to multiple chemicals of concern [ATSDR 2004]. For noncancer effects, individual contaminants detected are present at levels below that might be expected to result in adverse health effects. Therefore, ATSDR considers that the combined effects of the contaminants at the site are not likely to result in adverse noncancer health effects. For cancer effects, mixture of concern for the site are PAHs and dioxins. We assume that their interaction is additive because there is no information indicated that the two mixtures interact synergistically. The following table 18 is a summary of the calculations for combined cancer risks for exposure pathways that contain both contaminants.

Table 18 Combined Cancer Risks for PAHs and Dioxin

Exposure Pathway	PAHs Estimated Cancer Risk Range	Dioxin Estimated Cancer Risk Range	Total Estimated Cancer Risk Range
On-site surface soil	5.6E-05 to 4.5 E-06	3.6E-06 to 3.7 E-07	6.0E-05 to 4.9E-06
Off-site surface soil	5.4E-05 to 6.7 E-07	7.9E-06 to 3.9 E-06.	6.2E-05 to 4.6E-06
Off-site sediment	8.3E-05 to 3.5 E-06	2.5E-06 to1. 3 E-06	8.6E-05 to 4.8E-06

We interpret this as a low increased lifetime risk of developing cancer. As described in earlier in this document, ATSDR used very conservative exposure assumptions for the risk calculations so the actual cancer risks are likely lower than the estimated risks. Therefore, ATSDR considers that incidental ingestion of contaminants at the site was not expected to harm people's health.

Children's Health Considerations

ATSDR recognizes that infants and children might be more vulnerable than adults to exposures to contaminated air, water, soil, or food. This potential vulnerability results from the following factors:

(1) Children are more likely to play outdoors and bring food into contaminated areas;

(2) Children participate in activities and movement that make them more likely to contact dust and soil;

(3) Children's small body size results in higher doses of chemical exposure per kilogram of body weight; and

(4) Children's developing body systems can sustain permanent damage if toxic exposures occur during critical growth stages.

Because children depend completely on adults for risk identification and management decisions, ATSDR is committed to evaluating children's special interests at the site. ATSDR estimated children's exposures conservatively, using exposure assumptions for six age groups (see Appendix D for detailed exposure evaluation assumptions). ATSDR used health guidelines that are protective of children. CVs used for this health consultation were intended to represent exposures that could be continued for a lifetime for the general population—including potentially susceptible subgroups such as children—without appreciable health risks.

Health Outcome Data

Health outcome data can provide a more thorough evaluation of the public health implications of a given exposure. Health outcome data can include mortality information (e.g., the number of people dying, or who have died, of a certain disease) or morbidity information (e.g., the number of people who have a certain disease or illness). The review is most informative when:

(1) A completed human exposure pathway exists;

(2) Levels of potential contaminant exposures are high enough to cause measurable health effects;

(3) Sufficient time has passed since exposure occurred for the disease to have developed;

(4) The number of people affected is high enough for the health effect to be measured; and

(5) A database is available to identify rates of diseases plausibly associated with the exposure for populations of concern.

ATSDR did not conduct a comprehensive review of health outcome data for this site because

(1) Incidental ingestion of contaminated soil, sediment and surface water at the site is not expected to cause harmful non-cancer health effect;

(2) The excess cancer risks for residents at this site for adults and children are low or not increased, therefore it would be unlikely to see any increased cancer cases in the small population; and

(3) Limited information was available to identify rates of diseases plausibly associated with the exposure for populations of concern.

Uncertainty and Limitations in Deciding Harmful Effects

ATSDR wants to protect all people from harmful chemicals, thus we used conservative exposure parameters to estimate the highest likely dose in a population. For example, we assumed that some people were exposed to the maximum concentrations of a chemical for extended periods (as long as 30 years for non-cancer–causing exposures and 70 years for cancer-causing exposures). Some uncertainty exists in deciding whether harmful effects are expected, because uncertainty exists in estimating the chemical dose in people. For example, we are not sure exactly how much soil people ingest daily. Uncertainty also comes from determining the weight to use for various age groups and determining the chemical concentration in soil. In addition, uncertainty could exist in the human and animal studies that identify the chemical doses that cause harmful effects or the doses that do not cause harmful effects; this uncertainty varies with each chemical. When an MRL is exceeded or unknown, the estimated chemical dose in people is compared to the doses from human and animal studies. ATSDR uses this comparison, with a review of other information in our chemical-specific toxicological profile, to decide what harmful effects might be expected. The following uncertainties are specific to the ACW site.

(1) Environmental samples were collected at different periods; some were collected many years ago. Technology and method changes may affect the accuracy of environmental data. In addition, we assumed the limited number of off-site soil samples were representative of the residential soil exposures.

(2) This PHA evaluated those exposures associated with incidental ingestion exposures. ATSDR did not evaluate air exposure pathway because we do not have sampling data to evaluate this past potential exposure pathway.

(3) ATSDR calculated the estimated cancer risk using the cancer slope factor (CSF) for BaP, which may not be directly applicable to risk estimation for the wider range of PAHs

included in the derivation of the BaP equivalent calculation [Fitzgerald et al]. The risk calculation assumes that 100% of the BaP ingested is absorbed and probably overestimates the actual dose and resulting cancer risk.

Community Health Concerns

As part of the public health assessment process to investigate exposures to contamination, ATSDR staff participated in public meetings, reviewed site documents, and conducted in-person interviews to understand community members' concerns regarding the contamination, investigation, and remediation of the site. ATSDR also communicated with EPA about issues from the community.

In 2001, ATSDR regional operations staff attended a meeting conducted by EPA in Louisville, Mississippi. During the meeting, the residents expressed the following concerns related to the site.

Can contaminants found at the site cause rashes and stomach ailments?

Potentially, some of the contaminants found on and near the ACW site can cause rashes and stomach ailments. However, based on the information reviewed by ATSDR and the assumed exposure scenarios, exposure to site-related contaminants should not cause rashes or stomach ailments under normal circumstances. The main contaminants of concern at the site are the carcinogenic PAHs. For the ACW site, all PAHs concentrations were below levels that may cause non-cancer adverse health effects. In addition to contaminant exposures, other factors, including lifestyle, nutritional status, sex, age, family traits, and state of health, could cause adverse non-cancer or cancer health effects.

How will bad odors from the site while it was in operation affect me and my family?

There is no air-monitoring data collected when the site was in operation. In general, creosote is a mixture of PAHs; exposure to these vapors can irritate the respiratory tract. Exposure to high levels of the vapors also can cause sun sensitivity and damage to skin such as reddening, blistering, or peeling. Long-term exposure to creosote can cause lung cancer [ATSDR 2002]. ATSDR does not have exposure data for that period of time, therefore, we cannot evaluate the past exposures. You should discuss your concerns with a health care provider.

Conclusions and Recommendations

ATSDR reached the following conclusions in the PHA.

1. *Past exposures to on-site surface-soil PAHs, dioxin and dibenzofuran contaminations: ATSDR concluded that incidental ingestion of contaminants in surface soil on site by trespassers was not expected to harm people's health.*

Surface-soil samples collected on-site from 1999 to 2009 revealed PAH levels ranging from non-detect to 612.4 mg/kg. Dioxin concentrations ranged from 2.7 to 2,331 ng/kg. Concentrations of dibenzofuran ranged from non-detect to 1,400 µg/kg. Conservative exposure dose calculations indicated that PAHs, dioxin and dibenzofuran levels were below levels known to result in non-cancer harmful health effects. The estimated cancer risks (PAHs and dioxin combined) ranged from 6.0 E-05 to 4.9E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 6 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants in surface soil on site by trespassers was not expected to harm people's health.

2. *Off-site surface soil PAH, dioxin and dibenzofuran: ATSDR concluded that incidental ingestion of contaminated surface soil by residents in their yards is not expected to harm people's health.*

Surface-soil samples collected off-site near the facility from 1999 to 2009 revealed PAH levels ranging from negligible to 0.158 mg/kg. Surface-soil samples collected off-site near the facility from 1999 to 2009 revealed dioxin levels ranging from 4.9 to 7.8 ng/kg. One sample contained dibenzofuran at a concentration of 78 µg/kg (0.078 mg/kg). Using conservative residential exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful effects. The range of excess cancer risks (PAHs and dioxin combined) for residents is from 6.2E-05 to 4.6 E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 6 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants in surface soil on site by residents was not expected to harm people's health.

3. *Off-site Hughes Creek sediment PAHs, dioxin and dibenzofuran: ATSDR concluded that incidental ingestion of contaminated sediment in Hughes Creek by residents during occasional recreational activities is not expected to harm people's health.*

Concentrations of PAHs ranged from non-detect to 215.7 mg/kg. Concentrations of dioxin ranged from 0.474 to 44 ng/kg. Concentrations of dibenzofuran range from negligible to 440 mg/kg. ATSDR assumed that adults and children (aged 6 to 21 years) were exposed for 105

days (7 months and every other day) per year. The exposure period is based upon the assumption that adults or children played in the creek in the warmer months of the year (from April to October) every other day. Using conservative exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful effects. The estimated cancer risks (PAHs and dioxin combined) ranged from 8.6E-05 to 4.8E-06. Stated another way out of 1,000,000 people exposed to the same level over the same amount of time, we estimate that less than one to 9 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants during occasional recreational activities was not expected to harm people's health.

4. *Off-site Hughes Creek surface-water PAHs and dibenzofuran: ATSDR concluded that incidental ingestion of contaminated surface water in Hughes Creek by residents during occasional recreational activities is not expected to harm people's health.*

   Concentrations of PAHs ranged from non-detect to 1.64 µg/L. Dibenzofuran was found in only one sample at the concentration of 3 µg/L. ATSDR assumed that adults and children (aged 6 to 21 years) were exposed for 105 days (7 months and every other day) per year. The exposure period is based upon the assumption that adults or children played in the creek in the warmer months of the year (from April to October) every other day. Using conservative exposure assumptions, all estimated exposure doses were below levels known to result in non-cancer harmful effects. The estimated cancer risks for PAHs ranged from 5.4E-05 to 1.6E-06. Stated another way, out of 100,000 people exposed to the same level over the same amount of time, we estimate that less than one to 5 additional cases of cancer might occur due to the exposure. We also interpret this as a low increased lifetime risk of developing cancer. Therefore, ATSDR considers that incidental ingestion of contaminants during occasional recreational activities was not expected to harm people's health.

5. - *ATSDR cannot conclude whether eating fish from Railroad Lake and breathing outdoor air in the past could harm people's health because the information we need to make a decision is not available.*

   Railroad Lake was used for fishing in the past and the lake was drained during remediation. There was one fish sample from Railroad Lake and not enough information to evaluate the past exposure. Residents complained about strong odors while the facility was in operation. Inhalation of creosote components was a potential past completed exposure pathway. No data were collected in the past for evaluation.

6. - *ATSDR concluded that ground water, indoor air, and subsurface soil at the site are not expected to harm people's health because those exposure pathways are eliminated.*

   Residents are not drinking groundwater but use public water in the area, and municipal wells located near the ACW site are not contaminated. No private wells were used in this area. A deep clay layer in the aquifer prevents the contaminants moving into the municipal wells. Contaminated soil at the site of that potential future industrial warehouse area of the site was removed. The potential for migration of vapors into indoor structures is unlikely. For off-site residents, the levels of VOCs that were present in the groundwater were too low to cause a

concern for vapor intrusion. Because the subsurface contamination is beneath the ground, only people engaged in earth-moving activities should be exposed to this subsurface contamination. In addition, the on-site remediation activities have been completed. Most of the on-site contaminated soils have been removed and sent to a permitted landfill.

RECOMMENDATIONS

ATSDR recommends that:

EPA continue routine environmental monitoring activities for the ACW site.

Public Health Actions Planned:

- As needed, ATSDR will update this document, or prepare a new document, to reflect potential future sampling results and site remediation activities in relation to any completed or potential exposure pathways identified in this PHA.

Site Team

Jane Zhu
Environmental Health Scientist
Eastern Branch
Division of Community Health Investigations

Carl Blair
Regional Representative
Eastern Branch
Division of Community Health Investigations

References

[ATSDR] Agency for Toxic Substances and Disease Registry. 1996 September. ToxFAQs for Polycyclic Aromatic Hydrocarbons (PAHs). Atlanta: US Department of Health and Humans Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1998. Toxicological profile for chlorinated dibenzo-*p*-dioxins. Atlanta: US Department of Health and Human Services, Public Health Service. Available at http://www.atsdr.cdc.gov/toxprofiles/tp104.pdf [Accessed 1 Jan 2014].

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Agency for Toxic Substances and Disease Registry. Summary report for the ATSDR soil-pica workshop. Atlanta: US Department of Health and Human Services, Public Health Service. Available from: http://www.atsdr.cdc.gov/NEWS/soilpica.html, March 2001.

[ATSDR] Agency for Toxic Substances and Disease Registry. Toxicological profile for wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles. Atlanta: US Department of Health and Human Services; 2002 September.

[ATSDR] Agency for Toxic Substances and Disease Registry. Guidance Manual for the Assessment of Joint Action of Chemical Mixtures. Atlanta: US Department of Health and Human Services; 2004.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005. Public health assessment guidance manual (update). Atlanta: US Department of Health and Human Services; Jan. Available at URL: http://www.atsdr.cdc.gov/HAC/PHAManual/

[ATSDR] Agency for Toxic Substances and Disease Registry. 2009. Case Studies in Environmental Medicine. Polycyclic Aromatic Hydrocarbons (PAHs)
What Health Effects Are Associated With PAH Exposure? Available at URL:
http://www.atsdr.cdc.gov/csem/csem.asp?csem=13&po=11

[ATSDR] Agency for Toxic Substances and Disease Registry. 2012 November. Addendum to the toxicological profile for chlorinated dibenzo-*p*-dioxins. Atlanta: US Department of Health and Human Services, Public Health Service. Available at http://www.atsdr.cdc.gov/toxprofiles/cdds_addendum.pdf [Accessed 28 Jan 2014].

[ATSDR] Agency for Toxic Substances and Disease Registry. 2014. May 9th. Interim Guidance--Using California EPA's (Cal EPA) oral cancer potency information for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and dioxin-like compounds (DLCs)

[Baccarelli et al.] Baccarelli A, Giacomini SM, Corbetta C, *et al*. 2008. Neonatal thyroid function in Seveso 25 years after maternal exposure to dioxin. PLoS Med 5;e161.

[Barltrop] Barltrop D. The prevalence of pica. American Journal of Diseases of Children 1966;112:116-123.

[Braun JM et al] Braun JM, Kahn RS, Froehlich T, Auinger P, Lanphear BP. 2006. Exposure to environmental Toxicants and attention deficit hyperactivity disorder in US children. Environ Health Perspectives 114(12); 1904-1909.

[Calabrese and Stanek] Calabrese EJ, Stanek EJ. 1993. Soil-pica: not a rare event. J. Environmental Science and Health A28(2):373-284.
[Calabrese and Sorenson Calabrese EJ, Sorenson AJ. 1977. The health effects of PCBs with particular emphasis on human high risk groups. Rev Environ Health 2:285-304.

[Calabrese et al] Calabrese EJ, Barnes RB, Stanek ES, Pastides H, *et al.* 1989. How much soil do young children ingest: An epidemiologic study Regulatory Toxicology and Pharmacology 10:123-137.

[CalEPA] California Environmental Protection Agency. 2005. Air Toxics Hot Spots Program, Risk Assessment Guidelines, Part II, Technical Support Document for Describing Available Cancer Slope Factors. Available at: http://oehha.ca.gov/air/hot_spots/pdf/May2005Hotspots.pdf. Accessed 7 January 2014.

[Fitzgerald et al] Fitzgerald, DJ, Robinson NI, and Pester BA. Application of Benzo (a) pyrene and Coal Tar Tumor Dose-Response Data to a Modified Benchmark Dose Method of Guideline Development. Environmental Health Perspectives 112(14): 1341-1346. 2004.

[Jewell] Frank J. Jewell, Environmental Scientist, Tetra Tech EM Inc. Telephone conversation with Wilson Webb, Assistant Manager, Louisville Utilities, November 3, 1999

[MDEQ] Mississippi Department of Environmental Quality. Site Inspection Priorization Report, American Creosote Works, Inc. Jackson, Mississippi. 1994

[Mocarelli et al.] Mocarelli P, Gerthoux PM, Patterson DG Jr, *et al*. 2008. Dioxin exposure, from infancy through puberty, produces endocrine disruption and affects human semen quality. Environ Health Perspect 116;70-77.

[Mocarelli et al.] Mocarelli P, Gerthoux PM, Ferrari E, *et al.* 2000. Paternal concentrations of dioxin and sex ratio of offspring (Comment in: Lancet 355(9218): 1838-1839). Lancet. 355(9218);1858-1863.

[L.W. Stephenson, W.N. Logan, and G.A. Waring.] The Groundwater Resources of Mississippi" Water Supply Paper 576, U.S. Department of Interior, U.S. Geological Survey. 1928.

[NLM] *HSDB (Hazardous Substances Data Bank)*, Bethesda, MD, National Library of Medicine, searched March 2000 [Record No. 2163]

[Robischon] Robischon P. 1971. Pica practice and other hand-mouth behavior and children=s developmental level. Nursing Research 20:4-16.

[Shellshear] Shellshear ID. 1975. Environmental lead exposure in Christchurch children: soil lead a potential hazard. New Zealand Medical Journal 81;382-386.

[Tetra Tech] Tetra Tech EM Inc. Trip Report. American Creosote Works site, Louisville, Winston County, Mississippi. March 1999.

[Tetra Tech] Tetra Tech EM Inc. Final Expanded Site Inspection Report. American Creosote Works site, Louisville, Winston County, Mississippi. May 2000.

[USEPA] U.S. Environmental Protection Agency. Integrated Risk Information System. Available online at http://www.epa.gov/iris/subst/0429.htm

[USEPA] U.S. Environmental Protection Agency. Regional Screening Levels for Chemical Contaminants at Superfund Sites. Available online at http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm

[USEPA] U.S. Environmental Protection Agency. *Health Effects Assessment for Dibenzofuran*. EPA/600/8-88/026. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. 1988.

[USEPA] U.S. Environmental Protection Agency. 2000. Hazard ranking system documentation record, American Creosote Works, Inc. EPA Region IV, Atlanta, Georgia.

[USEPA] U.S. Environmental Protection Agency. 2007a. Final Investigation Report, American Creosote Works site, Louisville, Winston County, Mississippi. EPA Region IV, Atlanta, Georgia.

[USEPA] U.S. Environmental Protection Agency. 2007b. Feasibility Study, July 2007. Creosote Works site, Louisville, Winston County, Mississippi. EPA Region IV, Atlanta, Georgia.

[USEPA] U.S. Environmental Protection Agency. 2007c. Provisional Peer Reviewed Toxicity Values for Dibenzofuran. (CASRN 132-64-9). http://hhpprtv.ornl.gov/issue_papers/Dibenzofuran.pdf

[USEPA] US Environmental Protection Agency. 2008. Child-Specific Exposure Factors Handbook (Final Report). Washington, DC, EPA/600/R-06/096F, 2008. Available online at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=199243
Georgia.

[USEPA] U.S. Environmental Protection Agency. 2009. Final Remedial Design Basis of Design Report, August 2009. Creosote Works site, Louisville, Winston County, Mississippi. EPA Region IV, Atlanta, Georgia.

[USEPA] US Environmental Protection Agency. 2011. Exposure Factors Handbook: 2011 Edition (Final). Oct. Office of Research and Development, National Center for Environmental Assessment, Washington, DC. USEPA/600/R-09/052A. Available online at http://www.USEPA.gov/ncea/efh/report.html

[USEPA] US Environmental Protection Agency. 2012. 2, 3, 7, 8-tetrachlorodibenzo-*p*-dioxin (TCDD). Integrative Risk Information System (IRIS), US Environmental Protection Agency. Available at http://www.epa.gov/iris/subst/1024.htm#oralrfd . Accessed 23 Jan 2014.

[USDHUD] U.S. Department of Housing and Urban Development. Flood Insurance Rate Map for the City of Louisville, Winston County, Mississippi. June 1978.

[USDHHS] U.S. Department of Health and Human Services. Hazardous Substances Data Bank (HSDB, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.

[USEPA] U.S. Environmental Protection Agency. *Deposition of Air Pollutants to the Great Waters.* First Report to Congress. EPA-453/R-93-055. Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1994.

[USEPA] U.S. Environmental Protection Agency. *Integrated Risk Information System (IRIS) on Dibenzofuran*. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[Vermeer and Frate] Vermeer DE, Frate DA. 1979. Geophagy in rural Mississippi: environmental and cultural contexts and nutritional implications. American Journal of Clinical Nutrition 32:2129-2135.

[Watanabe] Watanabe, T. & Hirayama, T. (1992) Mutagenicity of nitro derivatives produced by exposure of dibenzofuran to nitrogen oxides. *Mutat. Res.*, **283**(1), 35-43

[Wong] Wong MS. 1988. The role of environmental and host behavioral factors in determining exposure to infection with Ascaris lumbriocoides and Trichuris trichlura. Ph.D. Dissertation, Faculty of Natural Sciences, University of the West Indies.

Appendix A. Explanation of Evaluation Process

In evaluating environmental data for the ACW site, ATSDR followed the general evaluation

Process as described below:

Comparison Values and Screening Process

To evaluate environmental data for the ACW site, ATSDR used comparison values (CVs) to determine which chemicals should be examined more closely. CVs are contaminant concentrations found in a specific media (air, soil, or water) that are not likely to cause adverse effects to people exposed to it. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of air, water, and soil that a person might inhale or ingest each day. CVs are generated to be conservative. CVs are not intended as environmental clean-up levels, or to indicate that health effects occur at concentrations that exceed these values.

CVs are set at concentrations below which no known or anticipated adverse human health effects are expected to occur. Different CVs are developed for cancer and non-cancer health effects. Non-cancer CV levels are based on valid toxicological studies for a chemical, with appropriate safety factors included, and the assumption that small children and adults are exposed every day. Cancer CV levels are based on a one-in-a-million excess cancer risk for an adult exposed to contaminated soil or contaminated drinking water every day for 70 years. Cancer-based CVs are calculated by using the U.S. Environmental Protection Agency's (EPA's) oral cancer slope factor (CSF) or inhalation risk unit. For chemicals for which both cancer and non-cancer CVs exist, we use the lower level to be protective. If a contaminant level exceeds a CV, it does not mean that health effects will occur, just that more evaluation is needed.

The following CVs were used in preparing this document.

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

Environmental Media Evaluation Guides (EMEGs) are concentrations of contaminants in water, soil, or air that are unlikely to produce any appreciable risk of adverse, non-cancer effects over a specified duration of exposure. EMEGs are derived from ATSDR minimal risk levels by factoring in default body weights and ingestion rates. ATSDR computes separate EMEGs for acute (≤14 days), intermediate (15–364 days), and chronic (>365 days) exposures.

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

Reference Media Evaluation Guides (RMEGs) are estimated contaminant concentrations in a medium where non-cancer health effects are unlikely. RMEGs are derived from EPA's reference dose (RfD); RfDs can be found at http://www.epa.gov/iris.
Regional Screen Levels (RSLs) are concentrations of chemical contaminants used by EPA as risk-based screening levels at hazardous waste sites. RSLs are calculated using the latest toxicity values, default exposure assumptions, and physical and chemical properties.

Determining Exposure Pathways

ATSDR identifies human exposure pathways by examining environmental and human components that might cause exposure to contaminants. A pathway analysis considers of five principal elements: a source of contamination, transport through an environmental medium, a point of exposure, a route of human exposure, and an exposed population. Completed exposure pathways are those for which the five principal elements exist, and exposure to a contaminant has occurred in the past, is now occurring, or will occur in the future. Potential exposure pathways are those for which exposure is possible, but one or more of the elements is not clearly defined, and exposure to a contaminant could have occurred in the past, could be occurring now, or could occur in the future. The identification of an exposure pathway does not imply that health effects will occur, and exposures might or might not be substantive. Therefore, even if exposure has occurred, is now occurring, or likely will occur in the future, human health might not be affected.

ATSDR reviewed site history, information on site activities, and the available sampling data. Based on this review, completed exposure pathways at the ACW site include incidental ingestion of contaminated surface soil, surface water, and sediment by occasional trespassers and nearby residents. ATSDR eliminated drinking water, air, biota and subsurface soil exposure pathways for the site.

Evaluating Public Health Implications

The next step of the process is to evaluate further those contaminants present at levels above the CVs to determine whether they may be a health hazard, given the specific exposure situations at this site. We calculate children and adult exposure doses for the site-specific exposure scenario using our assumptions of who goes on the site and how often they are exposed to the site contaminants. The amount of chemical that is swallowed or gets absorbed through the skin is called a dose. A detailed explanation of the calculation of estimated exposure doses is presented in Appendix D. Exposure doses are calculated in units of milligrams per kilograms per day (mg/kg/day). We conducted separate calculations to account for non-cancer and cancer health effects, if applicable, for each chemical based on the health effects reported for that chemical. Some chemical exposures are associated with non-cancer health effects, but are not associated with cancer-related health effects.

How non-cancer health effects are evaluated

The exposure doses calculated for each individual chemical are compared to an established health guideline, such as a MRL (Minimal Risk Level) or RfD (Reference Dose), to assess whether adverse health effects are expected. These health guidelines, developed by ATSDR and USEPA respectively, are chemical-specific values that are based on the available scientific literature and are considered protective of human health. Non-cancer effects, unlike cancer-related effects, are believed to have a threshold, that is, a dose below which adverse health effects will not occur. Because of these circumstances, the current practice for deriving health guidelines is to identify, usually from animal toxicology experiments, a No Observed Adverse Effect Level (NOAEL) or a lowest-observed-adverse-effect level (LOAEL). NOAEL is the experimental exposure level in animals (and sometimes humans) at which no adverse effect is observed. LOAEL is the lowest concentration or amount of a substance found by experiment or observation that causes an adverse health effect. The NOAEL and LOAEL are then modified with an uncertainty (or safety) factor, which reflects the degree of uncertainty that exists when experimental animal data are extrapolated to the general human population. The design of the uncertainty factor incorporates various factors such as sensitive subpopulations (for example, children, pregnant women, and the elderly), extrapolation from animals to humans, and the completeness of available data. Exposure doses at or below the established health guideline are not expected to cause adverse health effects because these values are much lower (and more protective of human health) than doses that do not cause adverse health effects in laboratory animal studies. For non-cancer health effects, the health guidelines are described in more detail in below. It is important to consider that the methods used to develop these health guidelines do not provide any information on the presence, absence, or level of cancer risk. Therefore, a separate evaluation is necessary to determine the potential risks from cancer-causing chemicals detected at this site.

Minimal Risk Levels (MRLs) – developed by ATSDR

ATSDR has developed MRLs for contaminants commonly found at hazardous waste sites. The MRL is an estimate of daily exposure to a contaminant below which non-cancer, adverse health effects are unlikely to occur. MRLs are developed for different routes of exposure such as inhalation and ingestion, and for lengths of exposure such as acute (less than 14 days), intermediate (15–364 days), and chronic (365 days or more). A complete list of the available MRLs can be found at http://www.atsdr.cdc.gov/mrls.html.

References Doses (RfDs) – developed by EPA

A reference dose (RfD) is an estimate of the daily, lifetime exposure of human populations to a possible hazard that is not likely to cause non-cancerous health effects. The design of the RfD considers exposures to sensitive sub-populations, such as the elderly, children, and the developing fetus. EPA has developed their RfDs using information from the available scientific literature and has calculated them for oral and inhalation exposures. A complete list of EPA's available RfDs can be found at http://www.epa.gov/iris.

If the estimated exposure dose for a chemical is less than the health guideline value, the exposure likely will not cause non-cancer health effects. If the calculated exposure dose is greater than the health guideline, the exposure dose is compared to known toxicological values for the particular chemical; this circumstance is discussed in more detail in the text of the PHA. The known toxicological values are doses derived from human and animal studies that are presented in the ATSDR Toxicological Profiles and EPA's Integrated Information System (IRIS). A direct comparison of site-specific exposure doses to study-derived exposures and doses that cause adverse health effects is the basis for deciding whether health effects likely will occur. This in-depth evaluation is performed by comparing calculated exposure doses with known toxicological values, such as the NOAEL and the LOAEL from studies used to derive the MRL or RfD for a chemical.

How cancer risk is evaluated

Information about the increased risk of cancer from exposure to these chemicals is also provided in each exposure scenario. Cancer is a complex subject, and some background information is provided before discussing cancer evaluations of specific chemicals. The probability that U.S. residents will develop cancer at some point in their lifetime is 1 in 2 for men (44.9 %) and 1 in 3 (38.5%) for women. This is considered the background risk of developing cancer. Stated another way, half of all men and one-third of all women will develop some type of cancer in their lifetime. This is based on medical data collected on all types of cancer, regardless of whether the cause was identified, the case was successfully treated, or the patient died (directly or indirectly) of the cancer.

Factors that play major roles in cancer development include:

- lifestyle (what we eat, drink, smoke; where we live);
- exposures to natural light (sunlight) and medical radiation;
- workplace exposures;
- drug use;
- socioeconomic factors; and
- chemicals in our air, water, soil, or food.

Infectious diseases, aging, and individual susceptibilities such as genetic predisposition are also important factors in cancer development.

We rarely know the environmental factors or conditions responsible for cancer onset and development. We have some understanding of cancer development for some occupational

exposures or for the use of specific drugs. Overall cancer risks can be reduced by eating a balanced diet, getting regular exercise, having regular medical exams, and avoiding high-risk behaviors such as tobacco use and excessive alcohol consumption. Using proper safety procedures, appropriate personal protective equipment, and medical monitoring programs can decrease workplace cancer risks.

To calculate a population's cancer estimate, ATSDR uses a quantitative risk assessment method. Using this method, site-specific doses and concentrations of cancer-causing contaminants are multiplied by EPA's cancer slope factor (CSF). Some cancer slope factors are derived from human studies; others are derived from laboratory animal studies involving contaminant doses much higher than people encounter in the environment. Using animal data requires extrapolation of the cancer potency obtained from these studies of high-dose exposures most people might not experience, which involves much uncertainty. The resulting risk of cancer is called an estimated excess cancer risk because it is the risk of cancer greater than the background risk of cancer that already exists (as mentioned above). This additional estimated cancer risk from chemical exposures is often stated as 1E-04 (the same as 1×10^{-4}), 1E-05, or 1E-06. Therefore, the excess cancer risk is between 0 and some number for every 10,000, 100,000, or 1,000,000 exposed people. For example, an estimated cancer risk of 2E-06 represents the possibility of 2 excess cancer cases in a population of 1 million. Put another way, 2×10^{-6} means that in a population of 1 million people exposed to a specific dose of a cancer-causing substance over a lifetime, 2 additional cases of cancer may occur because of the exposure. The "one-in-a-million" risk level is generally regarded as a very low increased risk. In a small exposed population, proving that cancer cases in a community are caused by chemical exposures is difficult, especially given that large number of people can get the same type cancer from other causes.
An estimated additional cancer risk of 1×10^{-4} means that in a population of 10,000 people exposed for a lifetime to a certain chemical dose, between zero and one additional cancer case may occur. Although a "one-in-ten thousand" risk level may be viewed as an increased level of risk, understanding the exposure assumptions for that calculation provides a more realistic view of the actual risk. In general, ATSDR use very conservative exposure assumptions when site-specific exposure parameters are not available. For example, for off-site sediment exposures, ATSDR assumed that adults and children would be exposed for 105 days (7 months and every other day) per year for 33 years. Since some sediment samples were collected from 0-12" and we consider that people are usually only exposed to the top few inches of sediment, we multiplied the results by 4 to represent the exposure. In addition, ATSDR used the 95% upper confidence level (UCL) concentration of the maximum likelihood mean (MLE) of the environmental data as the EPCs for dose calculation. Those assumptions are very conservative and are likely to overestimate exposures.

Appendix B. Glossary of Terms

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

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs only once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Adverse health effect
A change in body function or cell structure that might cause disease or health problems.

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

Cancer risk
A theoretical risk for cancer development if exposure to a substance occurs every day for 70 years (a lifetime exposure). The true risk might be lower or higher.

Carcinogen
A substance that causes cancer.

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

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].

Comparison value (CV)
The calculated concentration of a substance in air, water, food, or soil that likely will not cause harmful (adverse) health effects in people exposed the substance. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be further evaluated in the public health assessment process.

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law concerning the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. The Superfund Amendments and Reauthorization Act (SARA) later amended this law [see SARA further in Glossary].

Concentration
The amount of a substance that is in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other medium.

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

Dermal
Referring to the skin; dermal absorption means passing through the skin.

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

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration (no evidence of the chemical).

Dose
The amount of a substance to which a person is exposed over a period. Dose is a measurement of exposure and is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) that a person is exposed to contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is the amount of a substance encountered in the environment. An "absorbed dose" is the amount of a substance that the body absorbs through the eyes, skin, stomach, intestines, or lungs.

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that a substance can contaminate.

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

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

Exposure
Contact with a substance by swallowing, breathing, or touching it; exposure can occur if the substance touches your skin or gets in your eyes. Exposure may be short-term [acute], intermediate, or long-term [chronic].

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

Exposure point concentration (EPC)
The concentration of a contaminant within an exposed area under acute, intermediate, or chronic scenarios during past, current, and future period of the exposure duration. The estimated EPC represents the contaminant concentration that is used to evaluate exposure.

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

Health outcome data
Information from private and public institutions on the health status of populations. Health outcome data can include disease or illness (morbidity) and death (mortality) statistics, birth statistics, tumor and disease registries, or public health surveillance data.

Ingestion
Eating or drinking a substance, or simply putting a substance in the mouth as young children often do. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
Breathing; a hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days but less than 1 year [compare with acute exposure and chronic exposure].

Metabolism
The conversion or breaking down of a substance from one form to another that occurs in a living organism.

Metabolic byproduct
Any product of metabolism.

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

Morbidity
Illness or disease; the occurrence of a disease or condition that worsens health and quality of life.

Mortality
Death; usually the cause (a specific disease or condition, or an injury) is stated.

National Priorities List for Uncontrolled Hazardous Waste Sites
(National Priorities List or NPL) EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States; updated regularly.

Point of exposure
The place where someone is exposed to a substance in the environment [see exposure pathway].

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

Prevention
Actions that reduce exposures or other risks, keep people from getting sick, or keep diseases from getting worse.

Public health assessment (PHA)
A document ATSDR uses to examine hazardous substances, health outcomes, and community concerns at hazardous waste sites to determine whether people could be harmed from exposures to those substances. The PHA also provides needed actions to protect public health.

Reference dose (RfD)
An estimate determined by EPA of the daily lifetime dose of a substance, with uncertainty or safety factors built in, that is not likely to cause harm in humans.

Risk
The probability that something will cause injury or harm.

Route of exposure
How people are exposed to a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal].

Sample
A portion or piece of a whole. A selected subset of what is being studied. In a study of people, the sample is the number of people chosen to be a part of the study from a larger population [see population]. In an environmental study, a sample (e.g., a small amount of soil or water) would be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population (number of people) or an environment (number of soil or water samples) to examine.

Source of contamination
Where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum; the first part of an exposure pathway.

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

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA was enacted and amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to investigate the health effects caused by substance exposures at hazardous waste sites; conduct activities including health education, health studies, surveillance, and health consultations; and formulate toxicological profiles.

Toxicological profile
A synopsis ATSDR issues after examining, interpreting, and summarizing information about a specific hazardous substance to determine harmful exposure levels and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance, and describes areas where further research is needed.

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

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

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

Other glossaries and dictionaries:

Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)
National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

Appendix C. Explanation of the Carcinogenic Potential for Mixtures of Polycyclic Aromatic Hydrocarbons Evaluation

Polycyclic aromatic hydrocarbons (PAHs) are a group of different chemicals that are formed during the incomplete combustion of organic substances such as coal, oil and gas, garbage, tobacco, or charbroiled meat. Hundreds of individual PAHs are thought to be present as components of complex mixtures. The most-studied PAH is benzo (a) pyrene (BaP). PAHs may occur naturally or unintentionally through manufacturing processes. For example, PAHs are present in creosote, a complex mixture of many other chemical compounds.

Because PAHs exist in complex mixtures of different chemicals, assessing the potential health effects is difficult. Several different sets of factors for assessing the relative potency of PAHs or PAH derivatives compared to BaP have been published. In 1995, ATSDR's Toxicological Profile for PAHs reported the toxicity equivalency factors (TEFs). TEFs were recommended in EPA's Provisional Guidance for Quantitative Risk Assessment of PAHs (1993). In 2005, California Environmental Protection Agency (Cal EPA) published the Potency Equivalence Factors (PEF) for risk assessment of airborne PAHs and PAH derivatives. In 2010, EPA released a draft relative potency factor (RPF) approach for assessing carcinogenicity of selected PAHs in mixtures that included more recent data and a wide range of PAH compounds. The approach is not a re-assessment of individual PAH carcinogenicity; it is a method for estimating cancer risk from exposure to PAH mixtures by summing doses of component PAHs after scaling the doses (with RPFs) relative to the potency of BaP. For non-cancerous effects, PAHs are assessed individually using available toxicological data for each specific PAH (with no modification for relative potency). For cancerous effects, cancer risk is estimated using the dose-response curve for BaP. For this document, the PEF method that converts the total PAH concentration in a sample to a total carcinogenic PAH (cPAH) concentration was used [Cal EPA 2005]. Based on BaP toxicity, this approach uses potency factors specific for each cPAH to change the concentration of that PAH to a BaP-equivalent concentration. Then, the BaP-equivalent concentration of various individual cPAHs in a soil sample are summed to give the total cPAHs for that sample; in this document, BaP equivalents are referred to as cPAHs. The following table lists the PEFs that were used to calculate the BaP equivalent for this document.

Compound	CalEPA Potency Equivalence Factor Value
Anthanthrene	*
Anthracene	*
Benz[b,c]aceanthrylene, 11-H	*
Benz[e]aceanthrylene	*
Benz[j]aceanthrylene	* -
Benz[l]aceanthrylene	* -
Benz[a]anthracene	0.1 -
Benzo[b]fluoranthene	0.1 -
Benzo[j]fluoranthene	0.1 -
Benzo[k]fluoranthene	0.1 -
Benzo[c]fluorene	* -
Benzo[g,h,i]perylene	* -
Benzo(a)pyrene	1 (index compound)
Chrysene	0.01
Cyclopenta[c,d]pyrene	*
Cyclopenta[d,e,f]chrysene, 4H-	*

Dibenz[a,c]anthracene	*
Dibenz[a,h]anthracene	0.1
Dibenzo[a,e]fluroranthene	*
Dibenzo[a,e]pyrene	1.0
Dibenzo[a,h]pyrene	10
Dibenzo[a,i]pyrene	10
Dibenzo[a,l]pyrene	10
Fluoranthene	*
Indeno[1,2,3-c,d]pyrene	0.1 -
Naphtho[2,3-e]pyrene	* -
Phenanthrene	* -
Pyrene	* -

*No Potency Equivalence Factors value available

California Environmental Protection Agency (Cal EPA). Potency Equivalence Factors (PEF) for risk assessment of airborne PAHs and PAH. http://oehha.ca.gov/air/hot_spots/pdf/May2005Hotspots.pdf

For example, for sample ACSS01E1, the individual cPAH results were multiplied by their respective PEFs to get the BaP equivalent concentrations, then the BaP-equivalent concentrations in this soil sample were summed to obtain the total cPAH of 353 ppb for the sample.

PAHs	Results(ppb)	PEF	BaP equivalent (ppb)
BENZO(A)ANTHRACENE	150	0.1	15
BENZO(A)PYRENE	240	1	240
BENZO(B)FLUORANTHENE	410	0.1	41
BENZO(K)FLUORANTHENE	200	0.1	20
CHRYSENE	210	0.01	2.1
DIBENZ(A,H)ANTHRACENE	79	0.1	7.9
INDENO(1,2,3-CD)PYRENE	270	0.1	27
Total cPHA			353

Appendix D. Estimated Exposure-Dose Calculations

To evaluate contaminants (i.e., Dioxin, PAHs, and dibenzofuran) present at levels above the CVs to determine whether they may be a health hazard, we calculate children and adult exposure doses for the site-specific exposure scenario using our assumptions of who goes on the site and how often they are exposed to the site contaminants. Exposure doses are calculated in units of milligrams per kilogram per day (mg/kg/day). We conducted separate calculations to account for non-cancer and cancer health effects, if applicable, for each chemical based on the health effects reported for that chemical. Some chemical exposures are associated with non-cancer health effects, but are not associated with cancer-related health effects.

Following is a brief explanation of how we calculated the estimated exposure doses for the site.

Exposure Dose Formulas

(1) The exposure dose formula for accidental ingestion of chemicals in soil or sediment is:

$$\textit{Ingestion Dose (ID)} = \frac{\boldsymbol{C \times IR \times EF \times ED \times CF}}{\boldsymbol{BW \times AT}}$$

Where:

ID = *ingestion dose in milligrams per kilogram per day (mg/kg/day)*

C = *concentration of contaminant in soil in milligrams per kilogram (mg/kg or ppm)*

IR = *ingestion rate in milligrams per day (mg/day)*

EF = *exposure frequency (days/year)*

ED = *exposure duration (years)*

CF = *conversion factor (10^{-6} kg/mg)*

BW = *body weight (kg)*

***AT** = averaging time, days (ED x 365 days/year)*

For example, if a child (between 2 to 6 year old) was exposed to dibenzofuran in the yard from surface soil ingestion, using an EPC of 0.003 mg/kg, ingestion rate of 200 mg/day, EF of 365 days/year, exposure duration of 4 years, conversion factor of 10^{-6} kg/mg, body weight of 17.4 kg, and averaging time of 365 x 365 days/year, we calculated a ingestion dose of 0.000168 mg/kg/day.

(2) The exposure dose formula for accidental ingestion of surface water is:

$$D = \frac{C * IR * EF}{BW}$$

Where,

***D** = exposure dose (mg/kg-day)*

***C** = contaminant concentration (mg/L)*

***IR** = ingestion rate of contaminated water (L/day)*

***EF** = exposure frequency (days/week)*

***BW** = body weight (kg)*

Site-specific incidental water ingestion rates are not available for this evaluation. To be conservative, ATSDR used daily water intake rates as incidental ingestion rates. For example, for children 6-11 years old, using the body weight of 31.8 kg, mean ingestion rate of 1404 mL/day (1.404 L/day), and EF of 2/7 (two days per week) , and contaminant concentration of 3 µg/L (0.003mg/L), the exposure dose is 0.000038 mg/kg/day.

(3) The following equation is the calculation for excess cancer risk:

$$\text{Excess Cancer Risk} = (C \times CSF \times IR \times ED)/BW \times AT$$

Where,

***C** = contaminant concentration in mg/kg or µg/L/*

CSF (mg/kg/day) = *cancer slope factor*

IR = *ingestion rate in mg/day or L/day*

ED (years) = *Exposure duration*

BW (kg) = *body weight*

AT (lifetime in years) = *78 years*

EPA CSFs can be found at http://www.epa.gov/iris.

For example, for children 6-11 years old exposed to cPAH in the yard from surface soil ingestion, using a EPC of 0.632 mg/kg, CSF of 7.3E+00 $(mg/kg/day)^{-1}$ for BaP, ingestion rate of 100 mg/day, exposure duration of six years, body weight of 31.8 kg, and averaging time of 78 years, we calculated an estimated excess cancer risk of 2.8E-06.

Exposure parameter assumptions

Table D-1. ATSDR-recommended soil and sediment ingestion rates

Age Range in Years	Mean mg/day	Upper Percentile mg/day	Mean Body Weight kg
6 weeks to <1	60	100	7.8
1 to <2	100	200	11.4
2 to <6	100	200	17.4
6 to <11	100	200	31.8
11 to <16	100	200	56.8
16 to <21	100	200	71.6

≥21	50	100	80
Special Groups			
Children with pica behavior	5,000 per event		11.4 and 17.4
Gardeners (>21 year old)	100		80
Geophagy (>21 year old)	50,000		80
Trespassers	Varies with age		Varies with age
Workers—indoor	50		80
Workers—outdoor (low soil contact)	100		80
Workers—outdoor (high soil contact)	330		80

Table D-2: Recommended Values for Water Ingestion Rates

Group	CTE (L/day)	RME (L/day)	Body Weight (kg)
Child Birth to < 1 years old	0.504	1.113	7.8
Child 1 to < 2 years old	0.308	0.893	11.4
Child 2 to < 6 years old	0.376	0.977	17.4
Child 6 to < 11years old	0.511	1.404	31.8
Child 11 to < 16 years old	0.637	1.976	56.8
Child 16 to < 21 years old	0.77	2.444	71.6
Adults (≥21 years old)	1.227	3.092	80

On-site surface soil – Occasional Trespassers

Conservatively, ATSDR made the following assumptions for our dose calculations.
The exposure assessment assumes that hypothetically a person trespasses on the site over time, beginning in childhood (aged ≥6 years) and continuing into adulthood (aged ≥21 years). The trespasser scenario assumes that these trespassing events occurred twice weekly, or 104 days/year, for a total of 16 years from 1998 to 2014, assuming exposure stopped due to remediation activities. ATSDR used 104 ppt, 24,100 ppb, and 15,900 ppb as estimated exposure point concentrations (EPCs) for dioxin, BaP equivalent, and dibenzofuran, respectively. Those EPCs were estimated with a statistical method called stratified bootstrap analysis. The following paragraph discusses the reasons for using this method.

Because the data indicate varying contaminant levels at the site over time and locations, it is unlikely that people were exposed to the highest levels of contaminants for the entire time of consideration. On-site surface-soil–sampling results indicated that the former process and storages areas have higher levels of contamination. To calculate a reasonable EPC, ATSDR ran the ProUCL program first. As seen in Table D-3 below, the ProUCL recommended large confidence intervals for cPAHs (-1,812 to 34,880 ppb), dibenzofurans (-28,155 to 49,255 ppb) and dioxins (-3.88 to 178.5ppb) based on Chebychev ratio confidence intervals.

Table D-3 – BCa Stratified Bootstrap vs. ProUCL Recommended Method Confidence Intervals for c-PAH, Dibenzofuran and Dioxins

Pollutant	**90% Bias Corrected Accelerated Bootstap Confidence Interval(ppb)**	**ProUCL Recommended (method)(ppb)**
c-PAH	13,193–24,100	-1,812-34,880 (97.5% Chebyshev
Dibenzofuran	8,191–15,900	-28,155-49,255 (97.5% Chebyshev)
Dioxins	74.69–103.96	-3.88-178.5 (95% Chebyshev)

Chebyshev ratios often provide very high estimates of the confidence interval [Helsel and Gilroy 2012]. In fact, if ProUCL had included the lower end of the confidence interval it had calculated,

the interval would have contained zero for the carcinogenic PAH's, dibenzofurans, and dioxins (Table D-3). ATSDR examined the censored boxplots and qq plots for the chemicals and noticed high values that were located proximate to former processing areas on site, indicating likely stratification in the data. To better account for the stratification in the data, the 3 highest dioxin samples, the highest dibenzofuran sample, and the highest cPAH sample were assigned to its own strata. R 3.1.2 with packages boot 1.3-14 and NADA 1.5-6 were used to perform the bootstrapping, using Kaplan-Meier estimated means with the censored dibenzofuran and cPAH data. Diagnostic plots were examined in both the unstratified and stratified bootstraps to check assumptions [Davison and Hinkley 1997] and the stratification improved performance of the bootstrap markedly. Therefore, the stratified bootstrap analysis results for cPAHs (24,100 ppb), dibenzofurans (15,900 ppb) and dioxins (103.96ppb) were used.

See Table 13, 14, and 15 in Appendix E for the summary results of the dose calculations.

Off-site surface soil exposures

Only a few off-site surface soil samples were collected. There were 9 samples for PAH, 4 samples for dioxin, and 7 samples for dibenzofuran. With the limited number of samples, ATSDR used the maximum detected results to estimate the off-site EPC. In general, people are usually only exposed to the top 3 inches of soil. The sample with the highest concentrations were collected at a depth from 0 to 12 inches, so we multiplied the results by 4 to represent the exposure. Because if all the contamination were present in the top 3 inches, and the contaminated soil was averaged with 9 additional inches of clean soil, the surface soil contamination might actually be 4 times as high as measured. See Table 11 and 12 in Appendix E for the summary results of the dose calculations.

Off-site sediment and surface water exposures

We used the most current body weights and ingestion rates recommended by the EPA for children and adults as presented in Table D-1 and D-2 [EPA 2011b]. Default exposure frequency (EF=1) and durations (9 and 33 years for mean and 95% residential occupancy period) are used. For off-site sediment and surface water exposures, ATSDR assumed that adults and children (aged 6 to 21 years) would be exposed for 105 days (7 months and every other day) per year. The exposure period is based on the assumption that adults or children would play in the creek in the warmer months of the year (from April to October) every other day. The actual time spent in the creek may be more or less than that assumed by ATSDR. Since some sediment samples were collected from 0-12 inches and ATSDR considers that people are usually only exposed to the top 3 inches of soil, we multiplied the results by 4 to represent the exposure. ATSDR used ProUCL to estimate basic statistical parameters of the environmental data. We used the 95% upper confidence level (UCL) concentration of the maximum likelihood mean (MLE) of the environmental data as the EPCs for dose calculation.

See Table 15, 16 and 17 in Appendix E for the summary results of the dose calculations.

Table D-4 is a summary of relevant statistics of environmental data for the ACW site.

References

[ATSDR 1993] Agency for Toxic Substances and Disease Registry (ATSDR). Cancer Policy Framework. Atlanta (GA): US Department of Health and Human Services; 1993.

[EPA 1989] US Environmental Protection Agency, Office of Emergency and Remedial Response. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual. Washington: US Environmental Protection Agency; 1989.

[Helsel and Gilroy 2012].

[Davison and Hinkley 1997].

Table D-4. Summary of Statistics of Environmental Data for the ACW Site

Chemical Name/Media	Number of samples	Number of detected results	Percentage of Non-detects	Minimum detected concentration	Maximum detected concentration	G.O.F Statistics (R)	95% UCL	Nonparametric 95% UCL	Comparison Value (µg/L)
cPAH_Off-site_Surface Water	16	4	75	0.16 ppb	1.64 ppb	Lognormal ROS (0.995)	1.1 ppb (Percentile Bootstrap)	NA (<20 Detects)	0.005 ppb (CREG)
Dibenzofuran_Off-site_Surface Water	16	2	87.5	3 ppb	3 ppb	NA	NA (use Maximum)	NA (<20 Detects)	7.9 ppb (RSL)
Dibenzofuran_Off-site_Sediment	43	27	37.21	38 ppb	440,000 ppb	Lognormal (0.959)	NA	41,210 ppb (KM (BCA))	73,000 ppb (RSL)
Dioxin_Off-site_Sediment	18	18	0	0.474 ppt	44 ppt	Lognormal (0.987)	65.79 ppt (Chebyshev [MVUE])	NA (<20 Detects)	5.4 ppt (CEMEG)
cPAH_Off-site_Sediment	42	36	14.29	10.9 ppb	215,700 ppb	NA	NA	17,571 ppb (KM (BCA))	96 ppb (CREG)
cPAH_Off-site_Surface Soil	9	5	44.44	4.08 ppb	158.1 ppb	NA	NA	NA (<20 Detects)	100 ppb (CREG)
Dibenzofuran_Off-site_Surface Soil	10	1	90	78 ppb	78 ppb	NA	NA	NA	78,000 ppb(RSL)
Dioxin_Off-site_Surface Soil	4	4	0	4.9 ppt	7.8 ppt	NA	NA	NA	5.4 ppt (CEMEG)
Dioxin_On-site_Surface Soil	140	140	0	2.71 ppt	2,331 ppt	NA	Bootstrapped *stratified* Confidence Intervals 104 ppt	95% Chebyshev (Mean, Sd) 178.5 ppt (Not used)	5.4 ppt (CEMEG)
cPHA_On-site_Surface Soil	235	217	7.66	8 ppb	612,400 ppb	NA	Bootstrapped *stratified* Confidence Intervals	21,820 ppb (KM [BCA])	100 ppb (CREG)

							24,100 ppb		
Dibenzofuran _On-site_Surface Soil	238	87	63.45	41 ppb	1,400,000 ppb	NA	Bootstrapped *stratified* Confidence Intervals 15,900 ppb	49,255 ppb (KM [Chebyshev]) (Not used)	78,000ppb (RSL)

G.O.F = goodness of fit

CEMEG = child environmental media evaluation guide

CREG = cancer risk evaluation guide

NA = not applicable

ppb = part per billion

ppt = part per trillion

RSL = regional screen value

Appendix E. Tables

Table 1. American Creosote Works Sampling Event Summary 1999 - 2009

Sampling Event	Sediment Sample #	Surface Soil Sample #	Surface Water Sample #	Subsurface Soil Sample #	Ground Water Sample #
SI	3	2	3	0	0
ESI	12	4	0	4	3
Phase I - RI	13	133	13	8	13
Phase II- RI	0	29	0	29	0
Phase III- RI	30	95	21	95	0
Phase IV- RI	2	0	0	54	23
Supplemental- RI	31	7	0	0	11
RDI	25	86	0	138	30

SI = site investigation

ESI = expanded site investigation

Phase I-RI = phase I remedial investigation

Phase II-RI = phase II remedial investigation

Phase III-RI = phase III remedial investigation

Phase IV-RI = phase IV remedial investigation

Supplemental-RI = Supplemental remedial investigation

RDI = remedial design investigation

Table 2. Dibenzofuran and PAHs in Sediment, Surface Soil, and Surface Water

American Creosote Works (1999) Results

Sample ID	Sample Medium	Dibenzofuran (mg/kg)	CV for Dibenzofuran	BaP* equivalent Total (mg/kg)	CV for BaP (mg/kg)
AC-PS-01	Sediment	ND	78 mg/kg (RSL)	ND	0.1 (CREG)
AC-SD-HC-01	Sediment	**120**	78 mg/kg (RSL)	**60.52**	0.1 (CREG)
AC-SCW-01	Surface water	3.2	7.9 µg/L (RSL)	**0.284**	0.005 µg/L (CREG)
AC-SW-PC-02	Surface water	ND	7.9 µg/L (RSL)	ND	0.005 µg/L (CREG)
AC-SW-HC-01	Surface water	ND	0.016 µg/L(RSL)	ND	0.005 µg/L (CREG)

Sample ID	Sample Medium	Dibenzofuran (mg/kg)	CV for Dibenzofuran	BaP* equivalent Total (mg/kg)	CV for BaP (mg/kg)
AC-SS-02	Soil	**170**	78 mg/kg (RSL)	**71.40**	0.1mg/kg (CREG)
AC-CSS-01	Surface soil	75	78 mg/kg (RSL)	**62.50**	0.1mg/kg (CREG)

CV = comparison value

Bolded = concentration exceeds applicable comparison value (CV)

NA = not available

ND = not detected. Numbers in parenthesis are detection limits.

*= In order to calculate the carcinogenic potential of the PAHs, each carcinogenic PAH is assigned a potency equivalent factor (PEF), which is an estimate based on its relative potency to benzo (a) pyrene (BaP). The concentration of each PAH is multiplied by its PEF, and the sum of the products is described as the BaP equivalent. See Appendix C for more details.

mg/kg = milligrams of contaminant per kilogram of soil

µg/L= micrograms of contaminant per liter of water

CREG = cancer risk evaluation guide

RSL = regional screen level

Table 3. Analytical Results Summary for Sediment, Surface Soil, and Groundwater

American Creosote Works (1999)

Sample ID	Sample Medium	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	TCDD TEQ Total** (ppm)
AC-01-SD	Sediment	ND	ND	0.000003
AC-02-SD	Sediment	3.4	**3.32**	NT
AC-03-SD	Sediment	4.2	**4.33**	NT
AC-04-SD	Sediment	0.12	**2.58**	NT
AC-05-SD	Sediment	**1600**	**488**	NT
AC-06-SD	Sediment	25	**29.2**	NT
AC-07-SD	Sediment	4.8	**39.35**	**0.000018**
AC-08-SD	Sediment	**430**	**96.5**	NT

Sample ID	Sample Medium	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	TCDD TEQ Total** (ppm)
AC-09-SD	Sediment	**110**	**53.37**	NT
AC-11-SD	Sediment	**440**	**140.9**	**0.000044**
AC-13-SD	Sediment	ND	**0.16**	NT
AC-14-SD	Sediment	16	ND	NT
AC-01-SS	Surface soil	ND	0.004	NT
AC-02-SS	Surface soil	ND	**0.118**	NT
AC-03-SS	Surface soil	ND	**0.14**	NT
AC-01-TW	Groundwater	ND	ND	NT
AC-02-TW	Groundwater	0.059	**ND**	NT
AC-03_TW	Groundwater	2.2	**0.007**	NT

Bolded = concentration exceeds applicable comparison value (CV) of 78 ppm for dibenzofuran in soil/sediment and 16 ppb in water (RSL); 0.1 ppm for Benzo(a)pyrene (BaP equivalent) in soil/sediment and 0.005 ppb in water; TCDD 5.4 ppt in soil/sediment.

NT = not tested

ND = not detected. Numbers in parenthesis are detection limits.

*= In order to calculate the carcinogenic potential of the PAHs, each carcinogenic PAH is assigned a potency equivalent factor (PEF), which is an estimate based on its relative potency to BaP. The concentration of each PAH is multiplied by its PEF, and the sum of the products is described as the BaP equivalent. See Appendix C for more details.

** Dioxin toxicity equivalence factors (TEFs) are used to calculate TCDD toxicity equivalence (TEQ).

Table 6. Summary of on-site surface soil results for PAHs, dibenzofuran, and dioxin

Contaminant	Highest Concentration Detected in on-site surface soil Sample, µg/kg	CV µg/kg	Selected for Further Evaluation?
BaP Equivalent	612,400	96 (CREG)	Yes
Dibenzofuran	140,0000	78000 (RSL)	Yes
Dioxin	2.33	0.0054 (CREG)	Yes

Note:

CV = comparison value

RMEG = reference media evaluation guide

CREG = cancer risk evaluation guide

RSL = regional screen value

µg/kg = micrograms of contaminant per kilogram of soil

Table 7. On-Site Soil – Occasional Trespasser

PAHs Cancer Risk Estimation*

Age Group (year)	Mean Body Weight (kg)	RME***Soil Intake (mg/day)	CTE** Soil Intake (mg/day)	Estimated EPC (mg/kg)	Estimated Excess Cancer Risk RME***	Estimated Excess Cancer Risk CTE**
6 to < 11	31.8	200	100	24.1	6.1E-05	3.0E-05
11 to <16	56.8	200	100	24.1	3.4E-05	1.7E-05
16 to <21	71.6	200	100	24.1	9.0E-06	4.5E-06
Combined child	NA	NA	NA	24.1	4.3E-05	2.1E-05
≥21	80	100	50	24.1	1.3E-05	6.4E-06
Combined child+adult	NA	NA	NA	NA	5.6E-05	3.1-05

EPC = Exposure Point Concentration

***** *CalEPA's potency equivalence factor (PEF) that converts the total PAH concentration in a sample to a total carcinogenic PAH concentration was used for the dose calculation.*

***CTE = Central Tendency Exposure. Refers to people who have average or typical soil intake rate.*

******* *RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average but within a realistic range of exposure.*

NA = not applicable

Combined child = the risk for the total of children of all ages

Combined child +adult = the total of all children and adults

Table 8. On-Site Soil – Occasional Trespasser

Dioxin Dose Calculations

Age Group (year)	Estimated EPC (mg/kg)	RfD mg/kg/day	Estimated RME Doses (mg/kg/day)	Estimated CTE Doses (mg/kg/day)	Estimated Excess Cancer Risk RME**	Estimated Excess Cancer Risk CTE*
6 to < 11	1.0E-04	7×10^{-10}	1.8E-10	9.3E-11	1.6E-06	7.8E-07
11 to <16	1.0E-04	7×10^{-10}	1.04E-10	5.2E-11	8.7E-07	4.3E-07
16 to <21	1.0E-04	7×10^{-10}	8.3E-11	4.1E-11	6.9E-07	3.4E-07
Combined child	1.0E-04	7×10^{-10}	1.8E-10	9.3E-11	1.6E-06	7.7E-07
≥21	1.0E-04	7×10^{-10}	3.7E-11	1.8E-11	2.0E-06	1.6E-06

Combined child & adult	1.0E-04	7 x 10^{-10}	2.1E-10	1.1E-10	3.6E-06	2.3E-06

**CTE = Central Tendency Exposure. Refers to people who have average or typical soil intake rate.*

***RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average, but are still within a realistic range of exposure.*

RfD: reference dose.

Table 9 On-Site Soil – Occasional Trespasser Dibenzofuran Dose Calculations

Age Group (year)	Estimated EPC (mg/kg)	Estimated RME Doses (mg/kg/day)	Estimated CTE Doses (mg/kg/day)	Below RfD (0.001 mg/kg/day) (Yes/NO)
6 to <11	15.9	2.8E-05	1.4E-05	Yes
11 to <16	15.9	1.6E-05	8.0E-06	Yes
16 to <21	15.9	1.3E-05	6.0E-06	Yes
≥21	15.9	5.7E-06	3.0E-06	Yes

Table 10. Summary of Off-site Soil Sampling Results

Sample ID	Sample Event	Sampling Depth	Dibenzofuran (ppb)	*BaP Equivalent (ppb)	**TCDD TEQ (ppt)	Comments
AC-01-SS	ESI	0–6"	ND(410)	158.1	NT	
AC-02-SS	ESI	0–6"	ND(410)	ND(520)	NT	
AC-03-SS	ESI	0–6"	ND(410)	ND (440)	NT	
JS-01	RI	0–12"	78	4.98	NT	***A multiple factor of 4 is used in dose calculation to represent the scenario
SS-01	SRI	0–12"	ND(470)	4.08	7.8	A multiple factor of 4 is used in dose calculation to represent the scenario
SS-02	SRI	0–12"	ND(520)	118.13	6.9	A multiple factor of 4 is used in dose calculation to represent the scenario
SS-03	SRI	0–12"	ND(440)	140.53	4.9	A multiple factor of 4 is used in dose calculation to represent the scenario

Sample ID	Sample Event	Sampling Depth	Dibenzofuran (ppb)	*BaP Equivalent (ppb)	**TCDD TEQ (ppt)	Comments
SS-04	SRI	0–12”	ND(490)	ND(490)	5.4	A multiple factor of 4 is used in dose calculation to represent the scenario
SS-05	SRI	0–12”	ND(490)	ND(490)	NT	A multiple factor of 4 is used in dose calculation to represent the scenario

ND: not detected. Numbers in parenthesis are detection limits.

NT: not tested

ESI: expanded site investigation

RI: remedial investigation

SRI: supplemental remedial investigation

ppb: parts per billion

ppt: parts per trillion

*= In order to calculate the carcinogenic potential of the PAHs, each carcinogenic PAH is assigned a potency equivalent factor (PEF), which is an estimate based on its relative potency to BaP. The concentration of each PAH is multiplied by its PEF, and the sum of the products is described as the BaP equivalent. See Appendix C for more details.

**Dioxin toxicity equivalence factors (TEFs) are used to calculate TCDD toxicity equivalence (TEQ).

*** In residential settings, people are usually only exposed to the top 3 inches of soil. If all the contamination measure in these 0-12 inch samples was present in the top 3 inches, and the contaminated soil was averaged with 9 additional inches of clean soil, the surface soil contamination might actually be 4 times as high as measured. Therefore, we multiplied the result of 0.158 mg/kg by 4 to represent the exposure.

Table 11. Off-Site Soil – Residential PAHs Dose Calculations

Age Group (year)	Estimated EPC (mg/kg)	Estimated Excess Cancer Risk RME	Estimated Excess Cancer Risk CTE
0.5 to <1	0.632	5.7E-06	3.4E-06
1 to <2	0.632	1.0E-05	5.2E-06
2 to <6	0.632	8.2E-06	4.1E-06
6 to <11	0.632	5.6E-06	2.8E-06
11 to <16	0.632	3.1E-06	1.6E-06
16 to <21	0.632	8.3E-07	4.1E-07
Combined child	0.632	3.4E-05	1.7E-05
21 + (33 years) -- 95% residential occupancy period	0.632	2.4E-06	1.2E-06
Combined child & adult	0.632	3.6E-05	1.8E-05

Table 12. Off-Site Soil – Residential

Dioxin Dose Calculations

Age Group (year)	Estimated EPC (mg/kg)	Estimated Doses RME	Estimated Doses CTE	Cancer Risk RME	Cancer Risk CTE	RfD mg/kg/day
0.5 to <1	3.1E-05	3.3E-10	2.3E-10	5.0E-07	3.0E-07	7×10^{-10}
1 to <2	3.1E-05	5.4E-10	2.7E-10	9.1E-07	4.6E-07	7×10^{-10}
2 to <6	3.1E-05	3.5E-10	1.7E-10	2.4E-06	1.2E-06	7×10^{-10}
6 to <11	3.1E-05	1.9E-10	9.8E-11	1.6E-06	8.2E-07	7×10^{-10}
11 to <16	3.1E-05	1.1E-10	5.5E-11	9.2E-07	4.6E-07	7×10^{-10}
16 to <21	3.1E-05	8.7E-11	4.4E-11	7.3E-07	3.6E-07	7×10^{-10}

Combined Cancer Risk for Child	3.1E-05	NA	NA	7.1E-06	3.6E-06	NA
21 + (33 years) - - 95% residential occupancy period	3.1E-05	3.9E-11	2.0E-11	7.8E-07	3.9E-07	NA
Combined Cancer Risk child & adult	3.1E-05	NA	NA	7.9E-6	3.6E-06	NA
(pica) 1 <2 (EF = 3 d/week	3.1E-05	5.8E-09	NA	NA	NA	
(pica) 2 <6 (EF = 3 d/week	3.1E-05	3.8E-09	NA	NA	NA	

EPC = Exposure Point Concentration

CTE = Central Tendency Exposure. Refers to people who have average or typical soil intake rate.

RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average but still within a realistic range of exposure.

NA = not applicable

Table 13. ACW Off-site Sediment Samples Analytical Results Summary

Sample ID	Sampling Event	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	*DIOXIN (TEQ) (ppt)
AC-SD-HC-01	Site Investigation	120	60.52	NT
AC-01-SD	Expanded Site Investigation	ND(0.41)	ND(0.41)	3.0
AC-02-SD	Expanded Site Investigation	3.4	**3.32**	NT
AC-03-SD	Expanded Site Investigation	4.2	**4.33**	NT
AC-04-SD	Expanded Site Investigation	0.12	**2.58**	NT
AC-13-SD	Expanded Site Investigation	ND(NA)	0.16	NT
ACSD10	Field investigation	ND(0.33)	0.173	3.1259
ACSD11	Field investigation	ND(0.33)	**0.292**	**11.6065**
ACSD12	Field investigation	ND(0.33)	**0.109**	2.07485
ACSD13	Field investigation	ND(0.33)	ND(0.33)	0.4739

Sample ID	Sampling Event	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	*DIOXIN (TEQ) (ppt)
ACSD14	Field investigation	ND(0.33)	**0.201**	**7.253**
ACSD15	Field investigation	ND(0.33)	ND(0.33)	0.56336
ACSD16	Field investigation	0.038	**0.774**	4.5744
ACSD17	Field investigation	ND(0.33)	**0.014**	1.95135
ACSD18	Field investigation	ND(0.33)	**2.341**	**38.385**
ACSD19	Field investigation	0.38	**6.69**	**8.4756**
ACSD20	Field investigation	0.31	**15.62**	**17.8625**
ACSD21	Field investigation	0.78	**28.90**	**6.01495**
ACSD22	Field investigation	ND(0.33)	ND(0.33)	**6.5355**
ACSD23	Field investigation	0.12	1.02	2.2472
ACSD24	Field investigation	ND	0.062	2.06145

Sample ID	Sampling Event	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	*DIOXIN (TEQ) (ppt)
RFSD01	Field investigation	ND(0.33)	**ND(0.33)**	1.758055
SED01	Remedial Design Investigation	0.81	**4.734**	NT
SED02	Remedial Design Investigation	0.18	**3.115**	NT
SED03	Remedial Design Investigation	0.35	**2.656**	NT
SED04	Remedial Design Investigation	0.84	**5.639**	NT
SED05	Remedial Design Investigation	0.65	**4.655**	NT
SED06	Remedial Design Investigation	0.27	**11.029**	NT
SED07	Remedial Design Investigation	ND(0.13)	**0.416**	NT
SED07-02	Remedial Design Investigation	0.34	**2.25**	NT
SED08	Remedial Design Investigation	0.17	**1.953**	NT
SED09	Remedial Design Investigation	62	**19.56**	NT

Sample ID	Sampling Event	Dibenzofuran (ppm)	BaP* equivalent Total (ppm)	*DIOXIN (TEQ) (ppt)
SED10	Remedial Design Investigation	11	**3.771**	NT
SED11	Remedial Design Investigation	0.81	**0.901**	NT
SED12	Remedial Design Investigation	0.15	**2.349**	NT
SED13	Remedial Design Investigation	4.5	**3.736**	NT
SED14	Remedial Design Investigation	3.2	**2.922**	NT
SED15	Remedial Design Investigation	0.7	**7.966**	NT
SED16	Remedial Design Investigation	0.43	**6.405**	NT
SED17	Remedial Design Investigation	ND(0.13)	**1.150**	NT
SED18	Remedial Design Investigation	0.92	**9.492**	NT

Bolded = concentration exceeds applicable comparison value (CV) of 78 ppm for dibenzofuran in soil/sediment (RSL); 0.1 ppm for BaP equivalent in soil/sediment; and dioxin 5.4 ppt in soil/sediment.

NA = not available

ND = not detected. Numbers in parenthesis are detection limits.

NT = not tested

*In order to calculate the carcinogenic potential of the PAHs, each carcinogenic PAH is assigned a potency equivalent factor (PEF), which is an estimate based on its relative potency to BaP. The concentration of each PAH is multiplied by its PEF, and the sum of the products is described as the BaP equivalent. See Appendix C for more details.

Table 14. ACW Site Surface-water Samples Analytical Results Summary

Sample ID	BaP* equivalent Total (ppm)	Dibenzofuran (ppm)	Sample Location
RFSW01	ND(0.01)	ND(0.01)	Off-site
AC-SW-HC-01	ND(NA)	ND(NA)	Off-site
ACSW02	ND(0.01)	ND(0.01)	Off-site
ACSW06	0.0032	0.001	Off-site
ACSW23	ND(0.01)	0.003	Off-site
ACSW24	ND(0.01)	ND(0.01)	Off-site
AC-SCW-01	2.84	3.2	On-site
AC-SW-PC-02	ND(NA)	ND(NA)	On-site
ACSW01	ND(0.01)	ND(0.01)	On-site
ACSW03	ND(0.01)	ND(0.01)	On-site

Sample ID	BaP* equivalent Total (ppm)	Dibenzofuran (ppm)	Sample Location
ACSW04	ND(0.01)	ND(0.01)	On-site
ACSW05	ND(0.01)	ND(0.01)	On-site
ACSW07	ND(0.01)	ND(0.01)	On-site
ACSW08	ND(0.01)	ND(0.01)	On-site
ACSW09	ND(0.01)	ND(0.01)	On-site
ACSW10	ND(0.01)	ND(0.01)	On-site
ACSW11	ND(0.01)	ND(0.01)	On-site
ACSW12	ND(0.01)	ND(0.01)	On-site
ACSW13	ND(0.01)	ND(0.01)	On-site
ACSW14	ND(0.01)	ND(0.01)	On-site
ACSW15	ND(0.01)	ND(0.01)	On-site

Sample ID	BaP* equivalent Total (ppm)	Dibenzofuran (ppm)	Sample Location
ACSW16	ND(0.01)	ND(0.01)	On-site
ACSW17	ND(0.01)	ND(0.01)	On-site
ACSW18	ND(0.01)	ND(0.01)	On-site
ACSW19	ND(0.01)	ND(0.01)	On-site
ACSW20	ND(0.01)	ND(0.01)	On-site
ACSW21	0.0001	0.003	On-site
ACSW21(Dup)	0.00001	0.003	On-site
ACSW22	ND(0.01)	ND(0.01)	On-site
ACLC01	ND(0.01)	ND(0.01)	On-site
ACLC02	ND(0.01)	ND(0.01)	On-site
ACLC03	ND(0.01)	ND(0.01)	On-site

Sample ID	BaP* equivalent Total (ppm)	Dibenzofuran (ppm)	Sample Location
ACLC04	ND(0.01)	ND(0.01)	On-site

Bolded = concentration exceeds applicable comparison value (CV) of 16 ppb in water for dibenzofuran (RSL); 0.005 ppb for BaP equivalent in water.

NA = not available

ND = not detected. Numbers in parenthesis are detection limits.

*In order to calculate the carcinogenic potential of the PAHs, each carcinogenic PAH is assigned a potency equivalent factor (PEF), which is an estimate based on its relative potency to BaP. The concentration of each PAH is multiplied by its PEF, and the sum of the products is described as the BaP equivalent. See Appendix C for more details.

Table 15. Off-site Sediment PAH Dose Calculations*

Age Group (years)	Mean Body Weight (kg)	Upper Percentile Intake (mg/day)	CTE** Intake (mg/day)	Estimated EPC (mg/kg)	Cancer Risk RME***	Cancer Risk CTE**
6 to <11	31.8	200	100	17.57	4.5E-05	2.2E-05
11 to <16	56.8	200	100	17.57	2.5E-05	1.2E-05
16 to <21	71.6	200	100	17.57	6.6E-06	3.3E-06
Combined child Exposure	NA	NA	NA	NA	7.6E-05	3.8E-05
>21 (for 33 years duration)	80	100	50	17.57	7.1E-06	3.5E-06

Combined child+adult	NA	NA	NA	NA	8.3E-05	4.2E-05

EPC = Exposure Point Concentration

CTE = Central Tendency Exposure. Refers to people who have average or typical soil intake rate.

RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average but within a realistic range of exposure

NA = not applicable

Table 16. Off-Site Sediment

Dioxin Dose Calculations

Age Group (year)	Estimated EPC (mg/kg)	Estimated Doses RME	Estimated Doses CTE	Cancer Risk RME	Cancer Risk CTE
6 to <11	6.6E-05	1.2E-10	5.9E-11	9.9E-07	5.0E-07
11 to <16	6.6E-05	6.7E-11	3.3E-11	5.6E-07	2.8E-07
16 to <21	6.6E-05	5.3E-11	2.6E-11	4.4E-07	2.2E-07
Combined Cancer Risk for Child	6.6E-05	NA	NA	2.0E-06	9.9E-07
>21 (with 33 years duration)	6.6E-05	2.2E-11	1.1E-11	6.3E-07	3.2E-07

Combined Cancer Risk for child+adult	4.1E-05	NA	NA	2.5E-06	1.3E-06

EPC = Exposure Point Concentration

CTE = Central Tendency Exposure. Refers to people who have average or typical soil-intake rate.

RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average but still within a realistic range of exposure.

NA = not applicable

Table 17. Off-Site Surface Water PAH Dose Calculations *

Age Group (year)	Mean Body Weight (kg)	Upper Percentile Intake (L/day)	CTE** Intake (L/day)	Estimated EPC (mg/L)	Cancer Risk RME***	Cancer Risk CTE**
6 to <11	31.8	1.404	0.511	1.1E-03	2.0E-05	7.1E-06
11 to <16	56.8	1.976	0.637	1.1E-03	1.5E-05	5.0E-06
16 to <21	71.6	2.444	0.77	1.1E-03	5.1E-06	1.6E-06
Combined child	NA	NA	NA	NA	4.0E-05	1.4E-05
21 +(33 years)	80	3.092	1.227	1.1E-03	3.8E-05	1.5E-05
Combined child & adult	NA	NA	NA	NA	5.4E-05	1.9E-05

** CalEPA's potency equivalence factor (PEF) that converts the total PAH concentration in a sample to a total carcinogenic PAH concentration was used for the dose calculation.*

***CTE = Central Tendency Exposure. Refers to people who have average or typical soil intake rate.*

****RME = Reasonable Maximum Exposure. Refers to people who are at the high end of the exposure distribution (approximately the 95th percentile). The RME scenario is intended to assess exposures that are higher than average but within a realistic range of exposure.*

NA = not applicable

Appendix F. Statistical Analysis of Benzo(a)pyrene Soil Samples

The figure on the following page shows the sampling grid designed to collect benzo(a)pyrene (BaP) soil samples at the American Creosote Works (ACW) Superfund Site located in Louisville, Winston County, Mississippi. The following question was posed by the ATSDR site investigation team: Were the BaP concentrations of 6-inch (sample 1) soil composite samples collectively the same as 12-inch (sample 2) soil composite samples? (Please note the question was only selectively posed for the acquired samples per the colored-hatch grids within the figure.) The summary statistics, collectively as a whole, for each sample are shown below.

SAMPLE 1: 6-INCH COMPOSITE SAMPLES

NUMBER OF OBSERVATIONS	= 88
MEAN	= 24,078.95455
MEDIAN	= 13,519.5
LO VALUE	= 621
HI VALUE	= 157,410
STANDARD DEVIATION	= 29,059.73687
STANDARD ERROR OF THE MEAN	= 3,097.778359

SAMPLE 1: 12-INCH COMPOSITE SAMPLES

NUMBER OF OBSERVATIONS	= 24
MEAN	= 25,768.2125
MEDIAN	= 15,544.5
LO VALUE	= 230.8
HI VALUE	= 90,200
STANDARD DEVIATION	= 25,097.24055
STANDARD ERROR OF THE MEAN	= 5,122.952774

To address the question posed by the site investigation team, will conduct statistical/inference testing upon the data; however, before doing so, a few assumptions must be initially noted. Since the data does not contain any censored measurements (i.e., non-detects), will only use appropriate statistical/inference tests for non-censored data. Next assume that the variances for the two samples are

equal. Will not distinguish whether the data is normally distributed or it is not normally distributed since both parametric (i.e., normal distribution) and non-parametric (i.e., non-normal distribution) inference tests will be conducted. We are testing the hypothesis that the population means are equal for the two samples; however, in general, there are three possible alternative hypotheses and rejection regions for the inference tests (see table below). Finally, will set as an inference benchmark, a level of significance (α) of 0.05 (i.e., will reject null hypothesis of equality if significance probability or p-value is smaller than 0.05). Figure bellow is the ACW Soil Sampling Grid.

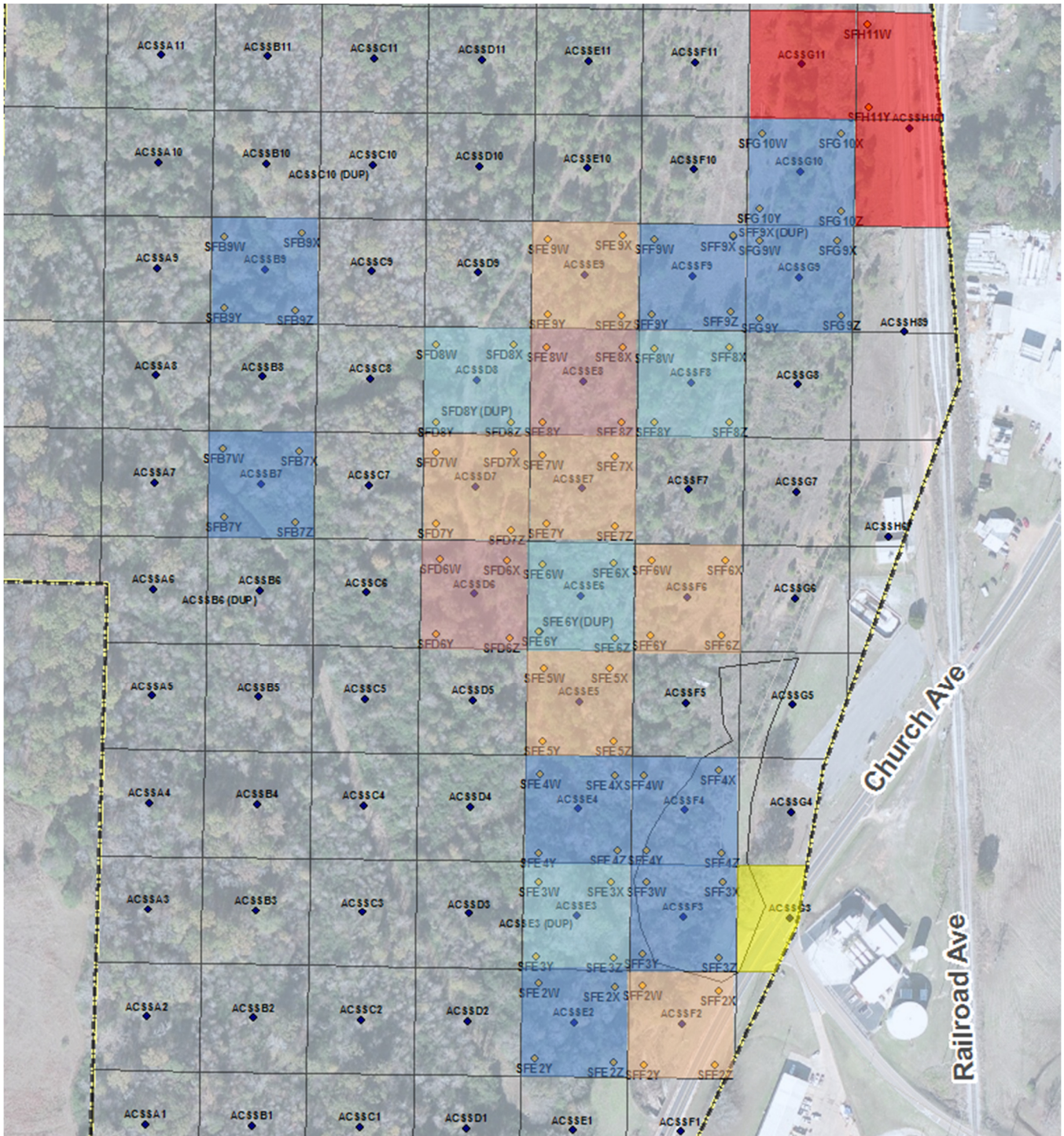

ACSSA11
ACSSB11
ACSSC11
ACSSD11
ACSSE11
ACSSF11
ACSSG11
SFH11W
SFH11Y
ACSSH10
ACSSA10
ACSSB10
ACSSC10
ACSSC10 (DUP)
ACSSD10
ACSSE10
ACSSF10
ACSSG10
SFG10W
SFG10X
SFG10Y
SFG10Z
SFF9X (DUP)
ACSSA9
SFB9W
SFB9X
ACSSB9
ACSSC9
ACSSD9
SFE9W
SFE9X
ACSSE9
SFF9W
SFF9X
ACSSF9
SFG9W
SFG9X
ACSSG9
SFB9Y
SFB9Z
SFE9Y
SFE9Z
SFF9Y
SFF9Z
SFG9Y
SFG9Z
ACSSH9
ACSSA8
ACSSB8
ACSSC8
SFD8W
SFD8X
ACSSD8
SFE8W
SFE8X
ACSSE8
SFF8W
SFF8X
ACSSF8
ACSSG8
SFD8Y (DUP)
SFD8Y
SFD8Z
SFE8Y
SFE8Z
SFF8Y
SFF8Z
ACSSA7
SFB7W
SFB7X
ACSSB7
ACSSC7
SFD7W
SFD7X
ACSSD7
SFE7W
SFE7X
ACSSE7
ACSSF7
ACSSG7
SFB7Y
SFB7Z
SFD7Y
SFD7Z
SFE7Y
SFE7Z
ACSSH7
ACSSA6
ACSSB6
ACSSB6 (DUP)
ACSSC6
SFD6W
SFD6X
ACSSD6
SFE6W
SFE6X
ACSSE6
SFF6W
SFF6X
ACSSF6
ACSSG6
SFE6Y (DUP)
SFD6Y
SFD6Z
SFE6Y
SFE6Z
SFF6Y
SFF6Z
ACSSA5
ACSSB5
ACSSC5
ACSSD5
SFE5W
SFE5X
ACSSE5
ACSSF5
ACSSG5
SFE5Y
SFE5Z
ACSSA4
ACSSB4
ACSSC4
ACSSD4
SFE4W
SFE4X
ACSSE4
SFF4W
SFF4X
ACSSF4
ACSSG4
SFE4Y
SFE4Z
SFF4Y
SFF4Z
ACSSA3
ACSSB3
ACSSC3
ACSSD3
SFE3W
SFE3X
ACSSE3
SFF3W
SFF3X
ACSSF3
ACSSG3
ACSSE3 (DUP)
SFE3Y
SFE3Z
SFF3Y
SFF3Z
ACSSA2
ACSSB2
ACSSC2
ACSSD2
SFE2W
SFE2X
ACSSE2
SFF2W
SFF2X
ACSSF2
SFE2Y
SFE2Z
SFF2Y
SFF2Z
ACSSA1
ACSSB1
ACSSC1
ACSSD1
ACSSE1
ACSSF1
Church Ave
Railroad Ave

TABLE I – INFERENCE TESTING CRITERIA

Null Hypothesis

H_0: $\mu_1 = \mu_2$

Alternative Hypothesis	Rejection Region	Test Type
H_a: $\mu_1 \neq \mu_2$	$\|T\| > t_{1-\alpha/2,\nu}$	*Two-Sided Test*
H_a: $\mu_1 > \mu_2$	$T > t_{1-\alpha,\nu}$	*Upper-Side Test*
H_a: $\mu_1 < \mu_2$	$T < t_{\alpha,\nu}$	*Lower-Side Test*

First, assumed data is normally distributed; thus, conducted two-sample parametric T-test. Collectively as a whole, the two-sample *t*-test (two-sided) indicates that the population means of the 6-inch and 12-inch composite samples are collectively the same (i.e., $p=0.7792 > \alpha=0.05$). Excluding the colored-hatch grids that are red and yellow, the rest of the grids contain at least one 6-inch and 12-inch composite samples. Collectively treating those samples as a whole, again the two-sample *t*-test (two-sided) indicates that the population means of the 6-inch and 12-inch composite samples are collectively the same (i.e., $p=0.4677 > \alpha=0.05$). Excluding the colored-hatch grids that are red and yellow, the rest of the grids can be matched and grouped per the criteria listed in the following table.

TABLE II – DATA GRID GROUPING CRITERIA		
Matched Group	**Data Description**	**Alternate Hypothesis**
1	data range overlaps per grid	H_a: $\mu_6 \neq \mu_{12}$ ($\mu_6 > \mu_{12}$)
2	data range overlaps per grid	H_a: $\mu_6 \neq \mu_{12}$ ($\mu_6 < \mu_{12}$)
–1	data range does not overlap per grid	H_a: $\mu_6 > \mu_{12}$
–2	data range does not overlap per grid	H_a: $\mu_6 < \mu_{12}$

The blue colored-hatch grids are indicative of Group 1. Per each grid, the 12-inch composite value lies within the range of the 6-inch composite values, indicating the population means are collectively the same; however, by some off chance, the mean of the 6-inch composite values is greater than the 12-inch composite value. The aqua (bluish-green) colored-hatch grids are indicative of Group 2. Per each grid, the 12-inch composite value lies within the range of the 6-inch composite values, indicating the population means are collectively the same; however, by some off chance, the mean of the 6-inch composite values is less than the 12-inch composite value. Conducting a two sample *t*-test collectively upon the data for both Groups 1 and 2, the two-sample *t*-test (two-sided) indicates that the population means of the 6-inch and 12-inch composite samples are collectively the same (i.e., $p=0.7263 > \alpha=0.05$). Assuming the population mean of the 6-inch composite samples is indeed greater than the population mean of the 12-inch composite samples for Group 1, an one-side (Upper) two sample *t*-test indicates it is not so and they are collectively the same (i.e., $p=0.08115 > \alpha=0.05$). (***Please note p-value is rather close to significance level of 0.05***.) Moreover, assuming the population mean of the 6-inch composite samples is indeed smaller than the population mean of the 12-inch composite samples for Group 2, an one-side (Lower) two sample *t*-test indicates it is not so and they too are collectively the same (i.e., $p=0.3965 > \alpha=0.05$).

The accented dark red colored-hatch grids are indicative of Group (–1). Per each grid, the 12-inch composite value does not lie within the range of the 6-inch composite values, indicating the population means are perhaps not collectively the same; however, by some off chance, the mean of the 6-inch composite values is greater than the 12-inch composite value. Now when all of the data for the grids in Group (–1) are grouped together, the 12-inch composite values still do not lie within the range of the 6-inch composite values, again indicating the population means are perhaps not collectively the same. The accented orange colored-hatch grids are indicative of Group (–2). Per each grid, the 12-inch composite value does not lie within the range of the 6-inch composite values, indicating the population means are perhaps not collectively the same; however, by some off chance, the mean of the 6-inch composite values is less

than the 12-inch composite value. When all of the data for the grids in Group (−2) are grouped together, the 12-inch composite values do lie within the range of the 6-inch composite values, which indicates the population means are perhaps collectively the same. Conducting a two sample *t*-test collectively upon the data for both Groups (−1) and (−2), the two-sample *t*-test (two-sided) indicates that the population means of the 6-inch and 12-inch composite samples are collectively the same (i.e., $p=0.1813 > \alpha=0.05$). Assuming the population mean of the 6-inch composite samples is indeed greater than the population mean of the 12-inch composite samples for Group (−1), an one-side (Upper) two sample *t*-test indicates that assumption is indeed so and they are not collectively the same (i.e., $p=0.01892 < \alpha=0.05$). Moreover, assuming the population mean of the 6-inch composite samples is indeed smaller than the population mean of the 12-inch composite samples for Group (−2), an one-side (Lower) two sample *t*-test indicates it too is indeed so and they are not collectively the same (i.e., $p=0.01288 < \alpha=0.05$).

The above inference tests assumed the data were normally distributed; however, the data could possibly be not normally distributed. Thus, a non-parametric test (Mann-Whitney-Wilcoxon Test) was also conducted on the data. The table below shows the comparative results of inference testing for both parametric (normally distributed) and non-parametric (not normally distributed) tests. Comparatively, the results are almost nearly the same, except when the data is collectively grouped as a whole for Groups (−1) and (−2). The parametric inference indicates the population means are collectively the same and the non-parametric inference does not.

TABLE III – COMPARATIVE INFERENCE RESULTS

Data Tested	Parametric Inference	Non-parametric Inference
all data collectively as a whole	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.7792	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.5443
all data collectively as a whole, excluding grids where 6-inch and 12-inch composite samples are not together (red and yellow colored-hatch grids).	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.4677	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.1662

all data collectively as a whole for Groups 1 and 2 (blue and aqua colored-hatch grids)	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.7263	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.8928
all data collectively as a whole for Group 1 (blue colored-hatch grids)	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.08115	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.6452
all data collectively as a whole for Group 2 (aqua colored-hatch grids)	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.3965	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.2269
all data collectively as a whole for Groups (–1) and (−2) (accented dark red and orange colored-hatch grids)	Accept null hypothesis: H_0: $\mu_6 = \mu_{12}$ p-value = 0.1813	Reject null hypothesis and Accept alternative hypothesis: H_0: $\mu_6 \neq \mu_{12}$ p-value = 0.03574
all data collectively as a whole for Group (–1); (accented dark red colored-hatch grids)	Reject null hypothesis and Accept alternative hypothesis: H_0: $\mu_6 > \mu_{12}$ p-value = 0.01892	Reject null hypothesis and Accept alternative hypothesis: H_0: $\mu_6 > \mu_{12}$ p-value = 0.02222
all data collectively as a whole for Group (−2); (accented orange colored-hatch grids)	Reject null hypothesis and Accept alternative hypothesis: H_0: $\mu_6 < \mu_{12}$ p-value = 0.01288	Reject null hypothesis and Accept alternative hypothesis: H_0: $\mu_6 < \mu_{12}$ p-value = 0.002073

In closing, the colored-hatch grids that are blue and aqua have population means collectively the same, inferring the 6-inch and 12-composite samples are perhaps collected from the same sampling population. Thus, for these grids, either composite sample (6-inch or 12-inch) can be used as a basis for soil ingestion screening. The accented dark red colored-hatch grids have population means

collectively not the same, inferring the 6-inch and 12-composite samples are perhaps collected from different sampling populations. Since the 6-inch composite samples are inferred to have perhaps a larger mean, then using it as a basis for soil ingestion screening is probably appropriate. The accented orange colored-hatch grids have population means collectively not the same too, inferring the 6-inch and 12-composite samples are perhaps collected from different sampling populations. Though, the 6-inch composite samples are usually used as a basis for soil ingestion screening, you may want to infer to the 12-inch composite samples if they are the larger of the two. The one soil sample collected in the yellow colored-hatch grid can be discarded since the sampling location is an asphalt roadway. Finally, the four samples collected from the red colored-hatch grids may rely on professional judgment.