

# **Health Consultation**

Lane Plating Works, Inc. Superfund Site

Dallas, Dallas County, Texas  
EPA FACILITY ID: TXN000605240

April 27, 2026

Final Release

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Under a Cooperative Agreement with  
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Agency for Toxic Substances and Disease Registry (ATSDR)  
Office of Capacity Development and Applied Prevention Science  
Atlanta, Georgia 30333

## Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. To prevent or mitigate exposures, a consultation may suggest specific actions, such as restricting the use of water, replacing water supplies, intensifying environmental sampling, restricting site access, or removing contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure trends in adverse health outcomes, conducting biological indicators of exposure studies to assess exposure, and providing health education for health care providers and community members.

This report concludes the health consultation process for this site, unless additional information is obtained by ATSDR. If the new information, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued, the consultation may resume.

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April 27, 2026

Kenneth Shewmake  
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U.S. EPA  
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Dallas, Texas 75270-2102

Dear Mr. Shewmake:

In response to your request, the Texas Department of State Health Services (DSHS), in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR), evaluated soil sampling results collected from a residential area west of Bonnie View Road and approximately 250 feet to the northwest of the Lane Plating Works Superfund site (the Site) in Dallas County, Texas. The Environmental Protection Agency (EPA) collected soil samples in 2022 and 2023. DSHS evaluated the sample results to determine if past, present, and future exposure to chemicals in soils may harm people's health. DSHS developed this letter health consultation, based on the results of the aforementioned evaluation, at EPA's request and in compliance with cooperative agreement requirements.

EPA collected soil samples in response to community concerns of contamination. Community members were primarily concerned about metals from the Superfund site contaminating their neighborhood. Based on the available information, DSHS determined

- Exposure to metals detected in residential neighborhood soils is not likely to cause harmful noncancer or cancer effects. Some of these metals include arsenic, hexavalent chromium, thallium, and manganese.
- There was not enough information to fully evaluate potential health effects for exposure to lead in soil.
- Per- and polyfluoroalkyl substances (PFAS) are not likely to migrate from the Superfund site to the offsite residential neighborhood through air or surface water runoff.

The remainder of the letter describes how DSHS arrived at these conclusions.

## **Background**

The Site is located in Dallas, Dallas County, Texas. The Site operated as an electroplating facility for about 90 years, until 2015. Operational processes included chromate dips; chromic acid anodizing; and copper, zinc, nickel, and aluminum plating. Lane Plating Works, Inc., also used a lead melting pot to repair anodes. Collectively, these procedures created large amounts of

hazardous wastes that were at times not properly removed, stored, or labeled [EPA 2023b]. Improper handling and storage of waste led to multiple inspections and violations from various regulatory agencies [TCEQ 2017; OSHA 2015]. Operations ceased in 2015.

From 2016 to 2020, EPA conducted a series of investigative and remedial activities to characterize the nature and extent of contamination and to evaluate potential remedial options. In a January 2023 health consultation, DSHS evaluated the results of these activities. DSHS concluded that exposure to metals detected at the Superfund site could cause harm to people trespassing onsite [DSHS 2023]. DSHS also determined that people are not likely to be harmed by exposure to arsenic and hexavalent chromium in offsite surface soil in a nearby residential area west of Bonnie View Road. However, EPA only collected a limited number of samples from the neighborhood, and the results may not accurately represent the entire neighborhood.

In 2022 and 2023, EPA collected additional soil samples from the residential neighborhood (Figure 1) [EPA 2023b]. DSHS evaluated these soil sampling results.

## **Sampling Data**

EPA collected 36 soil samples at depths ranging from 0 to 0.5 feet below ground surface (bgs) (not including field duplicates) at 25 properties across the residential neighborhood. A composite (5-point) soil sample was collected from either the front or backyard at each residential property. Samples were analyzed for 21 metals, including mercury and hexavalent chromium (Table 1). EPA used standard analytical laboratory methods to evaluate soil samples for metals [EA 2023]. DSHS used the higher concentration when duplicate samples were collected in the health evaluation.

On behalf of EPA, an environmental engineering firm independently reviewed the soil data and followed adequate quality assurance and quality control procedures for data collection and reporting. EPA labeled 27 of the 36 samples analyzed for lead as estimated levels with a low bias (“J- flagged”). This means EPA detected lead in these samples but not at a quantifiable level and the estimated level is likely lower than the actual amount. Due to the uncertainty of using estimated measurements, DSHS did not include these results in the evaluation. All other results were of adequate quality and included in the evaluation.

## **Scientific Evaluations**

### **Screening Analysis**

DSHS compared chemicals detected in soil with comparison values (CVs) published by ATSDR or alternative screening levels published by other agencies [ATSDR 2022]. CVs and other screening levels are media-specific (e.g., soil) levels below which no adverse health effects are expected to occur. If a chemical concentration exceeds a CV or other screening level, it does not necessarily mean there is a health hazard. Rather, it suggests the exposure scenario warrants further public health evaluation based on site-specific exposure conditions.

DSHS evaluated 36 composite surface soil (0 to 0.5 feet bgs) samples collected in 2022 and 2023 for metals (Table 1) [EA 2020]. Arsenic and hexavalent chromium were detected above

CVs and were evaluated further (Table 2). Manganese and thallium were also detected in residential soil. These metals were evaluated further because there are no screening levels for manganese and thallium. Although lead could not be fully evaluated because of the high number of estimated lead samples, the best available science indicates there is no safe level of lead in blood. Therefore, DSHS included a discussion of lead in the public health implications section of the letter health consultation.

### Exposure Pathway Analysis

Several factors affect exposure to a chemical. They include the following:

- the source of contamination (where the chemical comes from)
- how the chemical moves through the environment (e.g., surface runoff)
- a point of exposure where people could contact the contaminated media (soil, air, groundwater)
- a route of human exposure (breathing, eating or drinking, or skin contact)
- an identifiable exposed population (people living in the area with contaminated soil)

Contact with a chemical will happen only if there is a completed exposure pathway. All five of these factors must be present for an exposure pathway to be completed. DSHS evaluated relevant exposure pathways to determine if any were completed based on the available data. In the current analysis, DSHS determined incidental ingestion and skin contact with residential soil as a completed exposure pathway (Table 3). Chemicals were detected in 33 residential surface soils (Table 1). These included arsenic, chromium, iron, lead, manganese, mercury, thallium, and hexavalent chromium. People might be exposed to contaminants if they get soil on their skin or swallow small amounts (100-200 milligrams) of soil while participating in outdoor activities such as playing and gardening. Each residence was evaluated as an exposure unit.

DSHS determined that ingestion of groundwater is not a completed exposure pathway. This is because groundwater is not a source of drinking water for communities surrounding the Site, including the residential area being evaluated (Table 3). Residents near the Site get drinking water from the Dallas Public Water Utility, which is supplied from reservoirs and rivers located at least 13 miles from the facility. Additionally, no private water wells were identified within a one-mile radius of the Site. Therefore, the ingestion of groundwater and other uses of groundwater were eliminated as an exposure pathway.

DSHS further evaluated selected contaminants by calculating exposure point concentrations (EPCs) and exposure doses based on site-specific exposure conditions. An EPC is an estimate of the concentration of a contaminant at the point of human exposure. The maximum concentration of the composite sample (5-point) at each residence (front or backyard) was used as the EPC. The EPC was used to derive an exposure dose for the estimated amount of contaminant that gets into a person's body over a specific time. Exposure doses were compared to health guidelines. If health guidelines were exceeded, site-specific exposure doses were compared with levels at which adverse health effects have been observed in animal or human

studies. The health evaluation considers the potential noncancer and cancer health effects to the public, including sensitive groups such as children.

DSHS calculated potential exposures for residents, including adults and children, using a conservative scenario.<sup>1</sup> DSHS considered a scenario where adults and children would potentially be exposed to contaminated soils seven days per week for 52 weeks each year, for 21 years (children) and 33 years (adults). DSHS considered two exposure scenarios: central tendency exposure, or average (CTE) and reasonable maximum exposure, or higher than average (RME). CTE refers to individuals who have average or typical exposure to a contaminant. RME refers to people who are at the high end of the exposure distribution (95<sup>th</sup> percentile) but still within a realistic exposure range.

ATSDR's default values for standard body weight and exposure duration, as per the ATSDR's Public Health Assessment Site Tool (PHAST), were used to calculate the daily exposure doses (Tables 4 and 5). A bioavailability factor of 1 was used for all contaminants evaluated.

## **Exposure Point Concentrations and Exposure Calculations**

### **Noncancer evaluation**

To evaluate possible noncancer health effects, DSHS compared the estimated combined ingestion and dermal exposure dose to an appropriate health guideline, such as ATSDR's minimal risk level (MRL) or EPA's reference dose (RfD). A health guideline is an estimate of daily exposure to a substance over a specified duration that is unlikely to cause harmful, noncancer health effects in humans. If an estimated exposure dose is lower than the health guideline, adverse noncancer health effects are not expected to occur. If an estimated dose is higher, it does not necessarily mean it will harm people's health. In cases where the health guideline was exceeded, DSHS conducted further evaluation to determine if adverse health effects are possible and if the exposure poses a health hazard.

DSHS calculated hazard quotients (HQs) to compare estimated exposure doses to health guidelines. The HQs were calculated by dividing the estimated exposure dose by a health guideline, such as the MRL (Appendix A). If the HQ is less than 1, then adverse noncancer health effects are not likely. If the HQ is greater than 1, DSHS further evaluated the margin of exposure (MOE). The MOE is a measure of how close the estimated dose is to harmful levels. The smaller

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<sup>1</sup> A conservative scenario refers to a public health assessment approach that uses worst case estimates of exposure to ensure potential health risks are not underestimated. The goal is to be protective of public health, especially sensitive populations.

the MOE, the closer the exposure is to effect levels. When the MOE is less than 1, then exposures exceed the lowest effect levels.

### Cancer evaluation

To estimate cancer risk for cancer-causing contaminants, DSHS multiplied the estimated exposure dose by the contaminant's cancer slope factor (CSF). The calculated cancer risk is referred to as an excess lifetime cancer risk because it estimates the proportion of a population that may be affected by a carcinogen over their lifetime (Appendix A). An excess lifetime cancer risk represents the additional risk above the existing background cancer risk. It should be noted that cancer risk estimates are not a measure of the actual cancer cases in a community, but rather a tool used by DSHS for making public health recommendations.

DSHS evaluated the potential noncancer and cancer adverse health effects from current and future exposure to contaminants, including arsenic, hexavalent chromium, manganese, and thallium (Table 3). While each residence was considered as an exposure unit, levels of arsenic and hexavalent chromium in soil above the CV were detected at only one residence. Manganese and thallium were detected in all soil samples (Table 3). Because screening criteria for these metals are not available, an exposure evaluation was conducted.

### Arsenic

Arsenic is a naturally occurring element widely distributed in the earth's crust and found in air, water, and soil. In Texas, naturally occurring arsenic in soil generally range from 1 to 40 mg/kg, with the median background soil concentration for arsenic of 5.9 mg/kg [USGS 2013]. Arsenic can be found in the wood preservative called copper chromated arsenic, which was discontinued for residential usage in 2003. Arsenic can also be found in some pesticides and fertilizers [ATSDR 2007, ATSDR 2016].

### Arsenic noncancer health effects

DSHS used the chronic MRL of 0.0003 mg/day/kg as the health guideline. The MRL is based on a study by Tseng et al. [1977], in which Taiwanese farmers were exposed to high levels of arsenic in well water over a lifetime. Dermal effects such as hyperpigmentation (skin darkening) and hyperkeratosis (localized overgrowth of skin) were reported in farmers exposed to 0.014 mg/day/kg (lowest observed adverse effect level; LOAEL). A no observed adverse health effect level (NOAEL) was reported among farmers exposed to 0.008 mg/day/kg. The MRL was derived by dividing the NOAEL by an uncertainty factor of 3 to account for human variability. [ATSDR 2007, ATSDR 2016]. Other health effects at these exposure levels included an enlarged liver, bronchitis, gastrointestinal effects, and peripheral vascular effects such as cyanosis, gangrene, and the condition known in China as Blackfoot disease [ATSDR 2007]. However, the overall database for dermal effects is considerably stronger than for other end point effects and was used to derive the MRL.

DSHS detected arsenic levels (21.9 mg/kg) above the CV in one composite sample from a residential yard (Table 3). The highest estimated RME doses from combined incidental ingestion and dermal exposure to soil using the detected arsenic concentration were found in the following age groups:

- Birth to younger than age 1 year
- Age 1 year to younger than age 2 years
- Age 2 years to younger than age 6 years
- Age 6 years to younger than age 11 years (Table 6).

These estimated exposure doses are above the MRL of 0.0003 mg/kg/day (HQs greater than 1). However, there are large differences between the estimated doses and the LOAEL for dermal effects (MOEs ranged from 28 to 82). Therefore, harmful noncancer effects are not likely to occur (Table 6). Estimated doses for all other exposure groups were less than the MRL. Based on this comparison, it is unlikely that children and adults living at this residence would experience noncancer health effects from exposure to soil with arsenic contamination.

### Arsenic cancer health effects

The U.S. Department of Health and Human Services and EPA have determined arsenic to be a human carcinogen [ATSDR 2007, ATSDR 2016]. The CSF used to determine cancer risk was 32 (mg/kg-day)<sup>-1</sup>. The CSF represents the risk of developing bladder and lung cancer and was derived by combining the individual CSFs of 17.6 (mg/kg-day)<sup>-1</sup> for bladder cancer and 21.3 (mg/kg-day)<sup>-1</sup> for lung cancer [EPA 2025]. Multiple epidemiological studies suggest that ingestion of arsenic increases the risk of developing skin, bladder, and lung cancer. A growing number of studies also suggest the development of tumors in internal organs such as the liver, kidney, and lungs following long-term arsenic exposure [ATSDR 2007, ATSDR 2016].

DSHS estimated cancer risks for children and adults from exposure to arsenic detected in soil. The total cancer risk was determined to be eight additional cancer cases in children in a population of 10,000 (8E-4) and three additional cancer cases in adults in a population of 10,000 (3E-4) (Table 6). Because arsenic at this property is within background levels for Texas soils (1 to 40 mg/kg), there is no concern for an increased cancer risk.

Cancer risks are based on the arsenic soil concentration detected in one five-point composite sample. Arsenic was not detected above the CV in any of the other 32 soil samples from other properties. DSHS estimated cancer risk considering people may be exposed for decades to the maximum arsenic concentration; therefore, there is uncertainty in the risk estimates if people were exposed for shorter periods.

### Hexavalent Chromium

Hexavalent chromium is rare in nature and generally produced by industrial processes or man-made sources [NTP 2008]. It can be reduced to less toxic forms of chromium through reactions with organic materials [ATSDR 2012a].

### Hexavalent chromium noncancer health effects

DSHS used the chronic MRL for hexavalent chromium of 0.0009 mg/kg/day as the health guideline. Studies show that ingestion of high levels of hexavalent chromium affects the liver, intestines, lymph nodes, and pancreas in mice and rats [NTP 2008]. The most sensitive study showed an increase in lesions in the lining of the small intestines and additional tissue growth (epithelial hyperplasia) in the lesions in female mice. Rats showed lesions, but no tissue growth. The LOAEL that may cause liver (chronic inflammation) and intestinal effects was 0.38 mg/kg/day [NTP 2008]. ATSDR used information from these animal studies and estimated a lower confidence limit of the benchmark dose (BMDL) that is expected to show a response in 10 percent of the animals tested (BMDL10) of 0.09 mg/kg/day. ATSDR calculated the chronic MRL (0.0009 mg/kg/day) using the lowest BMDL10 and an uncertainty factor of 100 (10 for interspecies extrapolation and 10 for human variability) [ATSDR 2012a].

DSHS detected hexavalent chromium above the CV in one composite soil sample collected from a residential yard (Table 2). The highest estimated RME doses from combined incidental ingestion and dermal exposure to hexavalent chromium in soil at this property are 8.2E-5 for children (birth to less than 1 year) and 7.4E-6 mg/kg/day for adults (Table 7). These estimated exposure doses are below the MRL (HQs less than 1). Based on this comparison, it is unlikely that children and adults would experience noncancer health effects from exposure to hexavalent chromium in soil.

### Hexavalent chromium cancer health effects

The effects of chromium exposure on the human body vary according to the exposure route (i.e., inhalation, ingestion, or skin contact) and the chemical form of chromium. For example, inhaling high levels of hexavalent chromium aerosols can damage the kidneys and the nasal and respiratory tract. EPA has classified hexavalent chromium as a known human carcinogen through inhalation [EPA 2005]. Similarly, the National Toxicology Program (NTP) classified hexavalent chromium as a known human carcinogen based on occupational studies where workers exposed by inhalation developed lung cancer [ATSDR 2012a]. However, there have been mixed results in studies of people living in areas with high levels of hexavalent chromium in drinking water. Some human studies have reported an association with several cancer types, while others have not. In laboratory animals, hexavalent chromium compounds have been shown to cause cancer of the stomach, intestinal tract, and lungs [ATSDR 2012a]. Additionally, several in vitro studies have shown hexavalent chromium to be mutagenic and cytotoxic [ATSDR 2012a; NTP 2008; McCarroll 2010]. The NTP reported that ingestion of high levels of sodium dichromate dihydrate, a compound containing hexavalent chromium, was associated with an increase in oral and small intestine tumors in laboratory animals [NTP 2008]. In 2024, EPA estimated the hexavalent chromium oral slope factor for exposure to adults (i.e., without application of an age-dependent adjustment factor) to be 0.16 (per mg/kg-day). EPA based this on the development of tumors in the oral cavity of female rats [EPA 2024c].

DSHS estimated cancer risks for children and adults from exposure to hexavalent chromium detected in one composite soil sample from one residential yard. The total cancer risk was determined to be six additional cancer cases in children in a population of 1,000,000 (6E-6) and five additional adult cancer cases in a population of 10,000,000 (5E-7) (Table 7). These cancer risk estimates are not a concern.

Cancer risks are based on hexavalent chromium soil concentration detected in only one composite sample. Hexavalent chromium was not detected in any of the other 32 composite soil samples from other residential properties in the area. DSHS estimated cancer risk considering people could be exposed for decades to maximum hexavalent chromium concentration; therefore, there is uncertainty in the risk estimates, particularly for people who lived in the neighborhood for shorter periods.

### **Manganese**

Manganese is a naturally occurring element that is found in rock, soil, water, and food. In Texas, the median background soil concentration for manganese is 300 mg/kg [USGS 2013]. In humans and animals, it is an essential nutrient, and absorbing a small amount of manganese from food is important to staying healthy. Manganese helps the body form connective tissue, bones, and blood-clotting factors. It also plays a role in fat and carbohydrate metabolism, calcium absorption, and normal brain and nerve function [FNB 2002; ATSDR 2012b]. Although manganese is an essential nutrient, excess amounts through ingestion or inhalation may cause some adverse health effects.

### **Manganese noncancer health effects**

The nervous system is the primary target for mid- to long-term exposure through ingestion, and young children are particularly vulnerable because their developing nervous system is more sensitive to damage than adults. In an older population (older than age 67 years), drinking water with high concentrations of manganese (1.8–2.3 mg/L) was associated with unspecified neurological symptoms [Kondakis 1989b]. Kawamura et al. [1941] also reported adverse health effects in a Japanese community (25 individuals) that ingested high levels of manganese (29 mg/L) in contaminated well water over a three-month period. Observed signs and symptoms in the community included lethargy, increased muscle tone, tremor, mental disturbances, and even death. Children appeared to be less affected than adults. However, two other studies showed that ingestion of high levels of inorganic manganese resulted in signs of preclinical neurotoxicity in children. These studies show that children who drank water or ate food with high levels of manganese performed less well in school and on the World Health Organization neurobehavioral core test battery [Zhang 1995].

DSHS detected manganese levels above the CV in all 33 composite soil samples collected from residential yards. The average background level of manganese was 788 mg/kg [EA 2020]. The highest concentration (1,210 mg/kg) is about twice the expected manganese concentration in Texas soil in the area near the Superfund site and may represent natural variation.

The highest detected concentration of manganese (1,210 mg/kg) was used to determine the highest estimated RME doses from combined incidental ingestion and dermal exposure to soil. The estimated dose in young children ranged from 0.037 mg/kg/day (birth to age 1 year) to 0.015 mg/kg/day (ages 6 to 11 years) with a time-weighted average (TWA) dose of 0.02 mg/kg/day. The estimated RME dose in adults is 0.003 mg/kg/day (Table 8).

A health guideline for manganese is not available, and the TWA dose estimated from soil ingestion was compared to doses from human studies that cause harmful effects. The TWA dose of 0.02 mg/kg/day in young children is below the range (0.06 to 0.08 mg/kg/day) shown to cause neurological effects in children after about 10 years of exposure [Kondakis 1989b, Vieregge 1995, Wasserman 2006, Wasserman 2011]. Some uncertainty exists in this comparison because the composite soil sample consisted of only five discrete sample locations. The concentration of 1,210 mg/kg in this composite sample may not accurately reflect the actual manganese concentration in this yard. Uncertainty also exists because it is not known if manganese bound to soil would be absorbed into the body to the same extent as manganese in water. The source of manganese exposure in existing human studies was contaminated water [Kondakis 1989b].

Based on this comparison, children and adults are unlikely to experience noncancer health effects from exposure to manganese in soil at this location. Because adverse health effects are not expected with exposure to the highest level of manganese detected in soils at one residential property, noncancer effects are also not expected with exposure to lower levels at the other residential properties.

Manganese is an essential nutrient, and the Food and Nutrition Board of the Institute of Medicine has set adequate intake levels to be

- 1.2 mg/day (0.08 mg/kg/day) for children
- 2.2 mg/day (0.14 mg/kg/day) for adults.

The exposure doses calculated using the maximum level detected in soil are below the adequate intake doses for children and adults established by the Food and Nutrition Board [FNB 1989].

### **Manganese cancer health effects**

EPA has classified manganese as group D, or not classifiable as to human carcinogenicity. This is because the information about carcinogenicity in humans or animals is not available or inadequate [EPA 2011]. Therefore, cancer effects from manganese exposure were not evaluated.

### **Lead**

In nine of the 36 residential soil samples, DSHS detected lead levels below the EPA updated residential soil lead guidance regional screening level of 200 mg/kg [EPA 2024b]. Exposure to these levels is not a health concern as long as no other significant sources of lead exist in the

community. However, lead levels were reported as estimated levels (not quantifiable) in the other 27 residential soil samples. Because of the uncertainty of the estimated measurements, these results were not included in the DSHS's evaluation.

Although lead could not be fully evaluated for the dataset, the best-available science indicates there is no safe level of lead in blood. As a result, ATSDR and the Centers for Disease Control and Prevention (CDC) recommend reducing lead exposure as much as possible. Harmful health effects associated with lead are many, including renal, cardiovascular, hematologic, and developmental effects [ATSDR 2020]. Exposure to lead in young children can cause brain and nervous system damage, slowed growth, and developmental and learning problems [ATSDR 2020].

### Other Sources of Lead Exposure

Lead-based paint can be a significant source of lead exposure in older houses. According to the CDC, children under age 6 years are often exposed to lead by breathing or swallowing dust and paint in homes built before 1978 [ATSDR 2020]. Before 1955, there were no limits on lead in paint. It is estimated that most paint before 1955 contained between 2.5 and 5 percent lead, but some may have contained as much as 40 percent lead [ATSDR 2020]. After 1978, the limit was lowered to less than 0.06 percent lead in paint. According to the 2022 American Community Survey five-year estimates, approximately 6 percent of homes in the residential sector sampled were built before 1950 [USCB ACS 2020]. Plumbing and plumbing fixtures in homes can also contain lead. Other sources of lead include pottery, home remedies, spices, paint on toys, and candy.

### Thallium

Thallium is a naturally occurring metal in the earth's crust. It is used to manufacture electronics, thermometers, optical lenses, and plating processes [ATSDR 2024]. Production of thallium was banned in the United States in 1984; it is now solely imported from other countries. While thallium is highly toxic, it has not been extensively studied. There is a lack of supporting evidence that thallium is a carcinogen. Therefore, DSHS evaluated only the noncancer health effects.

From 2007 to 2010, the United States Geological Survey determined the median background thallium soil level of 7.6 mg/kg in the United States [USGS 2013]. DSHS determined thallium levels detected by EPA in residential soil samples are below the background levels.

### Thallium noncancer effects

ATSDR has not derived MRLs for thallium, nor has EPA published an RfD. However, EPA has proposed candidate oral RfDs of 2.0E-5 mg/kg/day for thallium sulfate and 1.0E-5 mg/kg/day for thallium that can be used for screening purposes [EPA 2012]. The candidate RfD (1.0E-5 mg/kg/day) is based on a NOAEL of 0.04 mg/kg/day for female rats with damage to the hair follicles. The same study identified a LOAEL of 0.2 mg/kg/day [EPA 2012].

The highest DSHS-detected concentration of thallium (0.6 mg/kg) in a composite sample was used to determine the highest estimated RME doses from incidental ingestion exposure to soil. The highest estimated dose in children was 1.2E-6 mg/kg/day (birth to younger than age 1 year), while the estimated dose in adults was 2.6E-7 mg/kg/day (Table 9). These doses are below EPA's provisional RfD of 1.0E-5 mg/kg/day (HQs less than 1) (Table 9).

Based on this comparison, it is unlikely that children and adults would experience noncancer health effects from exposure to thallium in soil at this location. No adverse health effects are expected considering exposure to the highest level of thallium detected in soils at one residential property. So, noncancer effects are also not expected with exposure to lower levels at the other residential properties. Some uncertainty exists in this conclusion because the composite soil sample consisted of only five discrete sample locations. The concentration of 0.6 mg/kg in this composite sample may not accurately reflect the actual thallium concentration in this yard. There is also uncertainty using the provisional RfD because no chronic-duration studies are available and only a small number of intermediate-duration animal studies are available.

#### Thallium cancer health effects

EPA has classified thallium as group D or not classifiable as to human carcinogenicity. This is because the information regarding carcinogenicity in humans or animals is not available or inadequate [ATSDR 2024]. Therefore, cancer effects from thallium exposure were not evaluated.

#### Per- and polyfluoroalkyl substances (PFAS)

Community members have expressed concern about potential exposure to per- and polyfluoroalkyl substances (PFAS), a large group of chemicals with similar molecular structure used in many products. PFAS in the environment can enter the food supply through plants and animals grown, raised, or processed in PFAS-contaminated areas. PFAS also may be associated with chrome plating operations, which is a type of electroplating [FR 2024]. Based on community concern, PFAS migration (by air or surface water runoff, for example) from the Site to the offsite residential neighborhood was qualitatively evaluated in this document.

Based on the available information from EPA's remedial investigations for the Lane Plating Site, the waste flowed from the process areas (where facility operations occurred) and was transported to on-site open areas next to it. This contaminated the soil at the site. EPA analyzed five onsite soil samples for 16 of the most persistent and common PFAS compounds. The highest concentrations of PFAS measured in surface soils in the onsite open areas at the Superfund site are below applicable ATSDR's screening levels [EPA 2020] (Table 10). Therefore, if PFAS did migrate to the neighborhood, the substances would be present at concentrations below ATSDR's screening level and thus not a health concern.

It is possible that chemicals generated during Site activities, including PFAS, migrated by air or surface water runoff from the Site to the offsite residential neighborhood. However, the

neighborhood is uphill from the Site, so surface water runoff from the facility would not impact it (Figure 1). Additionally, the residential area appears to be outside the flood zone (Figure 2) [EPA 2023b].

## Summary of Limitations and Uncertainties

DSHS has identified the following uncertainties and limitations in this evaluation report:

- DSHS estimated health risks from long-term exposure to chemicals detected in soil samples (seven days per week for 52 weeks per year) for 21 years (children) and 33 years (adults).. If actual exposure levels are lower than the health protective assumptions applied, the true risk to the population will be significantly lower.
- A limited number of samples (n=9) could be evaluated for lead. They may not represent lead exposure for those properties not sampled. DSHS could not evaluate soil lead levels in the residential community for properties that were not sampled.
- EPA evaluated a limited number of soil samples (n=5) for PFAS at the Superfund site. Five soil samples may not be enough to determine whether PFAS was a potential chemical of concern at the Site.
- Estimating an exposure dose requires determining how much, how often, and how long a person could have contact with contaminants. Although DSHS' approach was health-protective, each individual's exposure could vary depending on their lifestyle. Because of this uncertainty, DSHS estimated exposure for a typical person and for a person with high contact with soil.

## Conclusions

DSHS has reached the following conclusions based on the assessment of contaminants in soil:

- Incidental swallowing or contact with metals in soil at the residential area is not expected to harm people's health. Exposure doses for adults and children were either less than levels that cause harmful effects or below background levels. Therefore, short- and long-term (more than 1 year) exposures to arsenic, manganese, and hexavalent chromium in soil at the residential area are not expected to cause noncancer or cancer health effects.
- Other metals detected in soil were below comparison levels and are not expected to cause harmful effects. There was not enough information to fully evaluate potential health effects for exposure to lead in soil. This is because most samples had estimated lead measurements that were lower than the quantitation limit and therefore, had higher uncertainty. Lead was detected in soil at levels below the EPA screening level of 100 mg/kg in nine of the 36 residential soil samples. However, lead was not quantifiable and reported as estimated levels in the other 27 residential soil samples.
- Contaminants, including PFAS from the Superfund site, would not be expected to migrate into the neighborhood via soil or surface water runoff. Based on the data

provided by EPA, the highest concentrations of PFAS measured at the Superfund site outside of the onsite process area are below applicable screening levels for surface soils. Therefore, if PFAS did migrate to the neighborhood (by air or surface water runoff), it would likely be present in surface soil at concentrations below screening levels and thus not a health concern. Some uncertainty exists in this conclusion because only a limited number of on-site soil samples (n=5) were collected at the Superfund site.

## Recommendations

- EPA is encouraged to collect additional surface soil samples (less than 3 inches deep) from the neighborhood to evaluate lead.
- As a best preventive practice to reduce exposure to chemicals (including lead) in soils, community members are encouraged to take the following actions:
  - Wash hands and toys often with soap and water. Wash hands before eating, after handling soil, and after playing in dirt.
  - Take off your shoes at the door.
  - Change out of dirt-covered clothes and wash them separately if they contacted lead-contaminated soil or if you were working with lead.
  - Test your home for lead-based paint if it was built before 1978 and paint is deteriorating or chipping.
  - Use wet paper towels to clean up lead dust.
  - Clean around windows, indoor play areas, and floors.
  - Inspect your home for lead, particularly for lead paint, before renovating it.
  - Use a high-efficiency particulate air (HEPA) filter vacuum to minimize lead-paint dust inside home.
- Current DSHS Childhood Lead Poisoning Prevention Program (CLPPP) blood lead screening guidelines include:
  - Blood lead screenings for all children enrolled in Medicaid/TX Health Steps programs at their 12- and 24-month well child exams and then continued screening until age 6 years.

- If a child is not enrolled in Medicaid/TX Health Steps, medical providers may determine if the child is at risk for lead exposure by finding out if they live in a targeted zip code.<sup>2</sup>
- If the child does not reside in a targeted zip code, medical providers may decide if they need to be tested by using the DSHS CLPPP's PB 110 Lead Risk Questionnaire.<sup>3</sup>
- If parents or guardians ask for testing or there is clinical presentation of lead poisoning, medical providers are encouraged to test, preferably using a venous blood sample.
- Women who are pregnant or may become pregnant may also consider having their blood tested for lead if they believe they have come in contact with lead.
- Because the residential sector is across the street from the Superfund site, EPA is encouraged to continue to ensure the fence surrounding the Site remains in place to prevent access and trespassing pending completion of site remediation.
- Because of community concern and the low number of soil samples evaluated for PFAS at the Superfund site, EPA is encouraged to collect additional soil samples at the site for additional PFAS evaluation.

## Public Health Action Plan

- DSHS will evaluate additional data from this site upon request from EPA.
- DSHS will help EPA implement the recommendations in this document.
- DSHS will help EPA develop and deliver risk communication messages to the residential neighborhood.

This concludes the letter health consultation regarding the Lane Plating Works, Inc. Superfund site. Please feel free to contact me at [omar.valdez@dshs.texas.gov](mailto:omar.valdez@dshs.texas.gov) or by phone at 512-776-3714, if you have any questions.

Sincerely,

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<sup>2</sup> DSHS targeted zip codes have one or more associated census tract in which: (a) The percentage of children 1-2 years old with a blood lead level  $\geq 5$   $\mu\text{g}/\text{dL}$  is  $\geq 3\%$  among those tested in 2016 (prevalence), or (b) The percentage of residential structures built before 1950 is  $\geq 27\%$  (housing). To determine if a child resides in a targeted zip code see: [https://www.dshs.texas.gov/sites/default/files/lead/pdf\\_files/child\\_screening\\_2019\\_revised-june-20.pdf](https://www.dshs.texas.gov/sites/default/files/lead/pdf_files/child_screening_2019_revised-june-20.pdf)

<sup>3</sup> <https://www.dshs.texas.gov/sites/default/files/pb110.pdf>

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## Report Preparation

The Texas Department of State Health Services (DSHS), Health Assessment Toxicology program prepared this Letter Health Consultation for the Lane Plating Works, Inc. site, located in Dallas, Dallas County, TX. This publication was made possible by a cooperative agreement [program # CDC-RFA-TS-23-0001] with the federal Agency for Toxic Substances and Disease Registry (ATSDR). DSHS evaluated data of known quality using approved methods, policies, and procedures existing at the date of publication. ATSDR reviewed this document and concurs with its findings based on the information presented by DSHS.

For additional information, please contact us at 1-888-681-0927 or visit our web site at [epitox@dshs.texas.gov](mailto:epitox@dshs.texas.gov).

For more information about ATSDR, contact the Center for Disease Control and Prevention (CDC) Information Center at 1-800-CDC-INFO (1-800-232-4636) or visit the agency's web site at [www.atsdr.cdc.gov](http://www.atsdr.cdc.gov).

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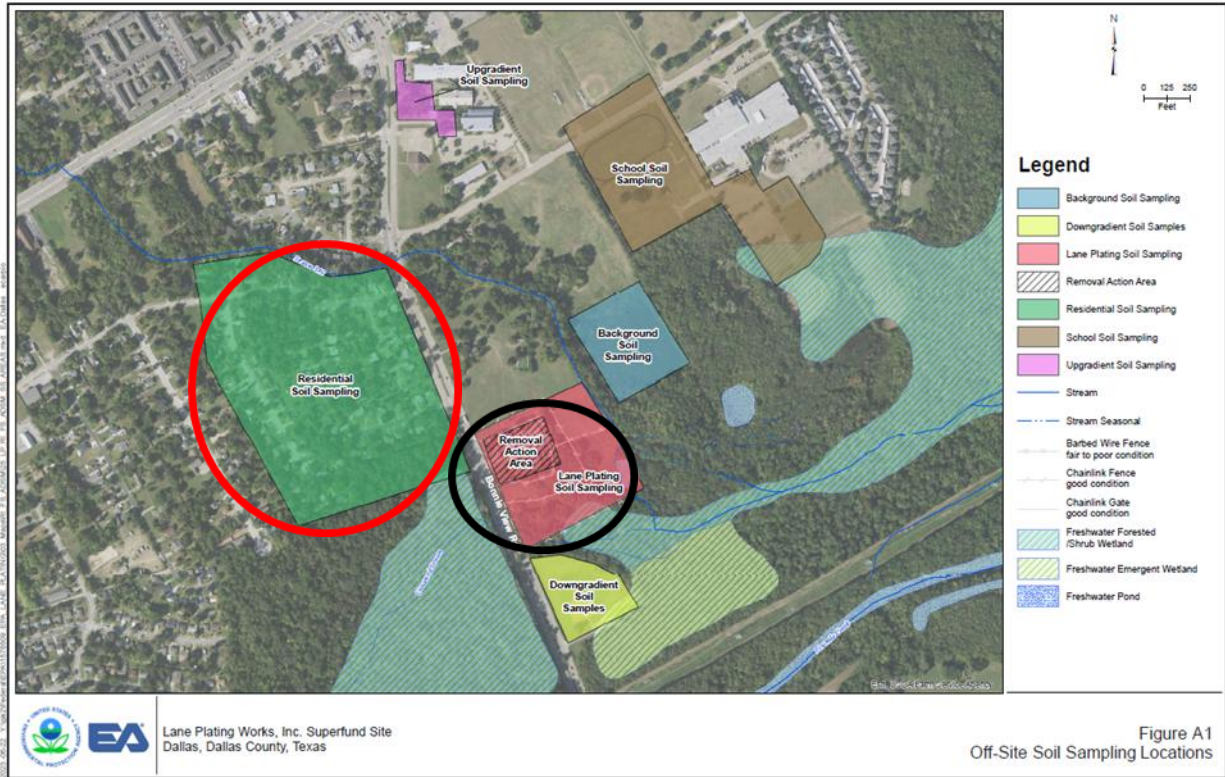
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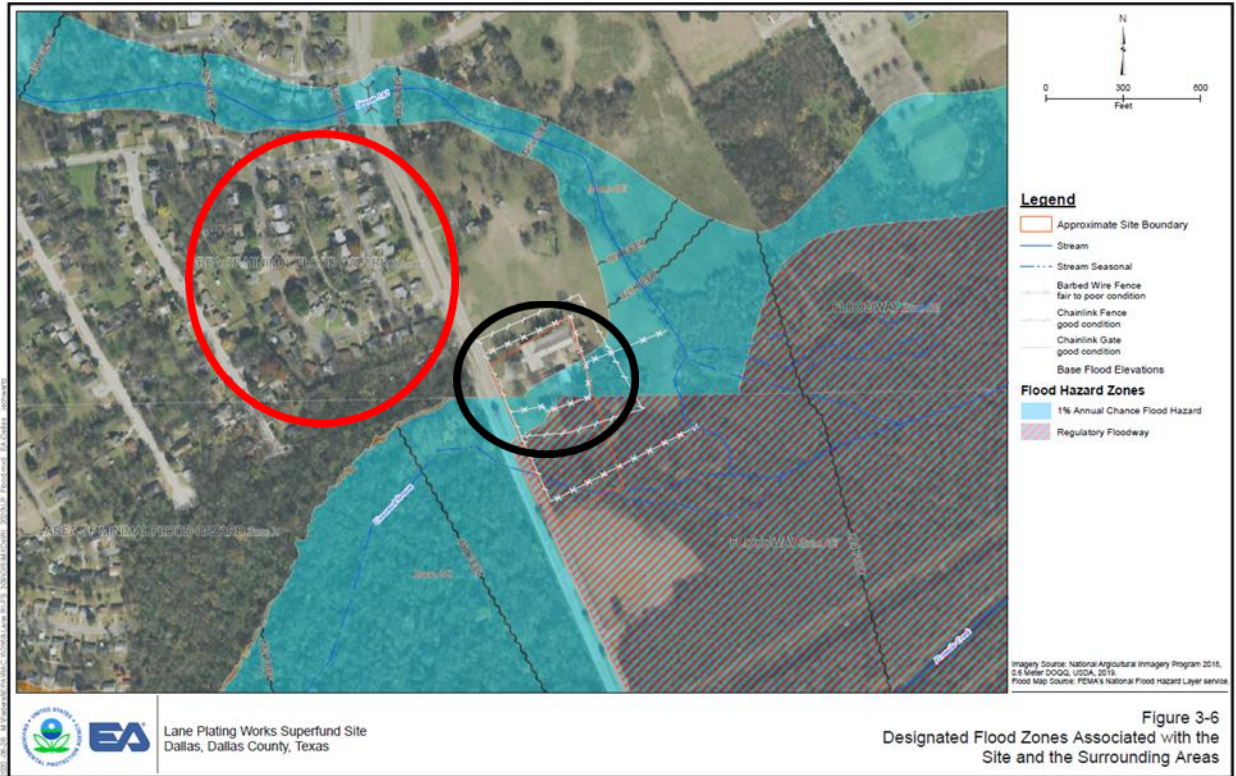
**Attachments**  
**Figures, Tables and Appendix A**

**Figure 1. Location of Lane Plating Superfund site and residential neighborhood**

The red circle indicates the residential neighborhood, and the black circle indicates the Lane Plating Superfund Site [EA 2023].



**Figure 2. Designated flood zones at the Lane Plating Superfund site and surrounding area**  
 The large circle indicates the residential neighborhood, and the smaller circle indicates the Lane Plating Superfund Site [EA 2023].



**Table 1. Summary of soil sampling results and screening criteria, 2022 and 2023**

Contaminant	Maximum Concentration (mg/kg)	Screening Level/ Type (mg/kg)	Exceeded Screening Level (yes/no)
Aluminum	34,000	52,000 (ATSDR chronic EMEG)	No
Antimony	1.4	21 (ATSDR chronic RMEG)	No
Arsenic	21.9	3.1 (ATSDR RMEG)	Yes
Barium	190	10,000 (ATSDR chronic EMEG)	No
Beryllium	1.3	100 (ATSDR chronic RMEG)	No
Cadmium	1.4	5.2 (ATSDR chronic EMEG)	No
Chromium	35	78,000 (ATSDR RMEG)‡	No
Chromium, hexavalent	2.2	0.69 (ATSDR CREG)	Yes
Cobalt	12.1	1,000 (ATSDR intermediate EMEG)	No
Copper	57	110 (ATSDR intermediate PICA)	No
Iron	26,000	55,000 (EPA soil RSL†)	No
Lead*	76.3	80 (CalDTSC)	No
Manganese	1,280	None	NA
Mercury	0.18	0.52 (ATSDR intermediate EMEG)	No
Molybdenum	7.1	260 (ATSDR chronic RMEG)	No
Nickel	50	1,000 (ATSDR chronic EMEG)	No
Selenium	7.1	260 (ATSDR chronic EMEG)	No
Silver	1.4	260 (ATSDR chronic RMEG)	No
Thallium	0.6	None	NA
Vanadium	84	520 (ATSDR intermediate EMEG)	No
Zinc	380	16,000 (ATSDR chronic EMEG)	No

mg/kg=milligrams; ASTDR=Agency for Toxic Substances and Disease Registry; EMEG=environmental media evaluation guides; RMEG=reference dose media evaluation guide; CREG=cancer risk evaluation guides; EPA=Environmental Protection Agency; RSL=Regional Screening Levels; CalDTSC=California Department of Toxic Substances Control; ND=not detected; NA=not applicable.

\*Soil concentrations for 27 of the 36 samples analyzed for lead were labeled as not quantifiable, and only an estimated concentration was reported. Because of the uncertainty of the estimated measurements, these results were not included in the DSHS’s evaluation.

†Noncarcinogenic screening level (target risk=1E-6, hazard quotient=1) Regional Screening Level (RSL) Resident Soil Table [EPA 2024a]

‡ATSDR’s child RMEG for trivalent chromium was used as the CV for total chromium.

**Table 2. Summary of soil screening results for metals evaluated for potential noncancer and cancer adverse health effects\***

Contaminant	Concentration Range (mg/kg)	Screening Level/ Type (mg/kg)	Total Number of Sample Locations	Number of Samples Exceeding Screening Level
Arsenic	3.7 – 21.9 <sup>†</sup>	3.1 / ATSDR Chronic RMEG/ RMEG Child	36	1
Manganese	560 <sup>‡</sup> – 1,210 <sup>‡</sup>	None	36	NC
Lead*	26 – 76.3	80/ CalDTSC	36	0
Hexavalent Chromium	ND – 2.2 <sup>†</sup>	0.69 / ATSDR CREG	36	1
Thallium	0.06 - 0.6 <sup>†</sup>	None	36	NC

mg/kg=milligrams; ASTDR=Agency for Toxic Substances and Disease Registry; EMEG=environmental media evaluation guides; RMEG=reference dose media evaluation guide; CREG=cancer risk evaluation guides; CalDTSC=California Department of Toxic Substances Control; ND=not detected; NC=not calculated.

\*Soil concentrations for 27 of the 36 samples analyzed for lead were labeled as not quantifiable, and only an estimated concentration was reported. Because of the uncertainty of the estimated measurements, these results were not included in the DSHS’s evaluation. Because there is no safe level of lead in blood, lead is included for future evaluation for potential health effects.

<sup>†</sup>Indicates value exceeds screening level or there is no screening level.

**Table 3. Exposure Pathways at Residential Neighborhood near the Lane Plating Superfund site**

Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	Time	Pathway Status
Soil	Soil on residential properties near the Site	Incidental ingestion, dermal contact	Residents (all ages)	Present, Future	Completed
Groundwater	Offsite	Ingestion	No wells within an 1-mile radius of the site have been identified	Past, Present, Future	Eliminated

**Table 4. Site-specific Exposure Factors**

Duration Category	Days per Week	Weeks per Day	Years	Exposure Group Specific EF <sub>noncancer</sub>	Exposure Group Specific* EF <sub>noncancer</sub>
Chronic	7	52.14	33	1	= EF <sub>noncancer</sub> x Exposure Duration for Cancer Exposure Group (years) ÷ 78 years

EF=exposure factor; Note: The dermal absorbed dose equation includes 1 event/day EF parameter.

\*Cancer risk is averaged over a lifetime of exposure (78 years).

**Table 5. Site-specific Exposure Parameters**

Exposure Group	Body Weight (kg)	Exposure Duration (years)	CTE Intake Rate (mg/day)	RME Intake Rate (mg/day)	Adherence Factor to Skin (mg/cm <sup>2</sup> /event)	Combined Skin Surface Area (cm <sup>2</sup> )
Birth to < 1 year	7.8	1	55	150	0.2	1,772
1 year to < 2 years	11.4	1	90	200	0.2	2,229
2 years to < 6 years	17.4	3	60	200	0.2	2,592
6 years to < 11 years	31.8	5	60	200	0.2	3,824
11 years to < 16 years	56.8	5	30	100	0.2	5,454
16 years to < 21 years	71.6	5	30	100	0.2	6,083
Total Child (all age groups)	-	21	-	-	-	-
Adult	80	33	30	100	0.07	6,030

cm<sup>2</sup>=centimeters square skin; CTE=central tendency exposure (typical); kg=kilograms; mg/cm<sup>2</sup>/event=milligram chemical per square centimeter of skin per event; mg/day=milligram soil per day; RME=reasonable maximum exposure; “-“= not determined.

**Table 6. Combined ingestion and dermal exposure doses for chronic exposure to arsenic (21.9 mg/kg) in a residential soil sample along with noncancer HQ, MOE, and cancer risk estimates\*<sup>†</sup>**

Exposure Group	CTE Dose (mg/kg/day)	CTE HQ	CTE MOE	CTE Cancer Risk	RME Dose (mg/kg/day)	RME HQ	RME MOE	RME Cancer Risk
Birth to < 1 year	0.00012	2.1‡	66	-	0.00028	4.7‡	28	-
1 year to < 2 years	0.00013	2.2‡	62	-	0.00026	4.3‡	31	-
2 years to < 6 years	6.5E-05	1.1‡	123	-	0.00017	2.9‡	47	-
6 years to <11 years	4.1E-05	<1	-	-	9.8E-05	1.6‡	82	-
11 years to < 16 years	2.0E-05	<1	-	-	3.6E-05	<1	-	-
16 years to < 21 years	1.7E-05	<1	-	-	3.0E-05	<1	-	-
Total Cancer Risk Child Birth to < 21 years	-	-	-	3E-4‡	-	-	-	8E-4‡
Adult	8.4E-06	<1	-	4E-5‡	2.0E-05	<1	-	3E-4‡

mg/kg/day=milligram per kilogram per day; CTE=central tendency exposure (typical); RME=reasonable maximum exposure (higher); “-“ = not determined; MOE=margin of exposure; HQ=hazard quotient.

\*The calculations in this table were generated using ATSDR’s PHAST v2.5.1.0.

<sup>†</sup>The noncancer hazard quotients for arsenic were calculated using the chronic (greater than 1 year) minimal risk level 0.0003 mg/kg/day, and the cancer risks were calculated using the cancer slope factor of 32 (mg/kg/day)<sup>-1</sup>.

‡ Indicates HQ above 1 and/or cancer risk above 1E-6.

**Table 7. Combined ingestion and dermal exposure doses for chronic exposure to hexavalent chromium (2.2 mg/kg) in a residential soil sample along with noncancer HQ and cancer risk estimates\*†**

Exposure Group	CTE Dose (mg/kg/day)	CTE HQ	CTE Cancer Risk	RME Dose (mg/kg/day)	RME HQ	RME Cancer Risk
Birth to < 1 year	5.5E-05	<1	-	8.2E-05	<1	-
1 year to < 2 years	5.3E-05	<1	-	7.4E-05	<1	-
2 years to < 6 years	3.4E-05	<1	-	5.2E-05	<1	-
6 years to <11 years	2.5E-05	<1	-	3.5E-05	<1	-
11 years to < 16 years	1.8E-05	<1	-	2.1E-05	<1	-
16 years to < 21 years	1.6E-05	<1	-	1.8E-05	<1	-
Total Cancer Risk Child Birth to < 21 years	--	--	4E-6‡	--	--	6E-6‡
Adult	5.5E-06	<1	1E-7	7.4E-6	<1	5E-7

mg/kg/day=milligram per kilogram per day; CTE=central tendency exposure (typical); RME=reasonable maximum exposure (higher); HQ=hazard quotient; "--" = not determined.

\*The calculations in this table were generated using ATSDR's PHAST v2.5.1.0.

†The noncancer hazard quotients for hexavalent chromium were calculated using the chronic (greater than 1 year) minimal risk level of 0.0009 mg/kg/day, and the cancer risks were calculated using the cancer slope factor of 0.16 (mg/kg/day)<sup>-1</sup> and age-dependent adjustment factors.

‡ Indicates HQ above 1 and/or cancer risk above 1E-6.

**Table 8. Combined ingestion and dermal exposure doses for chronic exposure to manganese (1,210 mg/kg) in residential soil samples and noncancer HQ<sup>\*,†</sup>**

Exposure Group	CTE Dose (mg/kg/day)	CTE HQ	RME Dose (mg/kg/day)	RME HQ
Birth to < 1 year	0.022	-	0.037	-
1 year to < 2 years	0.022	-	0.033	-
2 years to < 6 years	0.013	-	0.023	-
6 years to <11 years	0.0096	-	0.015	-
11 years to < 16 years	0.0064	-	0.0079	-
16 years to < 21 years	0.0056	-	0.0068	-
Adult	0.0020	-	0.0031	-

mg/kg/day=milligram per kilogram per day; CTE=central tendency exposure (typical); RME=reasonable maximum exposure (higher); HQ=hazard quotient; “-“ = not determined.

\*The calculations in this table were generated using ATSDR’s PHAST v2.5.1.0.

†A health guideline for manganese is not available, and the dose estimated from soil ingestion was compared to doses from human studies that cause harmful effects. The time-weighted average dose of 0.02 mg/kg/day in young children is below the range (0.06 to 0.08 mg/kg/day) shown to cause neurological effects in children after about 10 years of exposure [Kondakis 1989b, Vieregge 1995, Wasserman 2006, Wasserman 2011].

**Table 9. Combined ingestion and dermal exposure doses for chronic exposure to thallium (0.6 mg/kg) in residential soil samples and noncancer HQ<sup>\*,†</sup>**

Exposure Group	CTE Dose (mg/kg/day)	CTE HQ	RME Dose (mg/kg/day)	RME HQ
Birth to < 1 year	4.5E-06	<1	1.2E-06	<1
1 year to < 2 years	5.0E-06	<1	5.0E-06	<1
2 years to < 6 years	2.2E-06	<1	2.2E-06	<1
6 years to <11 years	1.3E-06	<1	1.3E-06	<1
11 years to < 16 years	4.3E-07	<1	4.3E-07	<1
16 years to < 21 years	3.5E-07	<1	3.5E-07	<1
Adult	2.6E-07	<1	2.6E-07	<1

mg/kg/day=milligram per kilogram per day; CTE=central tendency exposure (typical); RME=reasonable maximum exposure (higher); HQ=hazard quotient; NC=not calculated.

\*The calculations in this table were generated using ATSDR's PHAST v2.5.1.0.

†The HQ was determined using EPA provisional RfD for thallium of 1.0E-05 [EPA 2012].

**Table 10. Per- and Polyfluoroalkyl Substances (PFAS) concentrations detected in onsite surface soils at the Lane Plating Superfund site\* [EA 2019; EA 2020]**

Contaminant of Concern/ Sampling Location	Concentration Range of Detected Values (mg/kg)	Source of Screening Guideline	Screening Guideline (mg/kg)	Number of Detected /Total Number of Samples
Perfluorooctanesulfonic acid (PFOS)/ Process Area	0.00064 - 0.29†	Chronic RMEG Child	0.0052	3 / 3
Perfluorooctanesulfonic acid (PFOS)/ Open Area	0.03 – 0.032	Chronic RMEG Child	0.0052	2 / 2
Perfluorobutanesulfonic acid (PFBS) /Process Area	ND - 0.0015	Chronic RMEG Child	16	1 / 3
Perfluorobutanesulfonic acid (PFBS) /Open Area	0.00032 - 0.00041	Chronic RMEG Child	16	2 / 2

mg/kg=milligram per kilogram; RMEG=reference media evaluation guides; ND=not detected.

\*Onsite samples were collected from within the process area (area where facility operations occurred) and open area (adjacent area to the process area) at the Superfund site.

† Value indicates level above comparison value.

## Appendix A

### Calculation for the evaluation of noncancer effects from ingestion:

$$\text{Ingestion ED} = (C \times IR \times EF \times CF) / BW$$

Where:

Ingestion ED = exposure dose (mg/kg/day)

C = concentration (mg/kg)

IR = intake rate of contaminated soil

EF = exposure factor (unitless)

CF = conversion factor

BW = body weight (kg)

### Calculation of the hazard quotient for exposure dose:

$$HQ = ED / (\text{MRL or RFD})$$

Where:

HQ = hazard quotient (unitless)

ED = exposure dose for ingestion (mg/kg/day)

MRL = ATSDR minimal risk level (mg/kg/day)

RfD = EPA reference dose (mg/kg/day)

### Calculation of margin of exposure:

$$MOE = \text{Effects level} / ED$$

Where:

MOE = margin of exposure

Effects level = a critical study's LOAEL, BMDL or HED.

ED = exposure dose (mg/kg/day)

### Calculation for the evaluation of cancer effects from ingestion:

$$\text{Cancer Risk} = ED \times CSF \times (\text{estimated exposure years} / 78 \text{ years lifetime})$$

Where:

Cancer risk = theoretical excess cancer risk (unitless)

ED = exposure dose (mg/kg/day)

CSF = cancer slope factor (mg/kg/day)<sup>-1</sup>