

# Health Consultation

## Tronox LLC, Texarkana Facility

Texarkana, Bowie County, Texas  
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### Prepared by

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## 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

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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



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## 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)<sup>1</sup> or

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<sup>1</sup> 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.



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EPA's reference doses (RfDs)<sup>2</sup>. 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

<sup>2</sup> 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.



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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  $\mu\text{g}/\text{day}$ . Smoking one pack of unfiltered cigarettes per day increases this estimate by an additional 2 to 5  $\mu\text{g}/\text{day}$ ; smokers consuming three packs per day increase their exposure by an estimated 6 to 15  $\mu\text{g}/\text{day}$ . The intake of carcinogenic PAHs from the average American diet has been estimated to range from 1 to 5  $\mu\text{g}/\text{day}$ , mostly from the ingestion of unprocessed grains and cooked meats. This dietary estimate increases to 6 to 9  $\mu\text{g}/\text{day}$  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



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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

