# Health Consultation

## Tronox LLC,
## Texarkana Facility

**Texarkana, Bowie County, Texas**
**EPA ID TXD057111403**

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**Prepared by**

Texas Department of State Health Services
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

# Table of Contents

## Purpose and Statement of Issues

In response to community concerns, the Texas Commission on Environmental Quality (TCEQ) asked the Texas Department of State Health Services (DSHS) to evaluate the potential public health implications of contaminants found in sediment from three creeks in the vicinity of Tronox LLC, Texarkana Facility (Tronox). (Note: Appendix A lists abbreviations and acronyms used in this report).

## Background

### Site Description and History

The former Tronox wood treatment facility is located at 2513 Buchanan Road in Texarkana, Bowie County, Texas [1]. Since 1905, approximately 100 acres of the 600 acre property have been used by various owners as a production facility for railroad ties and other railroad timber products treated with a creosote-based preservative [2]. The facility was operated by Tronox (formerly known as Kerr-McGee Chemical LLC) from 1969 until 2003. The production facility was decommissioned in 2004 [1]. Although the site is posted with "no trespassing" signs and the majority of the perimeter of the site is fenced, there is access to the site using the highway right-of-way [2].

Days Creek, Howard Creek, and Waggoner Creek are in the vicinity of the Tronox facility (Figure). Days Creek flows in a southerly direction near the eastern property boundary [2]. Waggoner Creek crosses the northeastern portion of the property and empties into Days Creek. Howard Creek transects the middle of the property before entering into Days Creek. The site is underlain by the Wilcox unit which consists of a clay confining layer at 14 feet below ground surface [2].

Two other former wood treatment facilities are located upstream from Tronox (Figure); Koppers Company, Incorporated (Koppers), which operated from 1903 to 1961, and Texarkana Wood Preserving (Texarkana Wood) which operated from 1909 to 1984. Waggoner Creek flows along the southwest edge of Koppers [3]. Texarkana Wood drains southeast to Days Creek [4]. Soil and groundwater at both facilities are contaminated with polycyclic aromatic hydrocarbons (PAHs); both sites are federal National Priority List (NPL) "Superfund" sites. Contaminated soil at Koppers was excavated and replaced with clean soil. Groundwater remediation at Koppers is ongoing [3]. The Environmental Protection Agency (EPA) and TCEQ are working on a remedy for the soil and groundwater contamination at Texarkana Wood [4].

The groundwater corrective action program was voluntarily implemented at Tronox in 1985 [1]. Current remediation and monitoring activities include recovery of free product which settled on top of the Wilcox aquifer [2]. Two recovery trenches (constructed in 1989 [1]) intercept product before it is released into Days and Howard Creeks [2]. Additional remediation included the removal of contaminated soils from a series of six wastewater impoundments (1988-1989 [1]) and the removal of soil from and capping at the former above ground storage tank locations. The

former lagoon and above ground storage tank location areas are potential source areas for the onsite groundwater contamination [2].

### Environmental Sampling

Data evaluated in this health consultation included sediment samples collected by TCEQ from Days Creek, Howard Creek, and Waggoner Creek during two sampling events [5,6]. The first sampling event took place in June 2004 [5]. Four areas along the creeks were chosen as sampling locations, and three composite samples were collected at each sampling location (12 total sediment samples). Sampling locations included Waggoner Creek at Koppers; Days Creek at Texarkana Wood; Days Creek at Tronox; and Days Creek below the Howard Creek confluence (and downstream of all 3 facilities). The creek was surveyed in an attempt to find depositional areas in which fine sediments accumulate and where noticeable sheens were present. The 12 sediment samples were analyzed for semivolatile organic compounds (SVOCs) [5]. Four additional samples were collected in Waggoner Creek at Koppers; Days Creek above Texarkana Wood; Days Creek at Texarkana Wood; and Days Creek at Tronox. These four samples were analyzed for metals [5].

The second sampling event took place in November 2005 [6]. The intent of this sampling event was to characterize the extent of sediment contamination along Days Creek. Samples were collected every 200 feet along Days Creek as it flows through the Tronox property. Additional samples were collected in Waggoner Creek and Howard Creek. A five-point composite sample was collected at each of the 53 transects, and all samples were analyzed for PAHs and arsenic. Sediment samples downgradient from the recovery trenches were also analyzed for pentachlorophenol [6].

For this consultation, DSHS relied on the information provided in the referenced documents and assumed adequate quality assurance/quality control (QA/QC) procedures were followed with regard to data collection, chain-of-custody, laboratory procedures, and data reporting.

## Community Health Concerns

Counsel for concerned community members has provided affidavits alleging the following community concerns:

- Cancer, birth defects, and other diseases may result from exposure to creosote and pentachlorophenol
- The incidence rate of cancer and birth defects is extremely high for the community surrounding the facility
- The prevalence rate of cancer and birth defects for residents surrounding the site are far greater than what would be expected
- Numerous residents surrounding the facility have cancer and birth defects
- Skin burns were caused by creosote after traversing one of the creeks on an all terrain vehicle

# Public Health Implications

## Introduction

Exposure to, or contact with, chemical contaminants drives the Agency for Toxic Substances and Disease Registry (ATSDR) health consultation process. People may be adversely affected by chemicals only if exposure occurs; that is, they must come into contact with the chemicals. Unless the chemicals have direct effects on the skin, eyes, or mucous membranes, people also must absorb the chemicals into their bodies to potentially be affected. The presence of chemical contaminants in the environment does not always result in contact and contact does not always result in the chemical being absorbed into the body. Thus, chemicals have the potential to cause adverse health effects only when people actually come into contact with them through a completed exposure pathway.

## Pathways Analysis

The most common ways people come into contact with chemicals are by inhalation (breathing), ingestion (eating or drinking), or by dermal contact (absorption through skin) with a substance containing the contaminant. Generally, the exposure pathways of greatest concern for chemicals found in sediment are absorption through the gastrointestinal (GI) tract by incidental ingestion or through the skin by direct contact. Whether adverse health effects are possible depends on: 1) the toxicological properties of the chemicals; 2) the manner in which the person contacts the chemical; 3) the concentration of the chemical; 4) how often the exposure occurs; 5) how long the exposure occurs; and 6) how much of the chemical is absorbed into the body during each exposure event.

The presence of the "no trespassing" signs and the fence around the site should function to limit access to the site. The likelihood that either children or adults would regularly come into contact with creek sediments is low due to debris in the creek and the limited recreational use of the creek. There was some evidence of trespassing (the highway right-of-way had been used to access Days Creek with an all terrain vehicle); however, the frequency of accessing the creeks (legally or by trespassing) is not known.

## Determining Contaminants of Concern

To determine the potential health risks associated with the contaminants found in the creek sediment, we compared each contaminant detected with its media specific health-based assessment comparison (HAC) value for non-cancer and cancer endpoints. These values are guidelines that specify levels of chemicals in specific environmental media (soil/sediment, air, and water) that are considered safe for human contact with respect to identified adverse health effects. Non-cancer screening values are based on the ATSDR's minimal risk levels (MRLs)[1] or

[1] An MRL is a contaminant specific exposure dose below those which might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs generally are based on the most sensitive chemical-induced end point considered to be of relevance to humans.

EPA's reference doses (RfDs)[2]. Both of these are based on the assumption that there is an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects. Thus, MRLs and RfDs are estimates of daily exposures to contaminants that are unlikely to cause adverse non-cancer health effects even if exposure occurs for a lifetime. For contaminants that are considered to be known human carcinogens, probable human carcinogens, or possible human carcinogens we calculated cancer risk evaluation guides (CREGs) using EPA's chemical-specific cancer slope factors (CSFs) and an estimated excess lifetime cancer risk of one-in-one million persons exposed for a lifetime. Standard assumptions for body weight (70 kg adult and 16 kg child) and soil/sediment ingestion (100 mg/day for adults and 200 mg/day for a child) were used to calculate both non-cancer and cancer HAC values.

Standard HAC values to assess the potential health risks associated with the presence of carbazole and dibenzofuran in creek sediment samples were not available. Therefore, we derived a HAC value for these compounds. Using the Risk Assessment Information System's CSF [7] for carbazole we calculated a CREG of 35 mg/kg. Based on a provisional chronic oral RfD of 0.002 mg/kg/day derived by the Superfund Health Risk Technical Support Center [8], we calculated a chronic child noncarcinogenic HAC value of 160 mg/kg for dibenzofuran.

The exposure assumptions used to establish these screening levels are conservative with respect to protecting public health; thus, actual exposures are likely to be lower than those used to calculate the screening values. Exceeding a screening value does not mean that a contaminant represents a public health threat; rather, it suggests that the contaminant warrants further consideration. Assessing the public health significance of contaminants that exceed their respective screening levels involves reviewing and integrating relevant toxicological information with plausible exposures. We may estimate the magnitude of the public health significance by comparing the estimated exposures to identified "no observed" and "lowest observed" adverse effects levels (NOAELs and LOAELs) in animals and to known effect levels in humans, when available. We assess the public health significance of contaminants that exceed screening values by reviewing and integrating relevant toxicological information with reasonable maximum exposure scenarios.

Contaminants selected for further consideration are PAHs and arsenic. For both of these contaminants, at least one value exceeded the screening value. All other contaminants were below the detection limit or, if detected, below the screening value. The following sections discuss the potential public health implications of the contaminants selected for further consideration.

### *Polycyclic Aromatic Hydrocarbons (PAHs)*

The contaminants of most concern at the Tronox facility are PAHs in the creek sediment. PAHs are very common in the environment. They may occur naturally and also are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco

[2] An RfD is an estimate (with a level of uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.

and charbroiled meat [9]. There are more than 100 different PAHs (for example, benzo[a]pyrene or BaP) and they are generally found as mixtures, not as single compounds. While PAHs occur naturally, they also can be found in asphalt, crude oil, coal, coal tar pitch, creosote, and roofing tar. In general, PAHs do not dissolve well in water; rather, they tend to stick tightly to solid particles – such as soil/sediment – that can settle at the bottom of lakes, rivers, and creeks [9].

Because PAHs are common in the environment, people are exposed to them everyday. The most common sources of exposure to PAHs are tobacco smoke, food, wood smoke, and ambient air [9]. Exposure to PAHs via inhalation is estimated to range from 0.02 to 3 µg/day. Smoking one pack of unfiltered cigarettes per day increases this estimate by an additional 2 to 5 µg/day; smokers consuming three packs per day increase their exposure by an estimated 6 to 15 µg/day. The intake of carcinogenic PAHs from the average American diet has been estimated to range from 1 to 5 µg/day, mostly from the ingestion of unprocessed grains and cooked meats. This dietary estimate increases to 6 to 9 µg/day for individuals who eat large amounts of meat [9]. Estimated excess lifetime cancer risk estimates associated with common everyday exposures to PAHs are presented in Tables 1 and 2. Absorption through the skin by direct contact, and through the gastrointestinal (GI) tract by incidental ingestion, are generally the exposure pathways of greatest concern when dealing with contaminated sediment.

To assess the potential health risks associated with PAHs in the sediment, we evaluated the toxicity of the contaminants with respect to potential exposures. We considered the incidental ingestion of sediment as the primary pathway of exposure. To assess this pathway, PAHs were converted to BaP toxic equivalents (TEQ) using established toxicity equivalency factors (TEF, Table 3). Sediment data sampling results are presented in Table 4. Using our standard exposure scenario (16 kg child ingesting 200 mg of sediment per day) and the maximum BaP TEQ from each of the two sediment sampling events (21.26 mg/kg for the 2004 sampling event and 14.28 mg/kg for the 2005 sampling event), the estimated exposure dose of PAHs was 0.0003 mg/kg/day and 0.0002 mg/kg/day, respectively (Table 5).

While there is little evidence to indicate a relationship between ingestion of PAHs and adverse health effects in humans, animal studies have shown that ingestion of PAHs causes gastrointestinal (digestive system), hepatic (liver), reproductive, and developmental effects. The lowest doses associated with these effects have ranged from 40 mg/kg/day to 700 mg/kg/day [9], approximately five to six orders of magnitude higher than estimated exposure doses associated with the ingestion of PAHs in sediment at this site.

Although no MRL or RfD has been derived for PAHs, there are several LOAELs and NOAELs available [9]. An intermediate NOAEL of 1.3 mg/kg/day was observed for mice exposed to BaP. The LOAEL (2.6 mg/kg/day) is based upon the appearance of gastric tumors. There currently are no studies available that evaluated chronic exposures. Systemic effects occurred at much higher exposure doses in acute and intermediate duration exposures [9].

Several of the PAHs also have been shown to cause tumors in laboratory animals when they breathed these substances in the air, when they ate them, or when they had prolonged skin contact with them [9]. Perhaps the most toxicologically significant PAH is BaP, which, along

with several other PAHs, has been classified by the EPA as a "probable human carcinogen". This classification is based on animal data where repeated BaP administration in numerous strains of at least four species of rodents and several primates has been associated with increased incidence of total tumors and of tumors at the site of exposure [9]. Human data specifically linking BaP, or any of the other PAHs to a carcinogenic effect are lacking. Although lung cancer has been found in humans who had received exposure to various mixtures of PAHs known to contain BaP – including cigarette smoke, roofing tar, and coke oven emissions – it is not possible to conclude from this information that BaP or any other of the PAHs is the responsible agent [9].

Using EPA's cancer slope factor for BaP, based on an 30 year exposure scenario (a 70 kg adult ingesting 100 mg of sediment per day) and the maximum BaP TEQ, we estimated the excess lifetime risk of developing cancer from the incidental ingestion of sediment to be $9 \times 10^{-5}$ and $6 \times 10^{-5}$ for each of the two sampling events, respectively (Table 6). Qualitatively, we would interpret these as a no apparent to a low increased lifetime risk for developing cancer.

The risk for cancer due to exposure to BaP over a lifetime was calculated by evaluating cancer risk over life stages and summing these risks. Estimated exposure doses were calculated for preschool children, elementary school children, teenagers, and adults using standard assumptions for body weight (16 kg preschool child, 30 kg elementary school child, 50 kg teenager, and 70 kg adult), sediment intake rate (200 mg/day preschool child, 150 mg/day elementary school child, and 100 mg/day teenager and adult), and the maximum BaP concentration. The lifetime risk of developing cancer was estimated to be $4 \times 10^{-4}$. Qualitatively, we would interpret this as a low increased lifetime risk for developing cancer.

Both of these estimated lifetime risks of developing cancer are based upon the ingestion of sediment from the creeks. The plausibility of the daily ingestion of creek sediment, especially by young children, is remote, thus, these cancer risks are likely to be overestimated.

Another pathway of concern with PAHs in sediment is dermal contact with creek sediments. It is difficult to quantitatively evaluate dermal contact exposure scenarios involving recreational use of all terrain vehicles. The amount and type of protective clothing (and percentage of exposed skin) varies widely. Multiple riders on the same vehicle and multiple vehicles in the same area add additional uncertainty. The frequency and duration of exposure also are difficult to determine; and the absorption of PAHs into the body varies with length of contact with the skin and how tightly the compounds are bound to the sediment. Due to the many unknowns involved with a quantitative risk calculation, we were limited to a qualitative determination of the potential risks to human health.

Studies have shown that creosote products and PAHs are absorbed into the body following dermal contact with such products [9]. Dermal absorption of PAHs appears to be rapid, but the extent of absorption varies among the different compounds and the vehicle of administration. In monkeys, it was found that 51% of BaP in acetone was absorbed into the skin while only 13% of BaP in soil was absorbed [9]. Another study using rats had similar results in that absorption after exposure to soil containing BaP was 4-5 times less than when BaP was applied to the skin

directly. These studies show that soil (or sediment) binding of PAHs may slow absorption of these substances into the body with dermal exposure [9]. Elimination of PAHs also occurs rapidly in the urine and feces.

It is generally assumed that unless application of PAHs is frequent and at high doses, dermal toxicity is minimal [10]. Mixtures of PAHs are used to treat some skin disorders in humans [9]. Dermal toxicity including irritation and phototoxicity may be possible under some dermal exposures to coal tar shampoos and ointments used to treat skin disorders. Typically, in mice, no overt signs of toxicity are observed with chronic dermal exposure to PAHs [11]. In humans, regressive verrucae (warts) were reported after 4 months of exposure to BaP, and dermal effects following exposure to BaP have been seen in patients with preexisting skin conditions; however, when a similar solution was applied to patients with squamous cell cancer, investigators observed a general improvement and/or retardation of the lesion [9]. Skin tumors have been noted in mice treated with BaP after 18 weeks of treatment [9]. BaP has been shown to elicit an immune response in animals. Mice developed allergic contact hypersensitivity following acute exposure to BaP [9].

Dermal penetration of PAHs following contact with contaminated sediments in the creeks near Tronox is likely to be minimal in terms of frequency, duration, and amount; thereby reducing the potential significance to human health. Development of skin tumors in animals and humans due to PAHs is associated with frequent and prolonged skin contact with these contaminants, which are exposure scenarios that are unlikely to occur in a recreational setting. Additionally, washing the skin (soap and water) after 24 hours of exposure to BaP in soil has been shown to remove 91% of BaP from the skin surface [12].

General recommendations to reduce the absorption of PAHs following acute dermal exposure include removing the individual from the source of exposure, removing contaminated clothing, and decontaminating areas of the body. Skin should be washed with soap and water, and eyes should be flushed with water or saline [9].

In summary, it is not likely that exposure to PAHs in the creek sediment would result in adverse health effects. Estimated exposure doses for incidental sediment ingestion are approximately four orders of magnitude lower than exposure doses that resulted in adverse health effects, and there is a no apparent to a low increased lifetime risk for developing cancer due to exposure to PAHs in the sediment. Exposure dose and cancer risk calculations are conservative estimations because they are based upon daily exposure to sediment, a scenario that is unlikely to occur. Dermal contact with creek sediment is another exposure pathway of concern; however, contact with contaminated sediment is not likely to be frequent enough or long enough to result in adverse health effects. Additionally, washing the skin following acute exposure reduces the absorption of PAHs.

### *Arsenic*

Arsenic is a naturally occurring element in the earth's crust and usually is found in combination with other elements [13]. In the environment, arsenic most often is found as inorganic arsenic,

which is formed when arsenic combines with other elements such as oxygen, sulfur, and chlorine. Organic forms of arsenic, which result when arsenic combines with carbon and hydrogen, are generally considered less toxic than the inorganic forms. Children and adults normally take in small amounts of arsenic through inhalation of air and ingestion of food and water, with food being the largest source of exposure to arsenic. Fish and seafood contain the highest concentrations of arsenic; however, most of this is in the less toxic organic form of arsenic [13]. Background concentrations of arsenic in soil range from 0.1 to 97 mg/kg with an average value of about 7.0 mg/kg [14]. The mean background arsenic concentration for Texas soils is 5.9 mg/kg based on TCEQ Risk Reduction Rules [15].

To assess the potential health risks associated with arsenic in the sediment, we compared the concentrations to HAC values for both non-cancer and cancer endpoints for the more toxic form of the compound. The non-cancer HAC values for arsenic in soil (20 mg/kg for children and 200 mg/kg for adults) are based on ATSDR's chronic oral MRL for arsenic of 0.0003 mg/kg/day. The MRL was derived by dividing the identified NOAEL of 0.0008 mg/kg/day, obtained from human epidemiologic studies, by an uncertainty factor of three to account for the lack of data on reproductive toxicity and to account for some uncertainty as to whether the NOAEL accounts for all sensitive individuals [13]. The LOAEL associated with these epidemiologic studies was 0.014 mg/kg/day, where exposure to arsenic above this level resulted in hyperpigmentation of the skin, keratosis (patches of hardened skin), and possible vascular complications [13].

Arsenic was detected in all sediment samples; however, only 2 sediment samples (collected in November 2005) exceeded the non-cancer HAC value for children (20 mg/kg). It also is important to note that only 8 samples exceeded the average typical background concentrations for this area as identified by the TCEQ [12]. Using our standard exposure scenario (16 kg child ingesting 200 mg of sediment per day) and the maximum arsenic concentration from the two sediment sampling events (105 mg/kg, Table 4), the estimated exposure dose of arsenic was 0.001 mg/kg/day. Although this estimated exposure dose exceeds the chronic oral MRL for arsenic, the maximum arsenic concentration is much higher than all other sediment samples from the creeks; it is unlikely that a child would be exposed to the maximum level on a daily basis. The 95$^{th}$ percentile arsenic concentration for the November 2005 sampling event (the sampling event with the higher values) was 13.78 mg/kg, which would result in an estimated exposure dose of 0.0002 mg/kg/day, a dose below the chronic oral MRL for arsenic (Table 5).

Based on sufficient human data, inorganic arsenic is considered to be a known human carcinogen [13]. The most characteristic effect of long-term exposure to inorganic arsenic is a pattern of skin changes including darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. A small number of these corns may ultimately develop into skin cancer. Liver, bladder, kidney, and lung cancer also have been associated with exposure to arsenic [13]. The oral slope factor for arsenic is based on an increased incidence of non-malignant skin cancers observed in people who consumed water high in inorganic arsenic [13]. The carcinogenic HAC value (CREG) for arsenic of 0.5 mg/kg is based on EPA's CSF for skin cancer and an estimated excess lifetime cancer risk of one cancer in 1 million (1 x $10^{-6}$) people exposed for 70 years. All but two of the sediment samples had arsenic concentrations above the CREG (0.5 mg/kg).

Using EPA's cancer slope factor for arsenic, a 30 year exposure scenario (a 70 kg adult ingesting 100 mg of sediment per day), and the maximum arsenic concentrations, we estimated a theoretical excess lifetime risk for developing cancer from the incidental ingestion of sediment to be $7 \times 10^{-6}$ and $9 \times 10^{-5}$. Qualitatively, we would interpret this as a low to no apparent increased lifetime risk for developing cancer. Using the 95$^{th}$ percentile arsenic concentration, the risk of developing cancer was $1 \times 10^{-5}$ (no apparent increased risk for developing cancer).

The risk for cancer due to exposure to arsenic over a lifetime was calculated by evaluating cancer risk over life stages and summing these risks. Estimated exposure doses were calculated for preschool children, elementary school children, teenagers, and adults using standard assumptions for body weight (16 kg preschool child, 30 kg elementary school child, 50 kg teenager, and 70 kg adult), sediment intake rate (200 mg/day preschool child, 150 mg/day elementary school child, and 100 mg/day teenager and adult), and the maximum arsenic concentration. The lifetime risk of developing cancer was estimated to be $4 \times 10^{-4}$. Qualitatively, we would interpret this as a low increased lifetime risk for developing cancer. Using the 95$^{th}$ percentile arsenic concentration, the lifetime risk for developing cancer was $5 \times 10^{-5}$. Qualitatively, we would interpret this as no apparent increased lifetime risk for developing cancer.

These estimated lifetime risks of developing cancer are based upon the ingestion of sediment from the creeks. The plausibility of the daily ingestion of creek sediment is remote, thus these cancer risks are likely to be overestimated.

In summary, it is not likely that exposure to arsenic in the creek sediment would result in adverse health effects. Only two sediment samples exceeded the screening value for arsenic, and using a reasonable approximation of the arsenic concentration in sediment (the 95$^{th}$ percentile arsenic concentration for the November 2005 sampling event), the estimated exposure dose was 0.0002 mg/kg/day, a dose below the chronic oral MRL for arsenic. Additionally, there was a no apparent increased lifetime risk for developing cancer due to exposure to arsenic in the sediment. Exposure dose and cancer risk calculations are conservative estimations because they are based upon daily exposure to sediment, a scenario that is unlikely to occur.

## Response to Community Health Concerns

### *Cancer*

The Texas Cancer Registry has previously completed three cancer cluster investigations into the incidence and mortality of cancers in the Texarkana area [16-18]. In 1997, mortality data for cancers of the colon, liver, bone, and breast for the zip code 75501 was investigated [16]. Data from the years 1991 through 1995 was evaluated, and no significant excess of cancer mortality was observed either among males or females [16]. In 2000, incidence data (1995-1997) and mortality data (1990-1998) for cancers of the lung and bronchus, melanoma, prostate, kidney and renal pelvis, and brain and other nervous system in Bowie County were investigated [17]. Lung and bronchus cancer mortality was statistically significantly elevated in males (1.5 times greater than what would be expected to occur based on state rates) but not in females. Even though the lung and bronchus cancer mortality in males was statistically significantly elevated, a specific

cause or hazard was not identified or associated with the mortality excess. Cancer tends to be caused by various risk factors, and in the case of lung cancer, the most common risk factor is smoking. In 2001, incidence (1995-1997) and mortality (1990-1999) data for cancers of the colon, liver, pancreas, lung, breast, uterus, and brain and other central nervous system for the zip code 75501 were reviewed. No significant differences from expected cancer rates were detected [18]. The Texas Cancer Registry also completed an additional cancer cluster investigation using the most current incidence data available (1995-2003) for cancers of the prostate, breast, lung and bronchus, colon and rectum, corpus and uterus, pancreas, kidney and renal pelvis, brain and other nervous system, liver and intrahepatic bile duct, and bones and joints for the zip code 75501 and Bowie County [19]. Lung and colon and rectum cancer incidence in males was found to be statistically significantly elevated for zip code 75501. In Bowie county overall, lung cancer in males was statistically significantly elevated while cancers of the breast and corpus and uterus were statistically significantly less than expected among Bowie County females [19]. The reasons why these cancers are elevated are not known, and may not ever be known. Colorectal cancer is generally not considered to be environmentally related, rather, risk factors including aging, family history of colorectal cancer, personal history of intestinal polyps and chronic inflammatory bowel disease, diet, and physical inactivity are generally involved. Additionally, smoking accounts for 87% of all lung cancer, and it was not possible to separate out the possible effects of smoking in this population [19]. Based upon these cancer cluster investigations, there does not appear to be an increased cancer incidence in the Texarkana area that would be related to an environmental exposure.

### *Birth Defects*

The rates of birth defects among births to Bowie County residents has also been considered. Birth defects registry data for 49 types of birth defects during the years 1999 to 2003 (the most recent birth defects data available) indicated that, in comparison to statewide data for the same time period, none of the birth defects appear to be elevated in Bowie County, and the rates of birth defects in Bowie County may actually be lower than the state overall [20]. A two-year site-specific surveillance project involving community members living on or near Koppers was conducted to determine if these community members had different prevalence of diseases than a comparison community not situated near the site. Results of this investigation indicate that there was not a higher rate of birth defects; however, the prevalence of reported difficulties becoming pregnant was higher for women living on or near Koppers than comparison neighborhood women [21]. Based upon these birth defect investigations, there does not appear to be an increase in birth defects in the Texarkana area that would be related to an environmental exposure.

### *Other Concerns*

Residents living near the Tronox site have reported skin burns caused by creosote after traversing one of the creeks on an all terrain vehicle. In the Koppers surveillance project, it was determined that residents living on or near the Koppers site reported a higher prevalence of skin rashes than comparison neighborhood residents [21]. Among the residents living in the Koppers area, increased risk for rashes was associated with digging in their yards or having contact with soil in the neighborhood and wading or having contact with Waggoner Creek [21]. It is important to

note that in the investigation at Koppers, contamination was present in residential surface soils, with BaP TEQ levels ranging from 3.41 mg/kg to 754 mg/kg [22]. Unlike the Koppers site, there are no residences on-site, thus no residential surface soil data is available. Only creek sediment data is available, and the maximum BaP TEQ in creek sediment was 21.26 mg/kg. Although there is PAH contamination present in the creek sediment, contact with contaminated sediment is likely to be much less frequent than the contact the Koppers' residents had to the soil in their yards. Based upon the available information, it does not appear that PAH concentrations are high enough and contact with contaminated sediment is not frequent enough for adverse health effects to occur.

## Child Health Considerations

In communities faced with air, water, or food contamination, children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. A child's lower body weight and higher intake rate result in a greater dose of hazardous substance per unit of body weight. Sufficient exposure levels during critical growth stages can sustain permanent damage to the developing body systems of children. Children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

We evaluated whether children living in the vicinity of the Tronox would be likely to be exposed to site contaminants at levels of health concern. Even though access to areas known to be contaminated is limited we evaluated the potential risk to children by using conservative exposure scenarios – scenarios where the exposures are likely to be much higher than those that children might truly experience. We also considered that the creek, debris, and other materials hidden by tall vegetation could pose a physical hazard to small children if they were to trespass on the site; however, the likelihood of this occurring seems low.

## Conclusions

1. Access to known contaminated areas is limited, thereby reducing the likelihood for exposure. Regardless, in the interest of protecting public health we assessed potential risks to the public by assuming that people – both children and adults – would be exposed to site contaminants on a regular basis. Additionally, in our assessments, rather than use the average concentration for each of the contaminants of concern we used the maximum concentration for all contaminants as well as the 95$^{th}$ percentile concentration for arsenic. Based on available information we would not expect the incidental ingestion of contaminants at levels found in the creek to result in adverse health effects.
2. Due to limited access to contaminated areas in the creeks and limited dermal absorption of PAHs adhered to sediment; it is not likely that residents will have dermal exposure to site contaminants at concentrations high enough or at frequencies often enough to result in adverse health effects.
3. We have determined that the contaminants in sediment found in Days Creek, Howard Creek, and Waggoner Creek pose no apparent public health hazard.

## Recommendations

1. Provide education on ways that residents can reduce their exposure to elevated levels of contaminants.
2. Groundwater remediation activities should continue at Tronox.
3. The highway right-of-way should be fenced to reduce site trespassing and exposures.

# Public Health Action Plan

## Actions Completed

1. The DSHS evaluated contaminant data from sediment samples collected from Days Creek, Howard Creek, and Waggoner Creek during two sampling events (June 2004 and November 2005).
2. DSHS team members toured the Tronox facility and surrounding areas in May 2006.

## Actions Planned

1. The DSHS will provide a fact sheet about ways residents can reduce their exposure to elevated levels of contaminants to counsel for the concerned citizens. The fact sheet will be made available for community members.
2. A copy of this document will be provided to counsel for the concerned citizens and will be made available for community members.
3. The DSHS will review additional environmental data as it becomes available.
4. The DSHS will continue to work with environmental agencies to address community concerns by providing educational materials to concerned citizens.

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## Certification

This public health consultation on Tronox LLC in Texarkana, Bowie County, Texas was prepared by the Texas Department of State Health Services under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methods and procedures existing when the time the public health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

Technical Project Officer, CAT, CAPEB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with its findings.

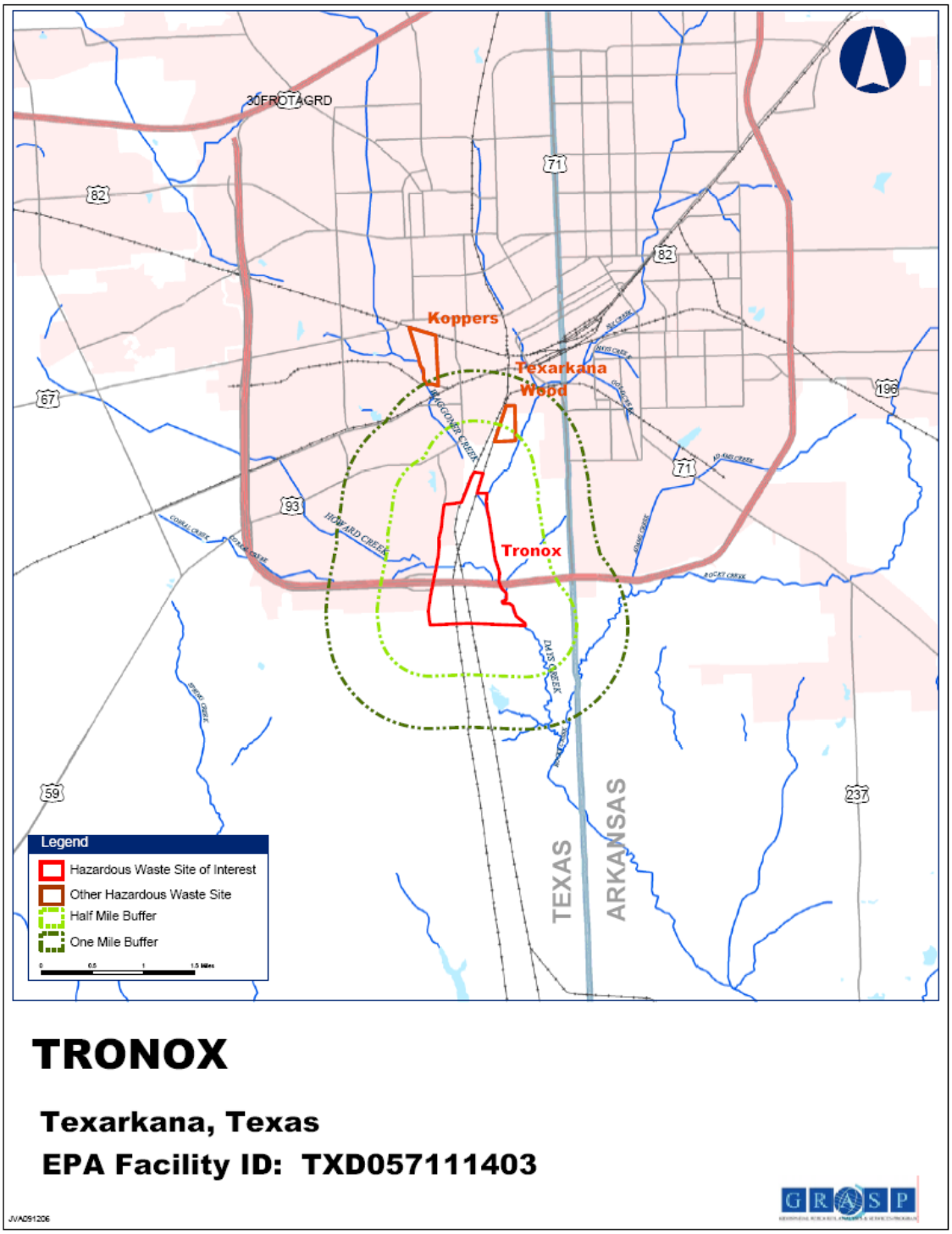
Team Lead, CAT, CAPEB, DHAC, ATSDR

## Appendix A: Acronyms and Abbreviations

| | |
|---|---|
| ATSDR | Agency for Toxic Substances and Disease Registry |
| BaP | benzo[a]pyrene |
| CREG | Cancer Risk Evaluation Guide |
| CSF | Cancer Slope Factor |
| DSHS | Texas Department of State Health Services |
| EMEG | Environmental Media Evaluation Guide |
| EPA | Environmental Protection Agency |
| GI | gastrointestinal |
| HAC | Health-Based Assessment Comparison |
| kg | kilogram |
| L | liter |
| LOAEL | Lowest Observable Adverse Effect Level |
| mg | milligram |
| mg/day | milligram per day |
| mg/kg | milligram per kilogram |
| mg/kg/day | milligram per kilogram per day |
| MRL | Minimal Risk Level |
| ng | nanogram |
| NOAEL | No Observable Adverse Effect Level |
| NPL | National Priority List |
| PAH | Polycyclic Aromatic Hydrocarbon |
| PHAGM | Public Health Assessment Guidance Manual |
| QA/QC | Quality Assurance/Quality Control |
| RfD | Reference Dose |
| SVOC | Semivolatile Organic Compound |
| TAC | Texas Administrative Code |
| TCEQ | Texas Commission on Environmental Quality |
| TEF | Toxic Equivalency Factor |
| TEQ | Toxic Equivalent |
| TRRP | Texas Risk Reduction Program |
| µg | microgram |
| µg/day | microgram per day |

## Appendix B: Figure

Map of Texarkana, indicting locations of Tronox, Koppers, and Texarkana Wood, as well as Days Creek, Howard Creek, and Waggoner Creek.



## Appendix C: Tables

Table1. Estimated lifetime cancer risk from ingestion of food containing average BaP levels seen in the American diet [23].

| Parameter Description | | Lower Value | Median Value | Upper Value |
|---|---|---|---|---|
| Total quantity of food ingested per day (kg food/day) | | 1.00 | 1.00 | 1.00 |
| Average concentration of BaP TEQ in food (µg BaP/kg food) | | 0.192 | 0.600 | 1.920 |
| Quantity BaP TEQ ingested per day (µg BaP/day) | | 0.19 | 0.60 | 1.92 |
| Percent of inhaled BaP TEQ absorbed by body | | 100% | 100% | 100% |
| Quantity BaP TEQ absorbed per day (µg BaP/day) | | 0.192 | 0.600 | 1.920 |
| Conversion factor: µg BaP to mg BaP | | 0.001 | 0.001 | 0.001 |
| Quantity BaP TEQ ingested per day (mg BaP/day) | | 1.920E-04 | 6.000E-04 | 1.920E-03 |
| Slope factor for BaP (per mg BaP/kg body weight/day) | | 7.3 | 7.3 | 7.3 |
| Body Weight (kg) | | 70.0 | 70.0 | 70.0 |
| Cancer risk from daily exposure for 70 years | | 2.002E-05 | 6.257E-05 | 2.002E-04 |
| Number of days per week exposed | | 7 | 7 | 7 |
| Number of weeks per year exposed | | 52 | 52 | 52 |
| Number of years exposed | | 70 | 70 | 70 |
| Cancer risk from limited exposure specified above: | | 2.002E-05 | 6.257E-05 | 2.002E-04 |
| Odds of getting cancer from above exposure: | 1 in | 49,943 | 15,982 | 4,994 |

Table 2. Estimated lifetime cancer risk from drinking water containing average BaP levels seen in public water supplies [23].

| Parameter Description | | Lower Value | Median Value | Upper Value |
|---|---|---|---|---|
| Total quantity of water ingested per day (L water/day) | | 2.00 | 2.00 | 2.00 |
| Average concentration of BaP TEQ in water (ng BaP/L water) | | 0.405 | 1.620 | 6.480 |
| Quantity BaP TEQ ingested per day (ng BaP/day) | | 0.810 | 3.240 | 12.960 |
| Conversion factor: ng BaP to µg BaP | | 0.001 | 0.001 | 0.001 |
| Quantity BaP TEQ ingested per day (µg BaP/day) | | 0.00081 | 0.00324 | 0.01296 |
| Percent of ingested BaP TEQ absorbed by body | | 100% | 100% | 100% |
| Quantity BaP TEQ absorbed per day (µg BaP/day) | | 0.00081 | 0.00324 | 0.01296 |
| Conversion factor: µg BaP to mg BaP | | 0.001 | 0.001 | 0.001 |
| Quantity BaP TEQ absorbed per day (mg BaP/day) | | 8.100E-07 | 3.240E-06 | 1.296E-05 |
| Slope factor for BaP (per mg BaP/kg body weight/day) | | 7.3 | 7.3 | 7.3 |
| Body Weight (kg) | | 70.0 | 70.0 | 70.0 |
| Cancer risk from daily exposure for 70 years | | 8.447E-08 | 3.379E-07 | 1.352E-06 |
| Number of days per week exposed | | 7 | 7 | 7 |
| Number of weeks per year exposed | | 52 | 52 | 52 |
| Number of years exposed | | 70 | 70 | 70 |
| Cancer risk from limited exposure specified above: | | 8.447E-08 | 3.379E-07 | 1.352E-06 |
| Odds of getting cancer from above exposure: | 1 in | 11,838,322 | 2,959,581 | 739,895 |

Table 3. Toxic Equivalency Factors (TEF) for PAHs [21].

| PAH | TEF |
|---|---|
| Acenaphthene | 0.001 |
| Acenaphthylene | 0.001 |
| Anthracene | 0.01 |
| Benzo(a)anthracene | 0.1 |
| Benzo(a)pyrene (BaP) | 1 |
| Benzo(b)fluoranthene | 0.1 |
| Benzo(g,h,i)perylene | 0.01 |
| Benzo(k)fluoranthene | 0.1 |
| Chrysene | 0.01 |
| Dibenzo(a,h)anthracene | 5 |
| Fluoranthene | 0.001 |
| Fluorene | 0.001 |
| Indeno(1,2,3-cd)pyrene | 0.1 |
| 1-Methylnaphthalene* | 0 |
| 2-Methylnaphthalene | 0.001 |
| Naphthalene | 0.001 |
| Phenanthrene | 0.001 |
| Pyrene | 0.001 |

* A TEF for 1-Methylnaphthalene was not available. It is assumed the toxicity of this compound is minimal (mainly eye and skin irritation) and so a TEF of 0 was assigned for this compound.

Table 4. Contaminants that exceeded HAC values in sediment samples collected June 2004 and November 2005 from Days Creek, Waggoner Creek, and Howard Creek. All other compounds were below the detection limit or, if detected, below the HAC value. For the compounds in this table, if the concentration was less than the detection limit, a value of one-half the detection limit was used for analysis.

| Contaminant | Concentration Range (mg/kg) | # Detected/ # Samples Collected | # Samples that exceed HAC value | HAC value (mg/kg) |
|---|---|---|---|---|
| BaP TEQ (June 2004) | 0.14-21.26 | 12/12 | 12 | 0.1 (CREG) |
| BaP TEQ (November 2005) | 0.03-14.28 | 53/53 | 51 | 0.1 (CREG) |
| Arsenic (June 2004) | 3.07-7.90 | 4/4 | 4 | 0.5 (CREG) |
| Arsenic (November 2005) | 0.42-105 | 53/53 | 51<br>2 | 0.5 (CREG)<br>20 (child chronic EMEG) |

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Table 5. Non-carcinogenic estimated daily doses for children exposed to contaminants via incidental ingestion of creek sediment.

| **Exposure Factor** | **EF=(F*ED)/AT** |
|---|---|
| **EF=exposure factor (unitless)** | **0.95890411** |
| F=frequency of exposure (day/year) | 350 [a] |
| ED=exposure duration (year) | 6 [b] |
| AT=averaging time (ED*365 day/year) | 2190 |
| | |
| **Sediment Ingestion Exposure Dose BaP TEQ** | **D=(C*IR*EF*CF)/BW** |
| **D=exposure dose (mg/kg/d)** | **0.0003** |
| C=contaminant concentration (mg/kg) | 21.26 [c] |
| IR=intake rate of soil (mg/day) | 200 [b] |
| EF=exposure factor (unitless) | 0.95890411 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 16 [b] |
| | |
| **D=exposure dose (mg/kg/d)** | **0.0002** |
| C=contaminant concentration (mg/kg) | 14.28 [d] |
| IR=intake rate of soil (mg/day) | 200 [b] |
| EF=exposure factor (unitless) | 0.95890411 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 16 [b] |
| | |
| **Sediment Ingestion Exposure Dose Arsenic** | **D=(C*IR*EF*CF)/BW** |
| **D=exposure dose (mg/kg/d)** | **0.00009** |
| C=contaminant concentration (mg/kg) | 7.90 [c] |
| IR=intake rate of soil (mg/day) | 200 [b] |
| EF=exposure factor (unitless) | 0.95890411 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 16 [b] |
| | |
| **D=exposure dose (mg/kg/d)** | **0.001** |
| C=contaminant concentration (mg/kg) | 105.00 [d] |
| IR=intake rate of soil (mg/day) | 200 [b] |
| EF=exposure factor (unitless) | 0.95890411 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 16 [b] |
| | |
| **D=exposure dose (mg/kg/d)** | **0.0002** |
| C=contaminant concentration (mg/kg) | 13.78 [e] |
| IR=intake rate of soil (mg/day) | 200 [b] |
| EF=exposure factor (unitless) | 0.95890411 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 16 [b] |

[a] TRRP standard default, 30 TAC 350.74(a) [22]
[b] PHAGM standard default, child 1-6 years old [11]
[c] Maximum concentration, June 2004 sampling event
[d] Maximum concentration, November 2005 sampling event
[e] 95% concentration, November 2005 sampling event

Table 6. Estimated lifetime excess cancer risk from exposure to contaminants via incidental ingestion of creek sediment.

| **Exposure Factor** | **EF=(F*ED)/AT** |
|---|---|
| **EF=exposure factor (unitless)** | **0.410958904** |
| F=frequency of exposure (d/y) | 350 [f] |
| ED=exposure duration (y) | 30 [f] |
| AT=averaging time (70*365 d/y) | 25550 |
| | |
| **Sediment Ingestion Cancer Risk BaP TEQ** | **CR=((C*IR*EF*CF)/BW)*CSF** |
| **CR=excess lifetime cancer risk (unitless)** | **9.1E-05** |
| C=contaminant concentration (mg/kg) | 21.26 [c] |
| IR=intake rate of soil (mg/day) | 100 [g] |
| EF=exposure factor (unitless) | 0.410958904 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 70 [g] |
| CSF=cancer slope factor $(mg/kg/d)^{-1}$ | 7.3 |
| | |
| **CR=excess lifetime cancer risk (unitless)** | **6.1E-05** |
| C=contaminant concentration (mg/kg) | 14.28 [d] |
| IR=intake rate of soil (mg/day) | 100 [g] |
| EF=exposure factor (unitless) | 0.410958904 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 70 [g] |
| CSF=cancer slope factor $(mg/kg/d)^{-1}$ | 7.3 |
| | |
| **Sediment Ingestion Cancer Risk Arsenic** | **CR=((C*IR*EF*CF)/BW)*CSF** |
| **CR=excess lifetime cancer risk (unitless)** | **7.0E-06** |
| C=contaminant concentration (mg/kg) | 7.90 [c] |
| IR=intake rate of soil (mg/day) | 100 [g] |
| EF=exposure factor (unitless) | 0.410958904 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 70 [g] |
| CSF=cancer slope factor $(mg/kg/d)^{-1}$ | 1.5 |
| | |
| **CR=excess lifetime cancer risk (unitless)** | **9.2E-05** |
| C=contaminant concentration (mg/kg) | 105.00 [d] |
| IR=intake rate of soil (mg/day) | 100 [g] |
| EF=exposure factor (unitless) | 0.410958904 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 70 [g] |
| CSF=cancer slope factor $(mg/kg/d)^{-1}$ | 1.5 |
| | |
| **CR=excess lifetime cancer risk (unitless)** | **1.2E-05** |
| C=contaminant concentration (mg/kg) | 13.78 [e] |
| IR=intake rate of soil (mg/day) | 100 [g] |
| EF=exposure factor (unitless) | 0.410958904 |
| CF=conversion factor ($10^{-6}$ kg soil/mg soil) | 0.000001 |
| BW=body weight (kg) | 70 [g] |
| CSF=cancer slope factor $(mg/kg/d)^{-1}$ | 1.5 |

[c] Maximum concentration, June 2004 sampling event
[d] Maximum concentration, November 2005 sampling event
[e] 95% concentration, November 2005 sampling event
[f] PHAGM standard default, cancer risk [11]
[g] PHAGM standard default, adult [11]