# **Health Consultation**

**Residential Soils** 

SOUTHEASTERN WOOD PRESERVING NPL SITE CANTON, MISSISSIPPI

EPA FACILITY ID: MSD000828558

SEPTEMBER 8, 2014

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES Agency for Toxic Substances and Disease Registry Division of Community Health Investigations Atlanta, Georgia 30333

#### Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or 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 concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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#### HEALTH CONSULTATION

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Prepared By:

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

#### **Summary**

Introduction The top priority of the Agency for Toxic Substances and Disease Registry (ATSDR) is to reduce harm to humans caused by chemicals in our environment. ATSDR is concerned about hazards posed to neighborhoods near the Southeastern Wood Preserving Site in Canton, MS. The purpose of this public health consultation is to evaluate available data and information on the contamination of household yards near the site to determine if people could be harmed by coming into contact with those substances. ATSDR is suggesting ways to reduce the hazards until they can be removed. Background The Southeastern Wood Preserving National Priorities List (NPL) site is located on Covington Road in Canton, MS, across from the Canton Municipal Utilities Wastewater Treatment. Between 1928 and 1979, several companies conducted wood treating operations at the site. Both coal tar creosote and pentachlorophenol were used. Three unlined wood treating and/or waste treating lagoons were located on site. Prior to the passage of environmental laws in the 1970's, waste materials were discharged directly into the adjacent Batchelor Creek. The Creek has flooded an unknown number of times in the past; the floodwaters may have carried contamination into the neighborhoods to the south of the site. Beginning in the fall of 2012, the US Environmental Protection Agency (EPA) sampled the yards of homes in the area along with other areas of concern related to the site. EPA requested ATSDR to evaluate the sample results in February 2013. ATSDR concludes that the dioxin contamination in soil south of the Southeastern **Conclusion 1:** Wood Preserving site and north of Barfield Street could harm the health of children and long-term residents in that neighborhood. This contamination also poses a slightly increased lifetime risk of cancer. This is a Public Health Hazard. **Basis:** Most of the individual cogeners of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), including TCDD itself, are at or below levels of health concern. When the environmental concentrations are adjusted to equivalent toxicity using the method developed by the World Health Organization, the mixture of all the dioxin and dioxin-like compounds found in some residential yards along Covington Street and the north side of Barfield Street pose a chronic health hazard under the exposure assumptions typical of residential areas. The hazard associated with exposure is possible effects on reproduction and child development as described in section 2 below. The equivalent toxicity concentration of the dioxin in this area may result in a slight increase in the estimated lifetime cancer risk for children and long-term residents. The long history of wood treating at this location and periodic flooding make it difficult to determine when exposure began and how long it has been occurring.

<u>Conclusion 2:</u>	ATSDR concludes that the concentrations of polycyclic aromatic hydrocarbons (PAHs) south of the Southeastern Wood Preseving Site and north of Barfield Street pose an increased risk of cancer for children and long-term residents in that neighborhood. While the PAHs are not at concentrations associated with non-cancer health effects, their presence near locations with dioxins above the ATSDR chronic Minimal Risk Level (MRL) could increase the likelihood of potential health effects for children and long-term residents. This is a Public Health Hazard. The PAHs add to the Public Health Hazard posed by the dioxin compounds found in the community.
<u>Basis:</u>	The individual PAHs identified in this dataset are below levels associated with non- cancer health effects. When added together using equivalency factors in accordance with the guidance published by the US Environmental Protection Agency (EPA) in 1993, the combined concentrations represent an increase in the estimated cancer risk in a residential setting. Because PAHs can target some of the same organs and result in some of the same health effects as dioxins, the presence of PAHs in the same locations as dioxins may result in an increased risk of those health effects occurring. As skin contact with soils contaminated by PAHs (described in section 2 below) may result in equal or higher internal doses, precautions to reduce such exposure are warranted.
Recommendations	ATSDR recommends that:
	• Surface soil contaminated by concentrations greater than EPA's current screening levels be removed as indicated by current EPA policy as resources permit.

- Information about ways and means of reducing potential exposure as well as demonstrations of methods to reduce exposure be given to homeowners and occupants of dwellings with contaminated soil. Expansion of this educational effort to workers in businesses in the area should be considered.
- Further characterization of the extent of contamination around the site and along the streambed of Batchelor Creek should be considered as indicated by current and planned sampling.

For MoreYou can call ATSDR at 1-800-CDC-INFO for more information on theInformationSoutheastern Wood Preserving Site.

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#### 1.0 Background

The Southeastern Wood Preserving National Priority List (NPL) site is located on Covington Road in Canton, MS, across from the Canton Municipal Utilities Wastewater Treatment Plant. After referral by the State and a number of site related activities, the site was proposed for the National Priorities List in March 2011. [1] This prompted a legislative mandate on the Agency for Toxic Substances and Disease Registry (ATSDR) to conduct a public health Assessment or such other public health activities as deemed appropriate under the Comprehensive Environmental Response, Compensation, and Liability Act, as amended, [42 USC 9604(i)(6)]. This public health consultation is being prepared to fulfill a part of that mandate. A glossary of terms and acronyms used in the document can be found at the end of the document before the appendices.

#### 1.1 Site History

Between 1928 and 1979, several companies conducted wood treating operations at the site. Both coal tar creosote and pentachlorophenol (PCP) were used. Three unlined wood treating and/or waste treating lagoons were located on site. The site was originally part of the much larger King Lumber facility. Several companies operated the facility either as a tenant of King or, after 1964, as an independent company. Prior to the passage of environmental laws in the 1970's, waste materials were discharged directly into the adjacent Batchelor Creek.[2] Other companies, including Southeastern Wood Preserving, acquired the assets of the facility over a period of years, but either did not treat wood or did so intermittently. Beginning in the 1970's, the State issued a number of violations and citations to the facility. Operations ceased in 1979 when the owner/operator at that time declared bankruptcy. Batchelor Creek has flooded an unknown number of times in the past and, according to anecdotal reports from long time residents, the floodwaters crossed over the site and into the residential yards to the south. [1] See Figure 2 in Appendix B. These floods likely contributed to the distribution of site contaminants through the community.

When operations ceased in 1979, there were large areas of the site visibly contaminated, piles of contaminated soil, creosote sludge storage tanks, and the 3 unlined lagoons. In 1985, the State conducted a preliminary assessment and site inspection of the facility. Various polycyclic aromatic hydrocarbons (PAHs) and PCP were detected in samples collected from on-site soils and sediments and water in the creek. In 1986, the US Environmental Protection Agency (EPA) conducted a removal action that stabilized and stockpiled the sludges from the 3 lagoons. These sludges were treated on site in the early 1990's and then stored in an on-site containment cell. In 1988, the US Department of Agriculture was conducting soil erosion prevention measures along Batchelor Creek and observed an oily waste entering the streambed from the site. Additional investigations and cleanup actions were undertaken by the State and EPA at various times through the 1990's and early 2000's. When these actions were completed by late 2010, creosote contaminated soils were excavated from the site and disposed of appropriately. A slurry wall was installed to prevent underground wastes from entering the creek. The site was proposed to the NPL by EPA in September 2011 and listed in early 2012. [1] Analysis of samples collected from residential soils primarily along Covington Street in 2008 detected the presence of PAHs and dioxins.[3] Additional details on the site history and the history of state and federal regulatory actions are available in the records at EPA and the State Department of Environmental Quality (DEQ).

In the fall of 2012, EPA initiated a remedial investigation of the site taking samples along Batchelor Creek and in the nearby community. On February 5, 2013, the EPA Remedial Project Manager (RPM) - the lead federal official for the site - shared some of the results from their sampling efforts and requested a health consultation from ATSDR on the soil contamination in residential yards. [4] Additional samples of water and sediments from Batchelor Creek were also collected, analyzed, and shared by EPA. However, those results are beyond the requested scope of this consultation and may be evaluated in later site documents.

Residential soil samples were collected (See Figure 3 in appendix B):

- along Covington Street across from the site between Miller Street and Hargong Street (east of the water plant);
- along both sides of Barfield Street between Miller Street and Parker Street; and,
- along both sides of Miller Street from North Street to the Batchelor's Creek Bridge and
- along the creek towards town.

From the distribution of contamination identified by the sampling, flooding of Batchelor's Creek appears to have affected the movement of the contaminants into the neighborhood.

Based on a preliminary evaluation of these sample results, EPA initiated a removal action in November 2013 to excavate the surface layer of contaminated soils from these homes and store it on the site pending final remediation. ATSDR concurred in this action. [5]

#### **1.2 Wood Treating**

Wood is treated with preservatives to protect it from mechanical, physical, and chemical influences. Preserved wood is used primarily in the construction, railroad, and utilities industries to prevent rotting when wood is exposed to damp soil, standing water, or rain, and as protection against termites and marine borers. There are multiple types of wood treating, but a relatively common method across the Southern US for many years involved organic chemical preservatives – creosote and pentachlorophenol (PCP). This method allowed the preservative to permeate, or soak into, the wood with little or no pressure to drive the chemical into the fabric of the wood. The moisture content of the freshly cut timber must be reduced first and then the lumber is placed in vats or lagoons filled with the preservative. The preservative then soaks into the wood more or less in place of the moisture which had been taken out. The wood is then removed and often excess preservative is allowed to drip off the timber into the ground. Originally the lagoons were unlined and the preservative may have leached into the ground. Later, treatment facilities began containing these materials better. [6]

Creosote is a mixture of many chemicals, including polycyclic aromatic hydrocarbons (PAHs) and methyl phenols (also known as cresols); many of the components in creosote are naturally occurring tars. Creosote used in treating wood is a thick oily liquid, usually amber to black in color. It may have an iridescent quality (i.e., rainbow colors) in sunlight. Released to the environment, creosote will separate into water soluble and water insoluble fractions. The smaller water soluble fraction tends to be more mobile in soil and wash away. The water insoluble fraction tends to be much less mobile and may appear like soft tar. [7] PAHs tend to be part of the water insoluble fraction.

Pentachlorophenol is a man-made chemical and is a solid in its pure state. In wood treating formulations, it is usually mixed with oils and other organic chemicals to form a liquid. Exposed to the open environment, PCP tends to break down in the environment within a few months. [8] Because it is a man-made chemical containing chlorine, a trace impurity commonly known as dioxin may be formed during production. Dioxin may also be formed when PCP is broken down by heat. Most dioxins are much more persistent in the environment than PCP. That is, when PCP is released into the environment, dioxins are often released with it. Over time, the PCP breaks down but the dioxin remains.

#### **1.3 Demographics**

According to the 2010 US Census, the population within a one mile radius of the site totaled 5,249 people living in 1,937 housing units. The bulk of these people live in the neighborhoods to the south of the site. Of these, almost 80% identified their race as black. Approximately 10% were 6 years of age or younger and another 10% were 65 years of age or older. Roughly 20% of the population were women of childbearing age (See Figure 1 in Appendix B). [9]

#### 2.0 Discussion

ATSDR considers the best available environmental data of known quality that are representative of a specific location when evaluating potential or actual health hazards. The highest quality data are generally collected and analyzed by objective professionals according to standardized methods using laboratories certified to meet independent standards. Data that do not meet all of these conditions may provide useful insights into hazards at a site, but can be more difficult to interpret.

EPA shared laboratory results from the samples collected during the recent remedial sampling event with ATSDR. During the quality assurance review of the data by EPA, a number of results were flagged as estimated values or tentatively identified compounds. Flagged results indicate that the collection and/or analysis of the sample were outside the calibration range of the instrument or other requirements intended to ensure the highest quality results. [10,11,12,13] In most cases, these results were between the quantification and the qualitative limits of the appropriate standardized method; rarely, the results may be above the calibration range of the instrument. That is, the lab results were between the concentration that can be accurately measured (the quantification limit) in the media being sampled (e.g., water or soil) and the concentration where the instrument can "see" the substance (the detection limit) but not measure it accurately. In those instances where the analytical results were above the calibration range, ATSDR accepts the EPA estimated value. ATSDR reviewed the laboratory results that were not flagged in the quality review or were higher than the calibration range of the analytical instrument and evaluated these results as described in Appendix A.

Comparison values represent a concentration or a dose of a substance at which harmful effects would not be expected. Exceeding a comparison value is an indication of the need for further review and evaluation. Comparison values do not predict adverse health effects, nor should they be used as the sole basis for setting clean-up levels. [14]

ATSDR's evaluation of data continues for those chemicals where the highest concentration at a site exceeds the comparison value and/or if a chemical that is present does not have a comparison value, as described in subsection 2.1 below. The next step is to estimate how humans might be exposed to the chemical – called the exposure pathway – as discussed in subsection 2.2. Then, the maximum dose for each chemical and pathway can be calculated as described in Appendix A and presented in subsection 2.3. ATSDR then discusses what the exposures, outcomes, and concerns imply for the public health of

the community (see subsection 2.4). ATSDR cannot predict actual health effects for individuals, but we can identify actual or potential health threats and recommend actions to prevent those threats from developing.

Of the chemicals identified in the samples from the community around Southeastern Wood Preserving, only PAHs and dioxins were higher than our comparison values. The concentrations reported for all the other compounds detected were below their comparison value or no comparison values have been established. The terms, PAHs and dioxins, actually refer to two different groups of similar chemicals. Approximately 100 individual chemicals are identified as PAHs; about 75 individual chemicals are identified as dioxins. As at most sites, a mixture of chemicals that fit each group's general properties were identified at Southeastern Wood Preserving. Appendix C describes how the toxicity of the PAH mixture is treated in this consultation while Appendix D describes the treatment of the dioxin mixtures.

#### 2.1 Chemicals above Comparison Values.

In Table 1, the maximum concentration of each substance detected above our comparison values in the residential areas is compared with the comparison values for childhood exposures. Concentrations from samples in areas not associated with homes (e.g., commercial or vacant properties) may be higher. [,10,11,12,13] PAH equivalents (PAH Eqs) are calculated using results of individual PAHs weighted according to their toxicity relative to benzo(a)pyrene (BaP); details are found in Appendix C. TCDD TEQ is calculated from the results for individual dioxin and furan chemicals weighted according to their toxicity relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD); details can be found in Appendix D.

Substance	Number of Samples above Comparison Value	Highest Concentration Measured	Range of values	Comparison Value
TCDD TEQ (ng/kg <sup>1</sup> )	19	1013	0.62 to 1013	50 <sup>2</sup>
PAH Eq $(ug/kg^3)$	12	5491	8.4 to 5491	96 <sup>4</sup>

Table 1: Chemicals detected above Comparison Values in Residential soil.

<sup>1</sup>-ng/kg = nanogram of dioxin per kilogram of soil (part per trillion);

<sup>2</sup> – Based on ATSDR Chronic Minimal Risk Level (MRL). See Appendix A

<sup>3</sup> - ug/kg=microgram of PAHs per kilogram of soil (part per billion)

<sup>4</sup> – Based on cancer risk. See Appendix A

#### 2.1.1 Dioxins or TCDD TEQ

The chronic MRL for TCDD results in a comparison value of 50 ng/kg for a child ingesting 200 mg of soil per day. Eleven out of 19 residential samples collected on Barfield Street had dioxin TEq over 50 ng/kg. On Covington Street, only 3 samples were considered residential samples and none of those had dioxin TEq above 50. Of the commercial properties, 7 samples were above the residential comparison value. On Miller Street, 10 samples were collected; 5 were residential samples. None of the residential samples and one of the commercial samples were above the residential comparison value (See Figure 3 in Appendix B).

#### 2.1.2 PAH or PAH Eq

The lifetime  $10^{-6}$  cancer risk for B(a)P is 96 ug/kg, commonly rounded to 100 ug/kg. That is, at this concentration in soil, one extra cancer case would be expected in a population of 1 million humans exposed to the soil over a 70 year lifetime. Eleven of fourteen residential samples collected on Barfield Street had B(a)P equivalents over 100 ug/kg. On Covington Street, none of the residential sample locations had a B(a)P equivalents over 100 ug/kg, but all of the commercial properties were above this comparison value . One home on Miller Street had B(a)P equivalents over 100 ug/kg. See Figure 3 in Appendix B. To help understand cancer risk, information from the American Cancer Society and the National Cancer Institute are provided in Appendix E.

#### 2.2 Exposure Pathway

Once contaminants that might be hazards have been identified, the next step is to estimate how individuals may come into contact with these hazards. People can be exposed to a chemical only if they breathe it in (inhale), eat (ingest) it - usually unintentionally, or come into skin contact (dermal) with the substance. If no one is exposed to a chemical, then no harmful health effects can occur. Additionally, harmful effects may not occur with every exposure. The type and severity of health effects a person may experience depends on a number of factors, including:

- 1. the concentration of the chemical (how much chemical),
- 2. the exposure frequency (how often),
- 3. the exposure duration (how long), and
- 4. the route or pathway of exposure.

Once an exposure occurs, characteristics such as age, sex, nutritional status, genetics, lifestyle, and preexisting health conditions of the individual influence how well the chemical is distributed, absorbed, and excreted. Together, the toxicity of the substance, the amount and kind of exposure and the characteristics of the individuals who are exposed determine the health effects that may occur.[13] These individual characteristics are the main reasons why ATSDR cannot predict health outcomes for any given person.

An exposure pathway is the process by which an individual is exposed to a chemical. ATSDR identifies and evaluates exposure pathways by considering the following 5 elements:

- 1. A source of contamination (Where the chemical comes from)
- 2. Transport through the environment (Where the chemical goes after it is released)
- 3. A point of exposure (Where people may come into contact with the chemical)
- 4. A route of exposure (How the chemical can get into the bodies of people)
- 5. A receptor population (A group of people that may be exposed to the chemical)

ATSDR categorizes an exposure pathway as completed or potential.

An exposure pathway is considered complete if all 5 elements of an exposure pathway exist at a site. An exposure pathway is considered potentially complete if some of the elements are known to exist and the

others are not known to be absent. No exposure pathway exists if any of the 5 elements are known to be absent.[14]

To illustrate the 5 elements, consider the following example:

Creosote from the lagoons on site [the source] may have been moved by flooding along Batchelor Creek [the transport mechanism] into the yards of the homes across the street [the point of exposure]. From the yards, family members living in the homes [the receptor population] may have unintentionally swallowed some of this contaminated dirt [the route of exposure].

The focus of this health consultation is residential soils near the Southeastern Wood Preserving site. Contaminants from the site detected in the yards of homes means that a completed exposure pathway exists for residents who come in contact with soil in their yards. ATSDR thus continues the evaluation to determine whether that exposure could cause harm.

#### 2.3 Dose Calculations

The concentrations reported in the soil in Table 1 do not represent what individuals may be absorbing. They have to be converted as described in Appendix A into doses in order to be compared with the relevant human or animal toxicological data available on the substance.[14] Many variables affect how well any individual will absorb a chemical from soil and how that individual may react to that chemical. It is generally impossible to predict or measure those variables, but we can estimate the potential ranges for many of the variables. By selecting the more conservative values from those ranges, we can calculate a "worst-case" dosage that may be absorbed. An exposure to a lower concentration would generate a lower dose, so the focus is on these higher values. For many chemicals, dermal absorption would also be considered for soil and sediment samples.

This oral dose calculation in Appendix A assumes 100% of the contaminant in the soil is absorbed by the person unintentionally eating the dirt. The exposure factor takes into account how often an individual is exposed (the frequency), how long each exposure generally lasts (the duration), and how long a person may experience this exposure over time (averaging time).[14] In order to apply this equation to the exposure scenario discussed in Section 2.2, the following will be assumed for this site:

- C = the maximum concentration in Table 1 above.
- IR = 200 mg/day for an infant or a child and 100 mg/day for an adult (200 mg is less than a quarter teaspoon.)
- The exposure factor will be assumed to be one, which means we are assuming all residents are home 24 hours per day and 7 days per week.
- Body weight will be 10 kg for an infant (less than 1), 16 kg for a child (age 1-6), and 70 kg for older children and adults.

For dermal exposure, the following assumptions will be made:

\* a toddler crawls in the dirt twice a day, exposing arms, hands, legs, and feet (about 2930 square centimeters of skin);

- \* a child walks barefoot in the yard every day, exposing legs and feet(about 5260 square centimeters of skin); and,
- \* an adult mows the lawn once a week, exposing arms and hands(about 5300 square centimeters of skin).

#### 2.3.1 Substances with Health Guidance Values

For those compounds in Table 1 above the comparison value, the doses for the various exposure scenarios described above are calculated in Table 2 and then compared with the health guidance value. Cancer risk is estimated in Table 3 assuming a lifetime of exposure by both incidental ingestion and skin contact and absorption.

#### Table 2: Estimated Oral Doses of Chemicals above Comparison Values.

Substance	Infant dose	Child dose	Adult Dose	Health Guidance Value
TCDD TEQ $(ng/kg/day^{1})$	0.02	0.01	0.001	0.001 <sup>3</sup>
$\frac{PAH Eq}{(\mu g/kg/day^2)}$	0.1	0.07	0.007	30, 000 <sup>4</sup>

<sup>1</sup> – nanograms of dioxin per kilogram of body weight per day

 $^{2}$  – micrograms of PAHs per kilogram of body weight per day

<sup>3-</sup>ATSDR Chronic MRL for exposures over 1 year.

<sup>4</sup> – EPA RfD for Pyrene for exposures over a lifetime

Bold Text=Dose exceeds Health Guidance Value.

#### Table 3: Estimated Dermal Doses of Chemicals above Comparison Values.

Substance	Infant dose	Child dose	Adult Dose	Health Guidance Value
TCDD TEQ	0.008	0.02	0.0001	0.001 <sup>3</sup>
$(ng/kg/day^{1})$				
PAH Eq	6	12	0.07	30, 000 <sup>4</sup>
$(\mu g/kg/day^2)$				

<sup>1</sup> – nanograms of dioxin per kilogram of body weight per day

<sup>2</sup> - micrograms of PAHs per kilogram of body weight per day

<sup>3</sup> – ATSDR Chronic MRL for exposures over 1 year.

<sup>4</sup> – EPA RfD for Pyrene for exposures over a lifetime

**Bold Text=Dose exceeds Health Guidance Value.** 

#### Table 4: Estimated Combined Oral and Dermal Doses of Chemicals above Comparison Values.

Substance	Infant dose	Child dose	Adult Dose	Health Guidance Value
TCDD TEQ (ng/kg/day <sup>1</sup> )	0.028	0.03	0.0011	0.001 <sup>3</sup>
PAH Eq $(\mu g/kg/day^2)$	6.1	12.07	0.077	30, 000 <sup>4</sup>

<sup>1</sup> – nanograms of dioxin per kilogram of body weight per day

<sup>2</sup> – micrograms of PAHs per kilogram of body weight per day

<sup>3</sup> - ATSDR Chronic MRL for exposures over 1 year.

<sup>4</sup> – EPA RfD for Pyrene for exposures over a lifetime

**Bold Text=Dose exceeds Health Guidance Value.** 

#### Table 5: Estimated Lifetime Risk of Cancer

Substance	Highest Concentration Measured	Estimated Cancer Risk *
TCDD TEQ (ng/kg soil)	1013	4.6 X 10 <sup>-4</sup>
PAH Eq (µg/kg soil)	5491	7.5 X10 <sup>-3</sup>

ng/kg = nanogram of dioxin per kilogram of soil (part per trillion)

ug/kg=microgram of PAH per kilogram of soil (part per billion);

\* Based on combined exposure of 6 years as a child and 27 years as an adult in the same home under the conditions described in Section 2.2.

#### 2.3.2 Dioxins or TCDD TEQ

Based on the exposure assumptions described here, the maximum daily dose (Table 4) exceeds the chronic MRL for TCDD equivalents as shown above for infants, children, and adults. Exposure to the maximum daily dose of the dioxin mixture in this residential area would represent a chronic health hazard (i.e., long-term exposure). Possible non-cancer health effects include harm to the liver and possible reproductive and developmental effects. Given the site history and the potential past exposures, activities to reduce current exposures pending the long term remedial action should be considered.[15] The short term removal action by EPA currently in progress should reduce the potential for future health hazards.

Dioxins have been classified as known human carcinogens by the International Agency for Research on Cancer (IARC) and the National Toxicology Program within the US. A cancer slope factor has not yet been derived by EPA; however, the State of California used EPA methodology to derive a cancer slope factor of 150,000 (mg/kg/day)<sup>-1</sup>. [16] ATSDR has chosen to use that slope factor in estimating the cancer risks posed by dioxin at this site shown in Table 5. The estimated increase would be an additional 4-5 cancer cases in a population of 10,000 over what is otherwise expected. This would be slightly above the normally acceptable range of cancer risk under the Superfund program. While diagnosis or incidence of all types of cancers in humans have been shown to increase after exposure to dioxins, the type of cancers once commonly associated with dioxin include lung cancer, non-Hodgkins lymphoma, prostate cancer, breast cancer, and rectal cancers. [17] Individual susceptibility and personal habits can significantly modify any person's risk of contracting cancer.

#### 2.3.3 Polycyclic Aromatic Hydrocarbons

The comparison value for PAHs in Table 1 is based on lifetime cancer risk of BaP while the health guidance value listed in Tables 2, 3, and 4 represents the lowest available non-cancer value for the chemicals in this class of compounds. The combined oral and dermal doses in table 4 are well below the non-cancer health guideline values for chemically similar compounds; therefore, non-cancer health effects would not be expected from exposure to these compounds.

The Cancer Slope Factor for BaP is 7.3 per mg/kg/day. [18] Assuming 33 years of residency (6 as a child and 27 as an adult) and a seventy year lifespan, the cancer risk represented by these maximums can be calculated by the formula discussed in Appendix A and is shown in Table 5. Based on the assumptions in this consultation, the estimated lifetime cancer risk due to exposure to these PAHs would be between 7 and 8 additional diagnosed cases of cancer in a population of 1000 typical Americans. These cancer risks are above the acceptable risk range for Superfund actions and would be considered a chronic health hazard. Individual susceptibility and personal habits can significantly modify any person's risk of contracting cancer. Studies of individual PAHs in humans have not shown a correlation with cancers; however, numerous studies in animals have associated exposure to PAHs with various cancers. The most common cancers associated with these exposures are lung, liver, stomach, and breast cancers. Based on these animal studies, several individual PAHs are considered probable or likely human carcinogens. However, not all cancer classification groups agree on which PAHs can be associated with cancer. [19] See Appendix E for additional information on cancer and cancer risks.

#### 2.3.4 Compounds with no Health Guidance Values

For those compounds with no existing health guidance values, additional review of pertinent data is required. This review is described in Appendix F. None of the chemicals in this category were detected at concentrations expected to represent a health threat.

#### 2.3.5 Cancer

Appendix E provides information from the National Cancer Institute and the American Cancer Society on cancers in general. Table 10 in that appendix compares the incidence or diagnosis rates for the types of cancers associated with the chemicals at this site for the United States, the State of Mississippi, and Madison County. Table 11 provides the same comparison for deaths caused by cancers. Additional information may be available from the Mississippi Department of Health or the State Cancer Registry. [20]

Because the cancer risks calculated in sections 2.3.2 and 2.3.3 are not for specific forms of cancer, this discussion will focus on all cancers first. Mississippi has higher cancer incidence and mortality rates than the national average. Madison County, in general, has higher rates than the state. The incidence rate of all cancers from 2006-2010 in Madison County is about 473 per 100,000 or about 4.7 per 1000 people or  $(4.7 \times 10^{-3})$ . Compared to these actual statistics from a 5 year period, the estimated cancer risk over a lifetime of exposure to the PAHs is slightly higher while the estimated cancer risk for dioxin is roughly an order of magnitude smaller.

Looking more closely, the actual rates over that 5 year period in Madison County for the types of cancer associated with exposure to PAHs - except breast cancers - are actually comparable to the national rates and below the state rates as shown in Table 10. The incidence of breast cancer in Madison County is slightly higher than the state or the national rate. This implies the higher actual rate of all cancer diagnosis in the county may be due to other forms of cancers than those associated with exposure to the chemicals at this site. Given that individual susceptibility and personal habits can significantly modify any person's risk of contracting cancer and the relatively small number of people living in the community, it is likely that further study would not be able to link the exposures at this site to the incidence of any cancer in the community.

#### **2.4 Public Health Implications**

The analytical results from these samples indicate the dioxins in portions of the area bounded by Covington Drive, Parker Street, Barfield Street, and Miller Street represent a chronic health hazard to long-term residents, both past and present. Health effects may include both cancer and non-cancer outcomes. The possibility of future health effects should be reduced significantly due to the current response operation being undertaken by the EPA. Individual health effects may not occur because of differences in susceptibility, personal habits, and the bioavailability of the specific contaminants in those yards with elevated concentrations. Results from outside this block of homes indicate the presence of contamination (e.g., in commercial properties or along the banks of Batchelor's Creek), but the exposure anticipated in those locations are likely to be shorter than in a residential setting and no hazard to human health would be expected.

Lifetime exposure to these concentrations of the PAHs has been associated with a higher incidence of cancer in animals. Other health effects would not be anticipated at the concentrations detected in the soils. As with the dioxin or any other chemical exposure, individual health effects may not occur because of difference in susceptibility, personal habits, and the bioavailability of these chemicals. The potential for future health effects should be reduced by the current operation by EPA.

Individuals most susceptible to the health effects of chronic hazards are those with the longer potential period of exposure, such as children or individuals who have always lived in this area. Depending on all of these variables, the concentration of dioxin and PAHs and the possible exposure doses in this one block could result in adverse health outcomes. A few simple precautions can temporarily reduce the exposure and potential threat to the residents in the community until the hazard can be reduced or eliminated. These precautions include frequently washing the hands of individuals playing or working in the yards; wiping soil and mud off your shoes before coming into the house; wearing clothing with long sleeves and pants legs to reduce direct contact; and other similar precautions. Please see Appendix G for further guidance on these steps to reduce potential harm.

The conclusions in this consultation are based on the maximum concentration detected in this round of sampling by EPA. While the area with concentrations above the comparison value described in section 2.1 covers a substantial portion of the block described above, the other samples reflect a lower threat. However, even the most complete and detailed sampling effort represents a snapshot of contamination over time and over an area. Assuming the maximum concentration detected is representative of the maximum doses received over time by residents, the most likely type of health effects would be reproductive or developmental.

Based on animal studies of chronic exposure to TCDD in the diet at doses similar to those represented by the maximum at this site, female monkeys experienced an increased number of miscarriages, difficulty conceiving, and a condition called endometriosis. The offspring of monkeys with chronic exposure to similar doses had decreased survival, altered social behavior, and increased learning impairments. Some rodents fed TCDD at daily doses close to the maximum found at this site developed abnormal cells in their livers. None of these effects were seen in humans exposed to TCDD. For dioxins other than TCDD, these effects did not occur in animals until much higher doses. [15]

Because both dioxins and PAHs cause some of the same health effects in humans and in animals (e.g., cancer, liver effects), the doses of these two classes needs to be considered in tandem. The relative doses of these chemicals in relationship to their health guidance values indicated that the effects can be considered additive.[21]

#### 2.5 Child Health Considerations

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults; this means they breathe dust, soil, and vapors close to the ground. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, 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.

This consultation uses child-specific exposure factors, such as body weights, intake rates, and skin exposure areas, as the basis for calculating exposures to contaminants in soil (Appendix A). The resulting exposure doses for children are higher than adult doses and represent the basis for the following public health conclusions and recommendations. Additionally, soil data evaluated in this consultation includes sample locations from residential play areas and gardens. Remediation has been initiated at the residential properties.

#### 2.6 Adequacy of Available Data and Data Limitations

The soil data collected by EPA and discussed in this health consultation appears generally adequate for the ATSDR conclusions and recommendations for residents in the area. The detection limits for dioxin are somewhat high compared to the lifetime excess cancer risks for exposure to these compounds based on the California cancer slope factor used here but represent state-of-the-art analysis of dioxins in soils. The information provided to ATSDR indicated the analytical results provide adequate precision and accuracy. Some results considered here were above the calibration range of the instrumentation and are estimated values. Other adjustments made by ATSDR to the data provided by EPA are described in the introductory paragraphs to Section 2 above.

In Subsection 2.4, ATSDR described a possible additive relationship between the effects of dioxin and PAHs. This is based on the fact that the two groups of chemicals affect the same target organs and, depending on individual susceptibility, may produce similar health effects. No studies to ATSDR's knowledge have demonstrated any interaction between these chemicals. The possibility is discussed

here primarily because both groups of chemicals are already considered health hazards in their own right.

The limitations of the data from the Mississippi Cancer Registry (discussed in subsection 2.3.5 and Appendix E) is best described by the state at their website. In general, data from such registries does not capture important factors such as lifetime exposures of the individuals or contributing factors to the disease. For instance, an individual may live their entire life elsewhere before moving to the state and discovering they have cancer. The environment where they lived before moving to Mississippi may have contributed significantly to the onset of the disease. Another important factor not typically captured in the statistics cited here is that some cancers do seem to run in some families. This is primarily due to genetic factors passed from parents to offspring.

In Subsection 2.3, dermal absorption of these chemicals was discussed. Many factors affect how well chemicals cross the skin barrier; not all of them are known. Some of these include the thickness of clothing worn, how long the contaminated dirt was allowed to remain in contact with the skin, and how tightly the contamination holds on to the soil particles. Even relatively small variations in these factors can affect how much of the chemicals are actually absorbed by any single person through this route of exposure.

#### 3.0 Conclusions

<u>Conclusion 1:</u> ATSDR concludes that the dioxin contamination in soil south of the Southeastern Wood Preserving site and north of Barfield Street could harm the health of children and long-term residents in that neighborhood. This contamination also poses a slightly increased lifetime risk of cancer. This is a Public Health Hazard.

<u>Basis:</u> Most of the individual cogeners of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), including TCDD itself, are at or below levels of health concern. When the environmental concentrations are adjusted to equivalent toxicity using the WHO method, the mixture of all the dioxin and dioxin like compounds found in some residential yards along Covington Street and the north side of Barfield Street present a chronic health hazard under the exposure assumptions typical of residential areas. The hazard associated with exposure would be possible effects on reproduction and child development as described above. The equivalent toxicity concentration of the dioxin in this area may result in a slight increase in the estimated lifetime cancer risk for children and long-term residents. The long history of wood treating at this location and the periodic flooding makes it difficult to determine when exposure began and how long it has been occurring.

<u>Conclusion 2:</u> ATSDR concludes that the PAHs concentrations south of the Southeastern Wood Preserving Site and north of Barfield Street pose an increased risk of cancer for children and long-term residents in that neighborhood. While the PAHs are not at concentrations associated with non-cancer health effects, their presence near locations with dioxins above the chronic MRL could increase the likelihood of potential health effects for children and long-term residents. This is a Public Health Hazard. The PAHs add to the Public Health Hazard posed by the dioxin compounds found in the community.

<u>Basis:</u> The individual PAHs identified in this dataset are below levels associated with non-cancer health effects. When added together using equivalency factors in accordance with the EPA 1993 guidance, the combined concentrations represent an increase in the estimated cancer risk in a residential setting. Because PAHs can target some of the same organs and result in some of the same health effects as

dioxins, the presence of PAHs in the same locations as dioxins may result in an increased risk of those health effects occurring. As skin contact with soils contaminated by PAHs (described above) may result in equal or higher internal doses, precautions to reduce such exposure are warranted.

#### 4.0 Recommendations

ATSDR recommends that:

- Surface soil contaminated by concentrations greater than EPA's current screening levels be removed as indicated by current EPA policy and as resources permit.
- Information about ways and means of reducing potential exposure as well as demonstrations of methods to reduce exposure be given to homeowners and occupants of dwellings with contaminated soil. Expansion of this educational effort to workers in businesses in the area should be considered.
- Further characterization of the extent of contamination around the site and along the streambed of Batchelor Creek should be considered as indicated by current and planned sampling.

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#### ATSDR Glossary of Environmental Health Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines some of the words used by ATSDR in communications with the public.

**Absorption** -The process of taking in. For a person or animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

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

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

**Background level** -An average or expected amount of a substance in a specific environment, or typical amounts of substances that occur naturally in an environment.

**Biologic uptake** -The transfer of substances from the environment to plants, animals, and humans. **Biota** -Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

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

**Cancer risk** -A statistical probability for getting cancer if a given population is exposed to a substance – typically calculated for an exposure of every day for 70 years (a lifetime exposure). The actual occurrence of cancer in that population might be different.

Carcinogen -A substance that causes cancer.

**Chronic exposure** -Contact with a substance that occurs over a long time (more than 1 year). **Comparison value (CV)** -Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

**Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) -**CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances.

**Concentration** -The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

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

**Dermal contact** -Contact with (touching) the skin.

**Dose** -The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An exposure dose is how much of a

substance is encountered in the environment. An absorbed dose is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

**Dose-response relationship** -The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media -soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

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

**EPA** -United States Environmental Protection Agency

**Exposure** -Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure]. **Exposure assessment** - The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure pathway -The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching); and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway. In most cases, response actions like remedial actions or removal actions are designed to interrupt the exposure pathway in order to reduce or eliminate harm. Groundwater -Water beneath the earth's surface in the spaces between soil particles and between rock surfaces.

Hazard -A source of potential harm from past, current, or future exposures.

Hazardous Materials – Substances that may cause harm to people, property, or the environment under some circumstances. In the US, Hazardous Materials are defined by the US Department of Transportation under the authority provided in the Hazardous Materials Transportation Act. See 49 CFR 172. All Hazardous Substances are Hazardous Materials, but not all Hazardous Materials are also Hazardous Substances.

Hazardous Substances – Substances that may cause harm to people or the environment under some circumstances. In the US, Hazardous Substances are defined by the US EPA under the authority provided in pollution laws such as CERCLA. See 40 CFR 302. Most Hazardous Wastes are also considered Hazardous Substances, but Hazardous Substances may not always be Hazardous Wastes. Hazardous waste -Potentially harmful substances that have been released or discarded into the environment. In the US, Hazardous Wastes are defined by the EPA under their authority provided by the Resource Conservation and Recovery Act. See 40 CFR 260.

**Ingestion** -The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way.

Inhalation -The act of breathing. A hazardous substance can enter the body this way. Intermediate duration exposure -Contact with a substance that occurs for more than 14 days and less

than a year.

Lowest-observed-adverse-effect level (LOAEL) -The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Migration - Moving from one location to another.

**Minimal risk level (MRL)** -An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects.

**National Priorities List (or NPL)** – EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis. Once a site is nominated for the NPL, certain actions in conjunction with a series of partner agencies are required of ATSDR by law.

**No-observed-adverse-effect level (NOAEL)** -The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

**Operable Unit** (OU) – A portion of a site with similar concerns. An OU may be an affected media such as the groundwater or a specific portion of a site like underground storage tanks.

**Point of exposure** -The place where someone can come into contact with a substance present in the environment.

**Prevention** -Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

**Public comment period** -An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

**Public availability session** -An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public health action -A list of steps to protect public health.

**Public health assessment (PHA)** -An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that are recommended to protect public health.

**Public health consultation (HC)** – An ATSDR product that answers a specific question regarding human health and hazardous substances. A HC may be verbal or written; if verbal, documentation of the HC will be developed after the fact. A HC may deal with a particular chemical or group of chemicals, a particular site, a specific release, a particular environmental media, a particular exposure, or a combination of all of these. A HC is a more focused document than a PHA, providing only enough information to answer the question posed to ATSDR. A series of HCs may be prepared in lieu of a single PHA for longer term responses like an NPL site.

**Reference dose (RfD)** -An EPA estimate, with uncertainty factors built in, of the daily dose of a substance that is unlikely to cause harm in humans over a lifetime of exposure.

**Remedial investigation (RI)** -The CERCLA process of determining the type and extent of hazardous material contamination at an NPL site. The data from an RI may be used to help determine the feasibility and scope of actions to remediate the site.

**Remedial Action -** Remedial Actions under Superfund are cleanup operations to resolve those hazards identified in the RI. Remedial actions may take years to complete and are often broken up into phases or specific portions of the site called operable units.

**Removal Action –** Removal Actions under Superfund are generally shorter-term response actions than Remedial Actions to address specific hazards at a site. Removals can happen at any time in the process from initial discovery until the site cleanup is determined to be complete.

**Risk** – The risk of harm exists when there is an exposure to a hazard. If the hazard can be removed, there is no further risk of harm. If the amount of exposure can be reduced, the risk of harm is also reduced. The management and elimination of risk due to exposure to hazardous substances at uncontrolled waste site is the reason why the Superfund process was put into place.

**Sample** -A portion or piece of a whole. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

**Soil/Sediment** – Sediments are soil samples taken from a streambed, lake, or other body of water. As opposed to soil samples, sediment samples usually have a high moisture content and may be more conducive to biological degradation of some chemicals than surface or subsurface soils.

**Site Investigation (SI)** – Any of a number of different types of field investigations of a site in order to determine the hazards associated with the site and the feasibility of the site being listed on the NPL. An SI may prompt further investigations or removal actions.

**Source of contamination** -The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Substance – As used here, a chemical.

**Surface/Subsurface Soil Samples –** Depending on the circumstances, the difference in depth between surface and subsurface samples is somewhat discretionary. Generally speaking, ATSDR assumes surface samples will be collected from a depth of 0-2 inches. With ground cover and caps, depths of up to 6 inches may be considered surface soils. Generally speaking any sample greater 6 inches would be considered subsurface.

**Surface water** -Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

**Survey** -A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment.

**Toxicological profile** -An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

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

**Uncertainty factor** -Mathematical adjustments applied when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Uncertainty factors as used in toxicology should not be confused with safety factors, as used in other disciplines like engineering.

#### **Glossary of Acronyms**

ATSDR	Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services
CERCL	A Comprehensive Environmental Response, Compensation, and Liability Act, commonly
CREG	Cancer Risk Evaluation Guide: Comparison Value based on a Lifetime one-in-one million increased risk
CV	Comparison Value
EMEG	Environmental Media Evaluation Guide: Comparison Value based on an ATSDR Minimal Risk Level.
EPA	US Environmental Protection Agency
MCL	Maximum Contaminant Level: an legally enforceable standard for drinking water
MICL	supplies
mø/kø	Milligram per kilogram or ppm in soil
mg/l	Milligram per liter or ppm in water
MRL	Minimal Risk Level: see glossary of terms for more information
NPL	National Priorities List CERCLA: part of the National Contingency Plan (see 40 CFR
	300)
NCP	National Contingency Plan: implementing regulation for CERCLA and other pollution
1101	laws Codified in Title 40 of the Code of Federal Regulations at part 300
OSC	On-Scene Coordinator: Lead federal or state Official for response to a site
PA	Preliminary Assessment: a phase of the CERCLA process for addressing sites
PHA	Public Health Assessment: a site specific report by ATSDR
pph	Parts per billion
nnm	Parts per million
RA	Remedial Action: a part of the CERCLA process for addressing sites on the NPL
RD	Remedial Design: a part of the CERCLA process for addressing sites on the NPL
RfD	Reference Dose see glossary of terms in annendix
RI	Remedial Investigation: a part of the CERCLA process for addressing sites on the NPL
RMEG	Reference Dose Media Evaluation Guide: comparison value based on EPA's RfD
RPF	Relative Potency Factor: a means of equating the toxicity of one compound to a similar
IXI I	compound using a specific health effect
RPM	Remedial Project Manager: Lead federal official for cleanup of a site
SI	Site Investigation or Site Inspection: a part of the CERLCA process for addressing
51	sites
SOP	Standard Operating Procedure
SVOCs	Semi-volatile Organic Compounds
TCLP	Toxicity Characteristic Leaching Procedure: a regulatory term for a procedure to
TCLI	classify a waste substance as hazardous by a physical characteristic of the material
TEF	Toxicity Equivalency Eactor: a means of equating the toxicity of one compound to a
	similar compound
VOCs	Volatile Organic Compounds
ug/kg	Microgram per kilogram or ppb in soil.
ug/l	Microgram per liter or ppb in water

#### Appendices

- A. ATSDR's Evaluation Process
- B. Figures
- C. Polycyclic Aromatic HydrocarbonsD. Polychlorinated Dibenzo Dioxins
- E. Cancer Classifications
- F. Compounds without Comparison Values Detected on site.G. Precautions to minimize contact with soil.

#### **Appendix A: ATSDR's Evaluation Process**

#### Step 1 – Comparison Values and the Screening Process

To evaluate the available data, ATSDR used comparison values (CVs) to determine which chemicals to examine more closely. CVs are the chemical concentrations found in a specific media (for example: air, soil, or water) and are used to select chemicals for further evaluation. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of air, soil, or water that someone may take into their body each day. CVs are generated to be conservative and non-site specific. These values are used only to screen out chemicals that do not need further evaluation. CVs are not intended as environmental clean-up levels or to indicate that health effects occur at concentrations that exceed these values.

CVs can be based on either carcinogenic (cancer-causing) or non-carcinogenic effects. Cancer-based comparison values are calculated from the U.S. Environmental Protection Agency's (EPA) oral cancer slope factor (CSF) or inhalation risk unit. CVs based on cancerous effects account for a lifetime exposure (70 years) with a theoretical excess lifetime cancer risk of 1 extra case per 1 million exposed people. Non-cancer values are calculated from ATSDR's Minimal Risk Levels (MRLs), EPA's Reference Doses (RfDs), or EPA's Reference Concentrations (RfCs). When a cancer and non-cancer CV exists for the same chemical, the lower of these values is used in the comparison for health protectiveness. The chemical and media-specific CVs utilized during the preparation of this document are listed below:

An Environmental Media Evaluation Guide (EMEG) is an estimated comparison concentration for which exposure is unlikely to cause adverse health effects, as determined by ATSDR from its toxicological profiles for a specific chemical.

A **Reference Dose Media Evaluation Guide** (**RMEG**) is an estimated comparison concentration that represents concentrations of chemicals (in water, soil, and air) to which humans may be exposed without experiencing adverse health effects.

A **Cancer Risk Evaluation Guide (CREG)** is a comparison concentration that is based on an excess cancer rate of one in a million persons and is calculated using EPA's cancer slope factor (CSF).

#### Step 2 – Evaluation of Public Health Implications

The next step in the evaluation process is to take those chemicals that are detected at concentrations above their respective CVs and further identify the site-specific exposure situations and the likelihood that these exposures could pose a health hazard. Therefore, calculations are performed to estimate the possibility of cancer and non-cancer health impacts. The calculations consider the activities of people living in the community.

#### Doses from Oral (Ingestion) Exposure

The calculation for oral exposure to contaminants in soil or sediment is accomplished using the following equation:

$$D = \frac{C X IR X EF X CF}{BW}$$

Where: D = exposure dose (in milligram of substance per kilogram of bodyweight per day or mg/kg/day or similar units)
C = concentration of the substance in the soil (in milligram of substance per kilogram of soils or mg/kg or similar units)
IR = intake rate of contaminated soil (in milligrams per day or mg/day or similar units) as described in the Discussion.
EF = exposure factor (unitless) - takes into account frequency and duration of exposure as described in the Discussion
CF = conversion factor – a constant value required by the formula
BW = Bodyweight (in kilograms or KG) as described in the Discussion.

#### Doses from Dermal Exposures (i.e., Skin Contact)

Many chemicals can be absorbed through the skin. To calculate a dose received from this router of exposure, a two step calculation is used. The first calculation is to estimate how well a chemical is absorbed in each exposure event. The second is to estimate how often such exposure events occur.

The calculation for the exposure event uses the following formula:

 $DA_{event} = C_{soil/sediment} X CF X AF X ABS_d$ 

Where: $DA_{event} =$  The absorbed Dose per Event (mg/cm<sup>2</sup>-event) $C_{soil/sediment} =$  Chemical concentration in soil or sediment (mg/kg)CF = Conversion Factor (10<sup>-6</sup> kg/mg)AF = Adherence factor of soil/sediment to skin (mg/cm<sup>2</sup>- event) (aka – contact rate) $ABS_d =$  Dermal Absorption fraction for soil and sediment

The calculation for the exposure frequency and duration results in the dermal absorbed dose as follows:

$$DAD = \frac{DAevent X EF X ED X EV X SA}{BW X AT}$$

Where:	DAD = Dermal Absorbed Dose (mg/kg/day)
	DAevent = Absorbed dose per event $(mg/cm^2 - event)$
	{as calculated in the preceding equation)
	EF = Exposure Frequency (days/year)
	ED = Exposure duration (years)
	EV = Event frequency (events/day)
	SA = Surface area available (cm2)
	BW = Body weight (kg)
	ATn = Averaging Time (non-cancer) = ED X 365 days/year
	ATc = Averaging Time (cancer)= 80 years X 365 days/year

#### Doses from Inhalation Exposures (i.e., Breathing)

Inhalation is not a significant pathway for these contaminants.

#### **Non-Cancer Health Effects**

The doses calculated for exposure to each individual chemical at the site are then compared to established health guidelines, such as ATSDR's Minimal Risk Levels (MRLs) or EPA's Reference Doses (RfDs), in order to assess whether adverse non-cancer health impacts from exposure are expected. These health guidelines, described in more detail in the following text, are chemical-specific values that are based on the available scientific literature and are considered protective of human health.

#### Minimal Risk Levels (MRLs)

ATSDR has developed MRLs for contaminants commonly found at hazardous waste sites. The MRL is an estimate of daily exposure to a contaminant below which non-cancer, adverse health effects are unlikely to occur. MRLs are developed for different routes of exposure, such as inhalation and ingestion, and for lengths of exposure, such as acute (less than 14 days), intermediate (15-364 days), and chronic (365 days or greater). At this time, ATSDR has not developed MRLs for dermal exposure. A complete list of the available MRLs can be found at <u>http://www.atsdr.cdc.gov/mrls.html</u>. For this health consultation, ATSDR utilized Oral MRLs for chronic exposures when possible. Oral Intermediate MRLs were used when chronic MRLs were unavailable.

#### **Reference Doses (RfDs)**

An estimate of the daily, lifetime exposure of human populations to a possible hazard that is not likely to cause non-cancerous health effects. RfDs consider exposures to sensitive sub-populations, such as the elderly, children, and the developing fetus. EPA's RfDs have been developed using information from the available scientific literature and have been calculated for oral and inhalation exposures. A complete list of the available RfDs can be found at http://www.epa.gov/iris.

Non-carcinogenic effects, unlike carcinogenic effects, are believed to have a threshold, that is, a dose below which adverse health effects will not occur. As a result, the current practice for deriving health guidelines is to identify, usually from animal toxicology experiments, a No Observed Adverse Effect Level (or NOAEL), which indicates that no effects are observed at a particular exposure level. This is the experimental exposure level in animals (and sometimes humans) at which no adverse toxic effect is observed. The NOAEL is then modified with an uncertainty factor, which reflects the degree of uncertainty that exists when experimental animal data are extrapolated (or applied) to the general human population. The magnitude of the uncertainty factor considers various factors such as sensitive subpopulations (for example; children, pregnant women, and the elderly), extrapolation from animals to humans, and the completeness of available data. Thus, exposure doses at or below the established health guideline are not expected to result in adverse non-cancer health effects.

Uncertainty factors can be a difficult concept to explain, but they are important in understanding health effects and risk. Otherwise, individual persons can become lost in evaluations based on health effects of groups. An analogy may help. For instance, if toxicity were a room, health guidance values could be the "floor". Known health effects would not be expected to occur until one reaches the concentration or dose in the primary study and perhaps higher. In that case, that primary study would represent the

"ceiling". The "height" of the room could represent the uncertainty factors associated with each health guidance value such as an ATSDR MRL or EPA RfD.

When site-specific exposure doses exceed health guidelines, it does not necessarily indicate that health effects will occur. Rather, it indicates that a more thorough look at the known toxicological values for the chemical and the site-related exposures are needed. The known toxicological values are doses derived from human and animal studies that are presented in the ATSDR Toxicological Profiles and EPA's Integrated Risk Information System (IRIS). A direct comparison of site-specific exposure doses to study-derived exposures and doses found to cause adverse health effects is the basis for deciding whether health effects are likely to occur. This in-depth evaluation is performed by comparing calculated exposure doses with known toxicological values, such as the no-observed adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from studies used to derive the MRL or RfD for a chemical.

It is important to consider that the methodology used to develop these health guidelines does not provide any information on the presence, absence, or level of cancer risk. Therefore, a separate cancer evaluation is indicated for potentially cancer-causing chemicals detected in samples at this site.

#### **Cancer Risks**

The estimated excess risk of developing cancer from exposure to chemicals associated with the site was calculated by multiplying the exposure doses by EPA's oral cancer slope factor (CSFs or cancer potency estimates) for the compounds, which is available on the EPA Integrated Risk Information System (IRIS) at <u>http://www.epa.gov/iris/subst/0278.htm</u>. The Cancer Risk is calculated according to the following formula:

$$CR = (D \ X \ CSF) \ X \ EF$$

Where:

CR = Cancer Risk (unitless)
D = Dose calculated as described above
CSF = Cancer Slope Factor (in units that are the reciprocal of the Dose)
EF = Exposure factor (unitless) as described in the Discussion.

Note that cancer risk calculated for exposures occurring during adulthood and childhood are combined and expressed as the risk of an individual developing cancer over his or her lifetime. An increased excess lifetime cancer risk is not a specific estimate of expected cancers. Rather, it is an estimate of the increase in the probability that a person may develop cancer sometime during his or her lifetime following exposure to a particular chemical. Therefore, the cancer risk calculation incorporates the equations and parameters (including the exposure duration and frequency) used to calculate the dose estimates, but the estimated value is divided by 25,550 days (or the averaging time), which is equal to a lifetime of exposure (70 years) for 365 days/year.

There are varying suggestions among the scientific community regarding excess lifetime cancer risk, due to the uncertainties regarding the mechanism of cancer. EPA targets the risk range of 1 in 1 million to 1 in 10,000 (as referred to as  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$ ) excess cancer cases for risk management in the Superfund program. Exposure to a lifetime cancer risk less than 1 in 1,000,000 (or  $1 \times 10^{-6}$ ) is not typically considered a health concern; between  $1 \times 10^{-6}$  and  $1 \times 10^{-4}$  may be a concern under some conditions; and more than  $1 \times 10^{-4}$  is generally considered a health concern except possibly under extraordinary conditions. An important consideration when determining cancer risk estimates is that

the risk calculations incorporate several very conservative assumptions that are expected to overestimate actual exposure scenarios. For example, the method used to calculate EPA's CSFs assumes that high-dose animal data can be used to estimate the risk for low dose exposures in humans. The method also assumes that there is no 'safe level' for exposure. Lastly, the method computes the 95% upper bound for the risk, rather than the average risk, suggesting that the cancer risk is actually lower, perhaps by several orders of magnitude.

Because of the uncertainties involved with estimating cancer risk, ATSDR also employs a qualitative approach in evaluating all relevant data. The actual environmental exposures have been given careful and thorough consideration in evaluating the assumptions and variables relating to both toxicity and exposure. A complete review of the toxicological data regarding the doses associated with the production of cancer and the site-specific doses is an important element in determining the likelihood of exposed individuals being at a greater risk for cancer.

<sup>1</sup> Agency for Toxic Substances and Disease Registry. Public Health Assessment Guidance Manual. Atlanta: US Department of Health and Human Services. January 2005.

U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Part A. December 1989.

U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Part E, Supplemental Guidance for Dermal Exposure. July 2004.

U.S. Environmental Protection Agency. Exposure Factors Handbook. September 2011.

#### **Appendix B: Figures**

NB: When printing this appendix, best results are achieved by producing the documents on 11X17 paper. When viewing it electronically, the zoom function may be useful.





#### Figure 2: FEMA Flood Hazard Zones. Available from https://hazards.fema.gov/wps/portal/mapviewer

Flood hazard areas identified on the Flood Insurance Rate Map are identified as a Special Flood Hazard Area (SFHA). SFHA are defined as the area that will be inundated by the "base flood" event having a 1-percent chance of being equaled or exceeded in any given year. The 1-percent annual chance (or 100 year) flood is shown in the cross hatch area. (This zone in Canton is classified as Zone AE, meaning the extent of floodwaters has been determined or estimated based on elevations.) Moderate flood hazard areas shown in light blue above are the areas between the limits of the base flood and the 0.2-percent-annual-chance (or 500-year) flood or annual flood depths of less than 1' above ground (FEMA NFIP Zone X). The areas of minimal flood hazard, which are the areas outside the SFHA and higher than the elevation of the 0.2-percent-annual-chance flood, are unshaded.



Ref: EPA, 2013. Email from EPA IV RPM to ATSDR IV re: SE Wood Figures Dated 2/26/2013 at 2:02 pm.



Figure 3: Sample locations.

#### **Appendix C: Polycyclic Aromatic Hydrocarbons**

Polycyclic aromatic hydrocarbons (PAHs) are a class of chemicals with similar structure often found together in our environment. In order to be considered a PAH, a chemical must consist only of hydrogen and carbon and must have at least two 6-carbon rings in its structure. PAHs can occur naturally in our environment, but the most common source in most areas is the burning of materials that contain carbon. PAHs are also a significant component of a common wood treating mixture of materials called creosote.

Like Dioxins, PAHs are often found as a mixture. Various methods have been developed to evaluate the potential health effects as a mixture. [22] Probably the best understood of the various PAHs is benzo(a)pyrene or BaP. The current method in the US is to compare the toxicity of individual PAHs to the toxicity of BaP, based on a series of factors developed by EPA in 1993. [23] The procedure is similar to that described above for dioxins. Table 2 contains the toxicity factors for the PAHs detected on site. The toxicity of the compounds relative to BaP is calculated and then compared to the Cancer Risk Evaluation Guide (CREG) for BaP of 96 ug/kg. [18] Then the relative toxicity is then added together for each sample area.

Substance	Toxicity Factor		
Benzo(a)pyrene	1.0		
Benz(a)anthracene	0.1		
Benzo(b)fluoranthene	0.1		
Benzo(k)fluoranthene	0.01		
Chrysene	0.001		
Dibenz(a,h)anthracene	1		
Indeno(1,2,3-cd)pyrene	0.1		

Table 6: PAH relative toxicity

In Table 7 these factors are applied to the samples with the highest concentrations for each of the streets discussed above. See Figure 3 in Appendix B. Below the table can be found a copy of the ATSDR ToxFAQs, available at www.atsdr.cdc.gov.

Street/	Substance	Concentration	TF	Е
Sample No.		(ug/kg)		(ug/kg)
Barfield/	Benzo(a)pyrene	3800	1.0	3800
SWP-235 0.5				
Location is	Benz(a)anthracene	4500	0.1	450
shown as		10.000	0.1	1000
green box with red fill on	Benzo(b)fluoranthene	10,000	0.1	1000
Figure 3	Benzo(k)fluoranthene	6300	0.01	63
	Chrysene	8200	0.001	8.2
	Indeno(1,2,3-cd)pyrene	1700	0.1	170
	B(a)P Equivalent Concentration			5491
Covington/	Benzo(a)pyrene	1800	1.0	1800
SWP-230 0.5 *				
Location is shown	Benz(a)anthracene	1800	0.1	180
as				
green box with	Benzo(b)fluoranthene	4800	0.1	480
Figure 3	Benzo(k)fluoranthene	3500	0.01	35
	Chrysene	3700	0.001	3.7
	Dibenz(a,h)anthracene	250	1	250
	Indeno(1.2.3-cd)pyrene	850	0.1	85
	B(a)P Equivalent Concentration			2834
Miller/	Benzo(a)pyrene	180	1.0	180
SWP-232 0.5				
Location is shown	Benz(a)anthracene	170	0.1	17
as				
green box with	Benzo(b)fluoranthene	200	0.1	20
Figure 3	Benzo(k)fluoranthene	160	0.01	1.6
	Chrysene	200	0.001	0.2
	Indeno(1,2,3-cd)pyrene	81	0.1	8.1
	B(a)P Equivalent Concentration			227

#### Table 7: Benzo(a)pyrene equivalency (E)

\* - This sample location is a commercial property, not a residential property.

The lifetime  $10^{-6}$  cancer risk for B(a)P is 96 ug/kg, commonly rounded to 100 ug/kg. That is, at this concentration in soil, one extra cancer case would be expected in a population of 1 million humans exposed to the soil over a 70 year lifetime.



## POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

#### Agency for Toxic Substances and Disease Registry ToxFAQs

#### September 1996

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

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

What are polycyclic aromatic hydrocarbons?

(Pronounced pŏl/ï-sī/klĭk ăr'ə-măt/ïk hī/drəkar/bənz)

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

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

# What happens to PAHs when they enter the environment?

- PAHs enter the air mostly as releases from volcanoes, forest fires, burning coal, and automobile exhaust.
- PAHs can occur in air attached to dust particles.
- Some PAH particles can readily evaporate into the air from soil or surface waters.
- PAHs can break down by reacting with sunlight and other chemicals in the air, over a period of days to weeks.

- PAHs enter water through discharges from industrial and wastewater treatment plants.
- Most PAHs do not dissolve easily in water. They stick to solid particles and settle to the bottoms of lakes or rivers.
- Microorganisms can break down PAHs in soil or water after a period of weeks to months.
- In soils, PAHs are most likely to stick tightly to particles; certain PAHs move through soil to contaminate underground water.
- PAH contents of plants and animals may be much higher than PAH contents of soil or water in which they live.

#### How might I be exposed to PAHs?

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

#### U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, Public Health Service Agency for Toxic Substances and Disease Registry

#### Page 2

#### POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

#### ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html

 Nursing infants of mothers living near hazardous waste sites may be exposed to PAHs through their mother's milk.

#### How can PAHs affect my health?

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

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

#### How likely are PAHs to cause cancer?

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

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

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

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

#### Has the federal government made recommendations to protect human health?

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

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

#### Glossary

Carcinogen: A substance that can cause cancer.

Ingest: Take food or drink into your body.

#### References

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

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

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Federal Recycling Program

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#### **Appendix D:** Polychlorinated DibenzoDioxins

The term dioxin refers to two oxygen atoms found in the formulas of the chemicals associated with this class. There are many chemicals with two oxygen atoms in their formula, but the ones generally referred to when people think of this term also have two carbons rings and many chlorine atoms. Also generally included in the term are chemicals called furans, which have the same chemical formula as their partner dioxins except that they usually have only one oxygen atom. The actual chemical names of the substances included in this class of compounds typically start with a series of numbers that indicate where the chlorine atoms are located in the structure. Sometimes there may be a letter that indicates where the oxygen atoms are located. Of the 75 substances with this pattern of formula and structure, about 25 chemicals are of potential concern to human health.

Figure 5: Dioxin Structure



The figure above shows the molecular structure of a dioxin. [24] The most widely known and most widely studied chemical in this class is 2,3,7,8-tetrachlorodibenzo-p-dioxin. This chemical has two carbon rings ("dibenzo") indicated by the letter "C", two oxygen atoms ("dioxin") indicated by the letter "O", and 4 chlorine ions ("tetrachloro") indicated by the letters "Cl",. There are hydrogen atoms, indicated by the letter "H", attached to the carbon rings wherever a chlorine or oxygen atom is not indicated. Moreover, the chlorine atoms are attached to the molecule in the 2,3 and 7,8 positions and the oxygen atoms are in the "p" or "para" orientation (opposite each other).

Because it is usual to find many different cogeners in the same sample, dioxins are typically evaluated as a mixture. Since we have more information on 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), all of the other individual dioxin compounds are assigned a toxicity equivalency factor, or TEF, that relates to how that compound compares to TCDD. The concentration of the chemical reported in the sample is multiplied by the TEF for that chemical. Then the results of

that multiplication for each dioxin in a given sample are added together. This sum is the TCDD Toxicity Equivalent or TCDD TEQ for that sample. As we learn more about relative toxicity of these chemicals, the TEFs can change over time. The most current TEFs were developed by the World Health Organization in 2009 and are provided in table 1 below. [25]

Congener	TEF <sup>a</sup>	Half-life <sup>b</sup> (years)
1,2,3,7,8-PeCDD	1	11.2
1,2,3,4,7,8-HxCDD	0.1	9.8
1,2,3,6,7,8-HxCDD	0.1	13.1
1,2,3,7,8,9-HxCDD	0.1	5.1
1,2,3,4,6,7,8-HpCDD	0.01	4.9
OCDD	0.0003	6.7
2,3,7,8-TCDF	0.1	2.1
1,2,3,7,8-PeCDF	0.03	3.5
2,3,4,7,8-PeCDF	0.3	7.0
1,2,3,4,7,8-HxCDF	0.1	6.4
1,2,3,6,7,8-HxCDF	0.1	7.2
1,2,3,7,8,9-HxCDF	0.1	7.2
2,3,4,6,7,8-HxCDF	0.1	2.8
1,2,3,4,6,7,8-HpCDF	0.01	3.1
1,2,3,4,7,8,9-HpCDF	0.01	4.6
OCDF	0.0003	1.4
PCB 77	0.0001	0.1
PCB 81	0.0003	0.7
PCB 126	0.1	1.6
PCB 169	0.03	7.3
PCB 105	0.00003	2.4
PCB 114	0.00003	10.0
PCB 118	0.00003	3.8
PCB 123	0.00003	7.4
PCB 156	0.00003	16.0
PCB 157	0.00003	18.0
PCB 167	0.00003	12.0
PCB 189	0.00003	22.0

Table 8: TEF and Cogener half-lives in humans
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<sup>a</sup> Toxicity equivalence factors from <u>Van den Berg et al. (2006)</u>

Half-life of congeners in humans based on <u>Milbrath et al. (2009)</u>	
CDD =ChlorinatedDibenzoDioxins	CDF = ChlorinatedDibenzoFurans
Pe=Penta or five chlorines	Hx=Hexa or six
Hp=Hepta or seven	O=Octo or eight

PCB = PolyChlorinated Biphenyl [not found at this site.]

In Table 9, these TEFs are applied to the samples with the highest concentrations for the residential community. See Figure 3 in Appendix B. Below the table can be found a copy of the ATSDR ToxFAQs available at www.atsdr.cdc.gov.

Street/	Substance	Concentration	TEF	TEQ
Sample No.		(ng/kg)		(ng/kg)
Barfield/	1,2,3,7,8-Pentachlorodibenzodioxin	35	1	35
SWP-235 0.5				
Location is	Octachlorodibenzodioxin	310000	0.0003	93
shown as				
green box with	1,2,3,4,7,8,9-Heptachlorodibenzofuran	1000	0.01	10
red fill on			0.000	• • •
Figure 3	Octachlorodibenzofuran	98000	0.0003	29.4
	1,2,3,4,7,8-Hexachlorodibenzodioxin	120	0.1	12
	1,2,3,7,8,9-Hexachlorodibenzodioxin	270	0.1	27
	1,2,3,7,8,9-Hexachlorodibenzofuran	51	0.1	5.1
	1,2,3,4,6,7,8-Heptachlorodibenzodioxin	31000	0.01	310
	1,2,3,6,7,8-Hexachlorodibenzodioxin	1200	0.1	120
	2,3,4,6,7,8-Hexachlorodibenzofuran	280	0.1	28
	1,2,3,6,7,8-Hexachlorodibenzofuran	130	0.1	13
	1,2,3,4,7,8-Hexachlorodibenzofuran	300	0.1	30
	1,2,3,4,6,7,8-Heptachlorodibenzofuran	30,000	0.01	300
	Dioxin TEQ for SWP-235 0.5			1013

#### Table 9: Calculation of Dioxin TEQ

The chronic MRL for TCDD results in a comparison value of 50 ng/kg for a child ingesting 200 mg of soil per day.

## CHLORINATED DIBENZO-p-DIOXINS (CDDs)

#### Division of Toxicology and Environmental Medicine ToxFAQs<sup>™</sup>

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

HIGHLIGHTS: Exposure to chlorinated dibenzo-p-dioxins (CDDs) (75 chemicals) occurs mainly from eating food that contains the chemicals. One chemical in this group, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, has been shown to be very toxic in animal studies. It causes effects on the skin and may cause cancer in people. This chemical has been found in at least 91 of the 1,467 National Priorities List sites identified by the Environmental Protection Agency (EPA).

#### What are CDDs?

CDDs are a family of 75 chemically related compounds commonly known as chlorinated dioxins. One of these compounds is called 2,3,7,8-TCDD. It is one of the most toxic of the CDDs and is the one most studied.

In the pure form, CDDs are crystals or colorless solids. CDDs enter the environment as mixtures containing a number of individual components. 2,3,7,8-TCDD is odorless and the odors of the other CDDs are not known.

CDDs are not intentionally manufactured by industry except for research purposes. They (mainly 2,3,7,8-TCDD) may be formed during the chlorine bleaching process at pulp and paper mills. CDDs are also formed during chlorination by waste and drinking water treatment plants. They can occur as contaminants in the manufacture of certain organic chemicals. CDDs are released into the air in emissions from municipal solid waste and industrial incinerators.

# What happens to CDDs when they enter the environment?

When released into the air, some CDDs may be transported long distances, even around the globe.
 When released in waste waters, some CDDs are broken down by sunlight, some evaporate to air, but most attach to soil and settle to the bottom sediment in water.

CDD concentrations may build up in the food chain, resulting in measurable levels in animals. How might I be exposed to CDDs?

Eating food, primarily meat, dairy products, and fish, makes up more than 90% of the intake of CDDs for the general population.

Breathing low levels in air and drinking low levels in water.

Skin contact with certain pesticides and herbicides.

Living near an uncontrolled hazardous waste site containing CDDs or incinerators releasing CDDs.

Working in industries involved in producing certain pesticides containing CDDs as impurities, working at paper and pulp mills, or operating incinerators.

#### How can CDDs affect my health?

The most noted health effect in people exposed to large amounts of 2,3,7,8-TCDD is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other skin effects noted in people exposed to high doses of 2,3,7,8-TCDD include skin rashes, discoloration, and excessive body hair. Changes in blood and urine that may indicate liver damage also are seen in people. Exposure to high concentrations of CDDs may induce longterm alterations in glucose metabolism and subtle changes in hormonal levels.

In certain animal species, 2,3,7,8-TCDD is especially harmful and can cause death after a single exposure. Exposure to lower levels can cause a variety of effects in

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#### ToxFAQs<sup>™</sup> Internet address is http://www.atsdr.cdc.gov/toxfaq.html

animals, such as weight loss, liver damage, and disruption of the endocrine system. In many species of animals, 2,3,7,8-TCDD weakens the immune system and causes a decrease in the system's ability to fight bacteria and viruses. In other animal studies, exposure to 2,3,7,8-TCDD has caused reproductive damage and birth defects. Some animal species exposed to CDDs during pregnancy had miscarriages and the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy often had severe birth defects including skeletal deformities, kidney defects, and weakened immune responses.

#### How likely are CDDs to cause cancer?

Several studies suggest that exposure to 2,3,7,8-TCDD increases the risk of several types of cancer in people. Animal studies have also shown an increased risk of cancer from exposure to 2,3,7,8-TCDD. The World Health Organization (WHO) has determined that 2,3,7,8-TCDD is a human carcinogen. The Department of Health and Human Services (DHHS) has determined that 2,3,7,8-TCDD may reasonably be anticipated to cause cancer.

#### How can CDDs affect children?

Very few studies have looked at the effects of CDDs on children. Chloracne has been seen in children exposed to high levels of CDDs. We don't know if CDDs affect the ability of people to have children or if it causes birth defects, but given the effects observed in animal studies, this cannot be ruled out.

#### How can families reduce the risk of exposure to CDDs?

Children should avoid playing in soils near uncontrolled hazardous waste sites.

Discourage children from eating dirt or putting toys or other objects in their mouths.  Everyone should wash hands frequently if playing or working near uncontrolled hazardous waste sites.
 For new mothers and young children, restrict eating

foods from the proximity of uncontrolled sites with known CDDs.

Children and adults should eat a balanced diet preferably containing low to moderate amounts of animal fats including meat and dairy products, and fish that contain lower amounts of CDDs and eat larger amounts of fruits, vegetables, and grains.

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

Tests are available to measure CDD levels in body fat, blood, and breast milk, but these tests are not routinely available. Most people have low levels of CDDs in their body fat and blood, and levels considerably above these levels indicate past exposure to above-normal levels of 2,3,7,8-TCDD. Although CDDs stay in body fat for a long time, tests cannot be used to determine when exposure occurred.

#### Has the federal government made recommendations to protect human health?

The EPA has set a limit of 0.00003 micrograms of 2,3,7,8-TCDD per liter of drinking water (0.00003  $\mu$ g/L). Discharges, spills, or accidental releases of 1 pound or more of 2,3,7,8-TCDD must be reported to EPA. The Food and Drug Administration (FDA) recommends against eating fish and shellfish with levels of 2,3,7,8-TCDD greater than 50 parts per trillion (50 ppt).

#### References

Agency for Toxic Substances and Disease Registry (ATSDR). 1998. Toxicological Profile for Chlorinated Dibenzo-p-Dioxins. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-62, Atlanta, GA 30333. Phone: 1-800-232-4636, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Federal Recycling Program



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#### **Appendix E: Cancer Classifications**

#### Known and Probable Human Carcinogens American Cancer Society

#### Introduction

Many people worry that substances or exposures in their environment may cause cancer. As part of the American Cancer Society's role in informing and educating people about cancer and its possible causes, this document provides lists of substances and exposures that are known or suspected to cause cancer. The lists below have been developed by two highly respected agencies – the International Agency for Research on Cancer (IARC) and the US National Toxicology Program (NTP). Some related information is included on how these and other agencies and groups test and classify possible carcinogens. The American Cancer Society does not keep detailed information on each of the exposures on these lists. If you are looking for more in-depth information on a particular item on these lists, please refer to the agencies in the "Additional resources" section of this document.

#### What is a carcinogen?

Cancer is caused by changes in a cell's DNA – its genetic "blueprint." Some of these changes may be inherited from our parents, while others may be caused by outside exposures, which are often referred to as *environmental factors*. Environmental factors can include a wide range of exposures, such as:

- \* Lifestyle factors (nutrition, tobacco use, physical activity, etc.)
- \* Naturally occurring exposures (ultraviolet light, radon gas, infectious agents, etc.)
- \* Medical treatments (chemotherapy, radiation, immune system-suppressing drugs, etc.)
- \* Workplace exposures
- \* Household exposures
- \* Pollution

\*

Substances and exposures that can lead to cancer are called *carcinogens*. Some carcinogens do not affect DNA directly, but lead to cancer in other ways. For example, they may cause cells to divide at a faster than normal rate, which could increase the chances that DNA changes will occur.

Carcinogens do not cause cancer in every case, all the time. Substances labeled as carcinogens may have different levels of cancer-causing potential. Some may cause cancer only after prolonged, high levels of exposure. And for any particular person, the risk of developing cancer depends on many factors, including how they are exposed to a carcinogen, the length and intensity of the exposure, and the person's genetic makeup.

#### How do researchers determine if something is a carcinogen?

Testing to see if something can cause cancer is often difficult. It is not ethical to test a substance by exposing people to it and seeing if they get cancer from it. That's why scientists must use other types of tests, which may not always give clear answers.

#### Lab studies

Scientists get much of their data about whether something might cause cancer from lab studies in cell cultures and animals. There are far too many substances (both natural and man-made) to test each one in lab animals, so scientists use what is already known about chemical structures, results from other types of lab tests, the extent of human exposure, and other factors to select chemicals for testing. For example, they can often get an idea about whether a substance might cause a problem by comparing it to similar chemicals that have already been studied.

Although lab studies alone can't always predict if a substance will cause cancer in people, virtually all known human carcinogens that have been adequately tested also cause cancer in lab animals. In many cases, carcinogens are first found to cause cancer in lab animals and are later found to cause cancer in people.

Most studies of potential carcinogens expose the lab animals to doses that are much higher than common human exposures. This is so that cancer risk can be detected in relatively small groups of animals. It is not always clear if the results from animal studies will be the same for people as they are normally exposed to a substance. For example, the effects seen in lab studies with very high doses of a substance may not be the same at much lower doses, or the effects of a substance when it is inhaled may not be the same as if it is applied to the skin. Also, the bodies of lab animals and humans don't always process substances in the same way.

But for safety reasons, it is usually assumed that exposures that cause cancer at larger doses in animals may also cause cancer in people. It isn't always possible to know how the exposure dose might affect risk, but it is reasonable for public health purposes to assume that lowering human exposure will reduce risk.

#### **Studies in people**

Another important way to identify carcinogens is through epidemiologic studies, which look at human populations to determine which factors might be linked to cancer. These studies also provide useful information, but they have their limits. Humans do not live in a controlled environment. People are exposed to many substances at any given time, including those they encounter at work, school, or home; in the food they eat; and in the air they breathe. It's very unlikely they know exactly what they've been exposed to or that they would be able to remember all of their exposures if asked by a researcher. And there are usually many years (often decades) between exposure to a carcinogen and the development of cancer. Therefore, it can be very hard to definitely link any particular exposure to cancer.

By combining data from both types of studies, scientists do their best to make an educated assessment of a substance's cancer-causing ability. When the evidence is conclusive, the substance is labeled as a carcinogen. When the available evidence is compelling but not felt to be conclusive, the substance may be considered to be a probable carcinogen. But in some cases there simply isn't enough information to be certain one way or the other.

#### Who determines how carcinogens are classified?

Several agencies (national and international) are responsible for determining the cancer-causing potential of different substances.

#### **International Agency for Research on Cancer**

The International Agency for Research on Cancer (IARC) is part of the World Health Organization (WHO). Its major goal is to identify causes of cancer. The most widely used system for classifying carcinogens comes from the IARC. In the past 30 years, the IARC has evaluated the cancer-causing potential of more than 900 likely candidates, placing them into one of the following groups:

Group 1: Carcinogenic to humans Group 2A: Probably carcinogenic to humans Group 2B: Possibly carcinogenic to humans Group 3: Unclassifiable as to carcinogenicity in humans Group 4: Probably not carcinogenic to humans

Perhaps not surprisingly, based on how hard it can be to test these candidate carcinogens, most are listed as being of probable, possible, or unknown risk. Only a little over 100 are classified as "carcinogenic to humans."

#### **National Toxicology Program**

The National Toxicology Program (NTP) is formed from parts of several different US government agencies, including the National Institutes of Health (NIH), the Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA). The NTP updates its *Report on Carcinogens* (RoC) every few years.

The Report on Carcinogens identifies 2 groups of agents:

"Known to be human carcinogens" "Reasonably anticipated to be human carcinogens"

The current version of the RoC lists about 240 substances and exposures. Unlike the IARC's list, the RoC does not list substances that have been studied and found not to be carcinogens.

#### **Environmental Protection Agency**

The US Environmental Protection Agency (EPA) maintains the Integrated Risk Information System (IRIS), an electronic database that contains information on human health effects from exposure to certain substances in the environment. The EPA uses a rating system similar to that of IARC when describing the cancer-causing potential of a substance:

Group A: Carcinogenic to humans Group B: Likely to be carcinogenic to humans Group C: Suggestive evidence of carcinogenic potential Group D: Inadequate information to assess carcinogenic potential Group E: Not likely to be carcinogenic to humans

#### Other agencies and groups

Other federal agencies, such as the CDC's National Institute for Occupational Safety and Health (NIOSH), the Food and Drug Administration (FDA), and the National Cancer Institute may comment on whether a substance or exposure may cause cancer and/or what levels of exposure to a particular substance might be considered acceptable.

Some state agencies also keep lists of known or probable carcinogens. For example, the California Environmental Protection Agency (CalEPA) maintains a list of "chemicals known to the state to cause cancer or reproductive toxicity." (Much of this list is based on the IARC and NTP lists below.)

#### American Cancer Society, 2012. Available at:

http://www.cancer.org/cancer/cancercauses/othercarcinogens/generalinformationaboutcarcinogens/know n-and-probable-human-carcinogens

#### National Cancer Institute Data **National Institutes of Health**

According to data available from the National Cancer Institute, the incidence rate for all cancers in the US is approximately 47 new diagnosis of cancer per group of 10,000 Americans per year. The prevalence of cancer in the US population is approximately 30-40%. Prevalence means that, out of any given group of 10,000 Americans, somewhere between 3000 and 4000 either now has or has had some form of cancer in their lifetime.

Also, according to the National Cancer Institute, cancer incidence data for Madison County, MS, for the types of cancer associated with the chemicals with this site compare as follows [20]:

Age Adjusted Number of cases diagnosed per 100,000 people			
Cancer	USA	Mississippi	Madison County
All Cancers	453.7	475.2	473.4
Lung *	65.0	80.1	69.6
Breast *	119.8	113.8	126.9
Non-Hodgkins	18.9	17.7	18.2
Lympnoma	1.42.0	166.2	1/77
Prostate	143.8	166.3	16/./
Stomach	6.6	7.0	6.9
Liver	6.7	6.2	4.3
Colon/Rectal	43.9	52.0	48.1

Table 10 **Cancer Incidence Rates** 

- Cancers associated with exposure to either dioxin or PAHs; otherwise cancers associated with only one chemical.

#### Table 11 **Cancer Mortality Rates** Age Adjusted Number of cases diagnosed per 100,000 people

Cancer	USA	Mississippi	Madison County
All Cancers	176.4	203.5	372.7 <sup>1</sup>
Lung *	49.5	64.3	118.9 <sup>1</sup>
Breast *	27.6	24.7	42.6
Non-Hodgkins	6.4	6.2	11.6 <sup>1</sup>
Lymphoma			
Prostate	23.0	31.2	73.2
Stomach	3.5	4.1	8.2
Liver	5.6	6.7	11.8 <sup>-1</sup>
Colon/Rectal	16.4	20.1	34.4

- Cancers associated with exposure to either dioxin or PAHs; otherwise cancers associated with only one chemical.

<sup>1</sup> – Highest rate in the state of Mississippi.

Table 12: Estimated Doses of Chemical without Health Guidance Values in residential soils					
Substance	Sample Number	Infant Dose (ug/kg/day)	Child Dose (ug/kg/day)	Adult Dose (ug/kg/day)	
Acenaphthylene	SWP-235 0.5	0.003	0.002	0.0002	
Carbazole	SWP-235 0.5	0.015	0.009	0.0009	
Dibenzofuran	SWP-244 0.5	0.003	0.002	0.0002	
Phenanthrene	SWP-235 0.5	0.018	0.011	0.001	
Benzo(g,h,i)perylene	SWP-244 0.5	0.04	0.025	0.003	

Appendix F: Toxicological Evaluation of Substances with no Health Comparison Values.

ug/kg/day=microgram of chemical per kilogram bodyweight per day

<u>Acenaphthylene</u>: Acenaphthylene is a non-carcinogenic PAH with 2 carbon rings. [26] Like most PAHs, little data can be found in the literature about the toxicity of this individual compound; as mentioned previously, most studies look at combinations of PAHs. In the absence of other data, pyrene – a PAH with 4 carbon rings - has a health guidance value associated with it and is chemically similar to benzo(g,h,i)perylene. [27] EPA has established an RfD for pyrene of 30 ug/kg/day. [28] The estimate child's dose in Table 7 is 0.002 ug/kg/day. Adverse health effects would not be expected from exposure to this compound.

<u>Benzo(g,h,i)perylene</u>: Benzo(g,h,i)perylene is a non-carcinogenic PAH with 6 carbon rings. [29] Like most PAHs, little data can be found in the literature about the toxicity of this individual compound; as mentioned previously, most studies look at combinations of PAHs. Benzo(g,h,i)perylene is a large molecule and absorption by humans at the cellular level is probably difficult. However, Cherng and others (2001) reported that benzo(g,h,i)perylene seems to enhance the effects of benzo(a)pyrene. [30] In other words, while benzo(g,h,i)perylene itself may not cause health effects, but its presence seems to increase the hazard of some other compounds (like benzo(a)pyrene). In the absence of other data, pyrene – a PAH with 4 carbon rings - seems to be the non-carcinogenic PAH that has a health guidance value associated with it and is most chemically similar to benzo(g,h,i)perylene. [30] EPA has established an RfD for pyrene of 30 ug/kg/day. [27] The estimated dose benzo(g,h,i)perylene for a child at this site is 0.025 ug/kg/day (see Table 7). Except by enhancing the health effects with co-located benzo(a)pyrene, benzo(g,h,i)perylene would not be expected to cause any adverse health effects.

<u>Carbazole:</u> Carbazole (Chemical Abstract number 86-74-8) is a PAH commonly found in tobacco smoke. This chemical is a primary intermediate in the production of blue dyes. [31] There are few data on humans exposed to Carbazole. A 1982 study by the National Cancer Institute found that mice fed a diet of 0.6%, 0.3%, and 0.15% carbazole had increased cancers in the liver and forestomachs compared to controls in all animals except males at the highest dose (0.6%). Because the increased cancer incidence (e.g., occurrence) was not seen at the highest dose in male rats, a dose response relationship is not likely. Dose response means that, as more of the chemical is

absorbed, more effects are seen. The absence of such a relationship implies that the cancers may not have been due to carbazole. In 1997, the International Agency for the Registry of Carcinogens (IARC) classified carbazole as a Group 3 carcinogen (i.e., inadequate evidence of carcinogenicity). [32] In 1995, Dutson and others evaluated the developmental toxicology of carbazole and a related compound in animals after the compounds were applied to the skin. No effects were seen at doses equal to 2.5 mg/kg/day, 25 mg/kg/day, or 250 mg/kg/day. [33] The lowest dose in animals that resulted in no adverse health effects (NOAEL) was equal to 2.5 mg/kg/day or 2500 ug/kg/day. The maximum estimated dose of carbazole for a child at this site is equivalent to 0.009 ug/kg/day (Table 7); it is unlikely that this compound poses a threat.

<u>Dibenzofuran:</u> Despite the name, dibenzofuran seems to be toxicologically closer in behavior to a 3 ring non-carcinogenic PAH than a furan. Most of the studies of this chemical include other PAHs or furans and identifying the contribution of this chemical to the effects of that mixture is difficult. There is a study in animals that evaluated the toxicity of 3 ring PAHs and included dibenzofuran. That study indicated that the lowest effect level of the mixture was a dose of 37 mg/kg or 37,000 ug/kg in animals. While this dose was also the lowest dose used in the study, the effects at that dose were much more minor than at the higher doses. [34] The estimated dose for a child for this compound at this site is 0.002 ug/kg/day or roughly 7 orders of magnitude (factors of ten) below the dose in that study. It seems unlikely that any adverse effects would be expected.

<u>Phenanthrene:</u> Phenanthrene is a 3 ring PAH. [35] Because of its frequency of detection, various studies have looked at aspects of phenanthrene metabolism. However, like many PAHs, there are few studies looking at the toxic effects of phenanthrene in humans or animals other than various species of fish. Fish ecological toxicity is generally not a good model for human toxicity. Pyrene is chemically similar to phenanthrene and they are often found together in the environment. Using the EPA RfD of 30 ug/kg/day for pyrene as a comparison, [28] the estimated dose of 0.011 ug/kg/day for a child at this site would not be expected to produce any adverse health effects.

Appendix G: ATSDR Fact Sheet - Precautions to reduce exposure from soil

# Ways to protect your health By keeping dirt from getting into your house and into your body

# Wash and peel all fruits, vegetables, and root crops



Wipe shoes on doormat or remove shoes



Don't eat food, chew gum, or smoke when working in the yard



Damp mop floors and damp dust counters and furniture regularly



Wash dogs regularly



Wash children's toys regularly



Wash children's hands and feet after they have been playing outside

ATSDR, 2003. Ways to Protect your Health by keeping dirt from getting into your house and into your body. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Atlanta, GA. [available as Appendix I to the Vasquez Boulevard-I70 Public Health Assessment dated August 12, 2003 at http://www.atsdr.cdc.gov/HAC/PHA/reports/vasquezblvd\_08122003co/images/appi.pdf