

Health Consultation

HEALTH IMPLICATIONS OF TEXARKANA TIRE FIRE

TEXARKANA, MILLER COUNTY, ARKANSAS

OCTOBER 26, 2004

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service

Agency for Toxic Substances and Disease Registry

Division of Health Assessment and Consultation

Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. 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 which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You May Contact ATSDR TOLL FREE at
1-888-42ATSDR

or

Visit our Home Page at: <http://www.atsdr.cdc.gov>

HEALTH CONSULTATION

HEALTH IMPLICATIONS OF TEXARKANA TIRE FIRE

TEXARKANA, MILLER COUNTY, ARKANSAS

Prepared by:

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

(left blank)

Table of Contents

SUMMARY AND STATEMENT OF ISSUES	1
BACKGROUND	1
Site Description and History.....	1
Demographics	2
Exposure Pathway Analysis.....	3
DISCUSSION	3
Polycyclic Aromatic Hydrocarbons (PAHs).....	4
Exposure Pathways	4
Cancer Risk Analysis.....	6
COMMUNITY HEALTH CONCERNS	7
CHILD HEALTH CONSIDERATIONS.....	7
SITE UPDATE	7
CONCLUSIONS.....	7
RECOMMENDATIONS.....	7
PUBLIC HEALTH ACTION PLAN.....	8
Completed Actions	8
Future Activities	8
AUTHORS, TECHNICAL ADVISORS	9
CERTIFICATION	10
REFERENCES	11
APPENDICES	13
Appendix A – Figure 1	14
Appendix B – Tables	16

(left blank)

SUMMARY AND STATEMENT OF ISSUES

This Health Consultation has been prepared in response to a request made by the Arkansas Department of Environmental Quality (ADEQ) for assistance in determining the potential health risks associated with a tire fire in Texarkana, Arkansas. Specifically, this document reviews the soil sampling data that was collected off site to evaluate the potential for exposure of area residents to polycyclic aromatic hydrocarbons (PAHs) released to soils during the Texarkana Tire Fire. The Arkansas Department of Health (ADH) prepared this Health Consultation under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR).

In February 2003 – approximately one month following the fire – three surface soil samples (taken at depths from 0-2 inches) were collected within ½ mile of the tire fire site. See Appendix A, Figure 1, for site photo and sample locations. Soil samples were analyzed for PAHs. PAH levels were then compared to health based comparison values to determine if health risks associated with the tire fire exist.

To properly assess the public health implications posed by contamination at a site, it is necessary to evaluate site data and information on the site's history, the types and levels of contamination at the site, site-specific exposure pathways, community health concerns, and available toxicological implications of the site's contaminants.

Based on the review of available soil data, ADH categorized the levels of PAHs to represent *No Apparent Public Health Hazard*. The limited environmental sampling data do not indicate that humans are being or have been exposed to levels of PAH contamination in the soil that would be expected to cause adverse health effects.

BACKGROUND

Site Description and History

The site of the Texarkana Tire Fire – herein after referred to as the Tire Fire – is located 5 miles east of the Arkansas Highway 245 and U.S. Highway 82 intersection near Texarkana. See Appendix A for aerial site photo. The site is located in a rural residential area. The area is sparsely populated with approximately 15 homes located within 1/4 mile northeast of the site. The nearest building is the Shiloh Church located approximately 1/5 mile northwest of the site.

The site had unrestricted access and held an estimated 200 tires and 3,600 cubic yards of tank tracks prior to the fire. The tank tracks contain rubber pads and bushings that must be removed before the metal tracks can be sold as salvage material. Because this is labor intensive it is easier to set the tracks on fire and burn the rubber off. However, this method is illegal.

On January 20, 2003, at approximately 3 o'clock in the morning, a fire was reported to the Miller County Sheriff's Office. The tires and tank tracks were ablaze when the Genoa Volunteer Fire Department arrived. The fire was advanced and no action was taken to put out the flames. The fire lasted approximately 2 ½ hours. The burning of the rubber created a plume of smoke that was carried in a north-northeasterly (NNE) direction at 11.5 miles per hour. Particulate fall-out

was visible on the property of residents who lived NNE of the fire. Residents expressed general health concerns relating to the inhalation of smoke and the particulate fall-out left on their property. Residents also alleged that the fire was set on purpose.

On January 29, 2003, ADEQ notified ADH of the Tire Fire. ADEQ also asked ADH to contact the residents regarding their health concerns and to assist in determining possible health risks. On February 28, 2003, three off-site surface soil samples (taken at depths from 0-2 inches) were collected in the vicinity of the Tire Fire in Miller County and analyzed by American Interplex Corporation Laboratories (see Appendix B). The three sampling sites were selected based on the prevailing wind direction at the time of the fire, visible evidence of particulate fall-out originating from the fire, and resident concerns regarding the selected areas.

Quality assurance, instrumentation maintenance and calibration were performed in accordance with guidelines established by the U.S. EPA standard methods of examination for soil sample collection. A limitation of the test equipment was its lack of detection capabilities for PAHs below 0.66 milligram per kilogram (mg/kg). Table 1, below, lists the PAHs tested that have U.S. EPA Risk-Based Concentrations below the instruments capability.

Table 1. Polycyclic Aromatic Hydrocarbons (PAHs) Below Instruments Capability			
PAHs	Sampling Results *(mg/kg)	† Risk-Based Concentrations for Residential Soil (mg/kg)	‡ Oral Cancer Slope Factors (mg/kg/day)
Benz[a]anthracene	ND (0.66)	0.62	0.73
Benzo[a]pyrene	ND (0.66)	0.062	7.3
Benzo[b]fluoranthene	ND (0.66)	0.62	0.73
Dibenz[a,h]anthracene	ND (0.66)	0.062	7.3
Indeno[1,2,3-cd]pyrene	ND (0.66)	0.62	0.73
*mg/kg = milligram per kilogram † RBC = US EPA Region 3 Risk-Based Concentration values are chemical concentrations corresponding to fixed levels of risk. ‡ Cancer slope factors determine the potential risk levels associated with exposure to carcinogens (mg/kg/day = milligram per kilogram per day). 0.66 = detection limit			

Demographics

ADH estimated that 30 people in 15 homes are within approximately 1/4 mile northeast of the Tire Fire site. The 2000 Census (US Census Bureau) reports 40,443 people live in Miller County.

Exposure Pathway Analysis

Potential exposure pathways to contaminants at the Tire Fire site were evaluated to determine if persons could be exposed to potentially unsafe contaminants from the site. Exposure pathways consist of five elements:

1. A source of contamination,
2. Transport through an environmental medium, such as soil or groundwater,
3. Point of exposure,
4. A route for the contaminant to enter the body, and
5. A receptor population.

For a person to be exposed to a contaminant, the exposure pathway must contain all the elements listed above, resulting in a completed exposure pathway. In some cases, a potential exposure pathway might exist in which at least one of the elements of the exposure pathway is missing, but could exist. Potential pathways indicate that exposure to a contaminant could have occurred, could be occurring, or could occur in the future. Potential exposure pathways refer to those pathways where (1) there is not enough information available to determine whether the environmental medium is contaminated, or (2) an environmental medium has been documented as contaminated, but it is unknown whether people have been, or may be, exposed to the medium, or may be exposed in the future.

DISCUSSION

People can be exposed to a site contaminant only if they come in contact with it. This section discusses the health effects that could result from exposure to contaminants at the Tire Fire site. When people are exposed to chemicals, the exposure does not always result in adverse health effects. In order to understand the health effects that might be caused by a specific chemical, many factors affecting how the human body responds to exposure need to be considered. These factors include the exposure concentration, the duration of exposure, route of exposure, and overall health status. Together, these factors determine the dose, or amount, of chemical contaminants a person is potentially exposed to, and what health effects, if any, might occur.

When tires burn three groups of emissions result:

- Airborne and smoke emissions, which include volatile organic compounds, semi-volatile compounds such as polycyclic aromatic hydrocarbons (PAHs), carbon monoxide, and particulate matter which may include metals;
- Ash, which may also include metals such as lead, arsenic and zinc; and,
- Pyrolysis oils such as naphthalene, anthracene, benzene, thiazoles, amines, ethyl benzene, toluene, and other hydrocarbons.

No samples were collected during the fire. Three off-site surface soil samples were collected after the fire was extinguished and were tested for the presence of PAHs (Appendix B, Table 2).

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, and other organic substances like tobacco and charbroiled meat [1]. PAHs are usually found in soot as a mixture containing two or more of these compounds. Some PAHs are manufactured. 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.

PAHs enter the air mostly from volcanoes, forest fires, burning coal, and automobile exhaust. PAHs can occur in air attached to dust particles. Some PAH particles 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.

Most PAHs do not dissolve easily in water. They stick to solid particles and settle to the bottoms of lakes or rivers. Generally, PAHs are not very soluble in water and are strongly bound to soil, so their migration is limited. However, a few PAHs are water-soluble and may migrate through soil and groundwater. Some 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.

The U.S. Department of Health and Human Services has determined that some PAHs may reasonably be expected to cause cancer. 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) [1].

Exposure Pathways

Inhalation Exposure Pathway

A completed exposure pathway existed if persons inhaled airborne contaminants from the Tire Fire. Air sampling was not conducted during the Tire Fire. Because no air sampling data is available for analysis, an indeterminate public health hazard existed during the fire. The risk of exposure through inhalation should have been limited because of the short duration of the fire, the time of the fire (approximately 3 o'clock in the morning), and the area is sparsely populated.

Surface Soil Ingestion Exposure Pathway

Incidental soil ingestion is considered a potential exposure pathway at this site. The U.S. EPA has developed a Toxic Equivalence Factor (TEF) approach for evaluating potential health effects associated with PAH exposure. Using the TEF approach we assess the carcinogenic potencies resulting from PAH emissions by determining the benzo[a]pyrene equivalent concentration (BaP_{eq}) for each individual PAH species. The determination of BaP_{eq} for each individual PAH

species requires the adjustment of its original concentration by reference to its toxic equivalent factor (TEF), which represents the relative carcinogenic potency of the given PAH species to the specific compound benzo[a] pyrene (BaP). The individual BaP_{eq} for all PAHs are summed and the total is reported as the total BaP_{eq}. Using this concept, the cancer potency of the other carcinogenic PAHs can be estimated on the basis of their relative potency to BaP [2,3].

The three off-site surface soil samples collected on February 28, 2003, indicated a “Non-Detect” (ND) result for all suspect PAHs, as seen in Table 2 of Appendix B. A number often follows the ND abbreviation, such as ND (0.66). This does not mean that the chemical was actually present. It means simply that anything below 0.66 mg/kg would not have been found because it was below the test equipment’s detection limit [4]. However, using the TEF approach discussed earlier we have calculated a total BaP_{eq} as a means of assessing worst-case scenario using the test equipment’s detection limit of 0.66 mg/kg as the concentration for each analyte. Using the parameters stated above, a single sampling site results in total BaP_{eq} of 4.2 mg/kg.

The estimated total BaP_{eq} concentration exposure dose for carcinogenic PAHs in this pathway (incidental ingestion of soil) was calculated using the following assumptions:

1. A 70 kilograms or kg (154 pound) adult ingesting 100 milligrams per day (mg/day) of contaminated soil
2. A 10 kg (22 pound) child ingesting 200 mg/day of contaminated soil
3. Exposure 7 days a week for 52 weeks
4. Exposure duration of 70 years (average human lifetime)

The derivation of a comparison value uses conservative exposure assumptions, resulting in values that are much lower than exposure concentrations observed to cause adverse health effects. This ensures that the comparison values are protective of public health in essentially all exposure situations. We did exposure calculations for individual PAHs, based on a worst-case scenario and they were all below U.S. EPA Risk Based Concentration values. Then we took the total BaP_{eq} and compared the calculated dose to threshold levels of BaP found in the literature. We determined that health concerns were not likely even if we assumed that all exposures were to BaP. Thus exposure to PAHs in soil is not of a health concern and no further analysis of the exposure medium pathway is required.

A level of concentration that is equal to or below a relevant comparison value is considered safe. However, the fact that a concentration exceeds a comparison value does not mean that the concentration is expected to produce adverse health effects. ATSDR uses highly conservative, health-based standards and guidelines to assist health professionals in recognizing and resolving potential public health problems. Soil contaminant concentration values were compared to levels of total PAH concentrations that produced No Observed Adverse Effect Levels (NOAELs) or Lowest Observed Adverse Effect Levels (LOAELs) in animal studies, to determine if adverse health effects may result from exposure to contaminated soil at the site [1]. Estimated doses of BaP total equivalent concentrations are below levels of health concern.

Estimated exposure doses for soil ingestion were calculated in the following manner. The total BaP_{eq} from Appendix B, Table 2 of 4.2 mg/kg was multiplied by the soil ingestion rate for adults, 0.0001 kg/day, or children, 0.0002 kg/day, then divided by the average weight for an adult, 70 kg (154 pound) or a child's body weight of 10 kg (22 pound). The result is the estimated exposure dose in units of milligram per kilogram per day (mg/kg/day).

The estimated exposure dose of PAHs in adults is 0.000006 mg/kg/day and 0.000084 mg/kg/day in children. This dose is far less than the concentrations that produced adverse effects in animal testing [1]. Therefore, it is unlikely that any health effects would result from incidental ingestion of contaminated soils.

Surface Soil Dermal Exposure Pathway

Dermal contact with contaminated soil at the site is also a potential exposure pathway. Potential contact with the soil would likely be infrequent, and involve small areas of the body (i.e., hands primarily). Due to levels of PAHs detected in the surface soil samples, dermal contact does not appear to be a significant route of exposure. No adverse health effects are anticipated via the dermal route of exposure.

Groundwater Exposure Pathway

The potential for groundwater contamination from PAHs generated during the Tire Fire was evaluated. As a class, many of these compounds do not migrate very far in soil, and will biodegrade. The lower molecular weight (smaller in size) PAHs are more water soluble, and have a greater potential to migrate through soil and groundwater. Since the area residents are on individual wells, the potential for well contamination to occur was evaluated to determine if a potential health hazard exists. No raw water sampling data was available for analysis; however, the soil samples collected and analyzed for PAHs indicated that there were not elevated levels of PAHs in the surface soil (see the "Non-Detect" result for all suspect PAHs in Appendix B, Table 2). Therefore, well contamination from the PAHs in the surface soil – as a result of the fire – is not expected.

Cancer Risk Analysis

Estimated individual PAH doses and U.S. EPA's oral cancer slope factors were used to calculate cancer risk. These oral cancer slope factors are theoretical risks, based on conservative (i.e., protective) assumptions [5]. Table 1 shows the oral cancer slope factors for the five PAHs that have U.S. EPA Risk-Based Concentrations below the testing instruments capability. U.S. EPA Risk-Based Concentration values are concentration levels for individual chemicals that correspond to a specific cancer risk level of 10⁻⁶. Each of those five PAHs is discussed further in Appendix B, Table 3. The estimated cancer risk for both adults and children was below cancer health screening values.

COMMUNITY HEALTH CONCERNS

The residents living near the site raised general health concerns relating to the inhalation of smoke and the soot left on their property from the Tire Fire. During follow-up of the site, ADH contacted the concerned citizens. The residents indicated that the clean up of the Tire Fire site was to their satisfaction.

CHILD HEALTH CONSIDERATIONS

Both ADH and ATSDR recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with air, water, soil, or food contamination. 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. Exposure doses via ingestion were estimated for children who might live in the vicinity of the Tire Fire site and no adverse health effects are anticipated.

SITE UPDATE

The site has been cleared of all material associated with the Tire Fire. At the request of area residents, a screen to restrict site access was placed along U.S. Highway 82 on the Tire Fire site property. No future sampling is scheduled for the site. There are no current plans for future use of the site.

CONCLUSIONS

The environmental sampling data from the Tire Fire do not indicate that humans are being or have been exposed to levels of PAH contamination in the soil that would be expected to cause adverse health effects. Since no air samples were collected, an indeterminate public health hazard existed during the fire. However, risk of exposure through inhalation would have been unlikely due to the short duration of the fire, the time of the fire, and the sparsely populated area. Based on the review of available data and under conditions that were present at the time of soil sample collection at the Texarkana Tire Fire site, ADH categorized the levels of PAHs to represent *No Apparent Public Health Hazard*.

RECOMMENDATIONS

No recommendations are indicated at this time.

PUBLIC HEALTH ACTION PLAN

The purpose of the Public Health Action Plan is to ensure that this document not only identifies any current or potential exposure pathways or related health hazards, but also provides a plan of action to mitigate and prevent adverse human health effects resulting from exposures to hazardous substances in the environment. The first section of the Public Health Action Plan contains a description of completed actions to mitigate exposures to environmental contamination. In the second section, there is a list of future public health actions that will be implemented in the future.

Completed Actions

- ADH personnel initially contacted the complainant in January 2003.
- ADH performed a site assessment in February 2003.
- ADH provided Polycyclic Aromatic Hydrocarbons (PAHs) fact sheets to the concerned residents along with the soil sampling results via mail in April 2003.
- ADH personnel contacted the complainant in April 2004 to follow up on the site.

Future Activities

- ADH will provide the concerned residents with a copy of this completed Health Consultation.

AUTHORS, TECHNICAL ADVISORS

Health Assessor:

Dan Seaton
Arkansas Department of Health

Designated Reviewer:

Lori Simmons
Arkansas Department of Health

ATSDR Regional Representative:

George Pettigrew
ATSDR Region VI

ATSDR Technical Project Officer:

Tammie McRae
ATSDR Technical Project Officer
Division of Health Assessment and Consultation

(left blank)

CERTIFICATION

This Health Consultation for Texarkana Tire Fire was prepared by the Arkansas Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedure existing at the time the health consultation was initiated.



Tammie McRae, M.S.
Technical Project Officer
Division of Health Assessment and Consultation (DHAC)
ATSDR

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



Roberta Erlwein
Cooperative Agreement Team Leader, DHAC, ATSDR

(left blank)

REFERENCES

1. Agency for Toxic Substances and Disease Registry. Toxicological profile for polycyclic aromatic hydrocarbons, update. Atlanta: US Department of Health and Human Services; 2000.
2. Nisbet I, Lagos P. Toxic equivalency factors (TEQs) for polycyclic aromatic hydrocarbons (PAHs). *Regul Toxicol Pharmacol* 1992;16:290–300.
3. Lee W-J, Hsieh L-T, Chen T J-H, Tsai P-J. Impact of polycyclic aromatic hydrocarbon emissions from medical waste incinerators on the urban atmosphere. [Electronic version]. *J Air Waste Manag Assoc* 2003;53:1149–57.
4. Alaska Department of Environmental Conservation. How to interpret laboratory data. (Series 1, Fact Sheet #15); 2004 Mar. Retrieved April 20, 2004, from http://www.state.ak.us/dec/spar/csp/guidance/lab_data.pdf.
5. US Environmental Protection Agency. Region VI human health medium-specific screening levels 2003-2004; 2004. Retrieved April 20, 2004 from URL: http://www.epa.gov/earth1r6/6pd/rcra_c/pd-n/screen.htm.
6. US Environmental Protection Agency. Integrated Risk Information System – Benz[a]anthracene (CASRN 56-55-3); 2003. Retrieved April 20, 2004 from URL: <http://www.epa.gov/iris/subst/0454.htm>.
7. US Environmental Protection Agency. Integrated Risk Information System – Benzo[a]pyrene (BaP) (CASRN 50-32-8); 2003. Retrieved April 20, 2004 from URL: <http://www.epa.gov/iris/subst/0136.htm>.
8. US Environmental Protection Agency. Integrated Risk Information System – Benzo[b]fluoranthene (CASRN 205-99-2); 2003. Retrieved April 20, 2004 from URL: <http://www.epa.gov/iris/subst/0453.htm>.
9. US Environmental Protection Agency. Integrated Risk Information System – Dibenz[a,h]anthracene (CASRN 53-70-3); 2003. Retrieved May 19, 2004 from URL: <http://www.epa.gov/iris/subst/0456.htm>.
10. Snell KC, Stewart HL. Pulmonary adenomatosis induced in DBA/2 mice by oral administration of dibenz[a,h]anthracene. *J Natl Cancer Inst* 1962;28(5): 1043–9.
11. Snell KC, Stewart HL. Induction of pulmonary adenomatose in DBA/2 mice by the oral administration of dibenz[a,h]anthracene. *Acta Un Int Cancer*. 1963;19:692–4.
12. Van Duuren BL, Langseth L, Goldschmidt BM, Orris L. Carcinogenicity of epoxides, lactones and peroxy compounds. VI. Structure and carcinogenic activity. *J Natl Cancer Inst* 1967;39(6)1217–28.
13. Wynder EL, Hoffmann D. A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. *Cancer* 1959;12:1079–86.
14. US Environmental Protection Agency. (2003). Integrated Risk Information System – Indeno[1,2,3-cd]pyrene (CASRN 193-39-5). Retrieved April 20, 2004 from <http://www.epa.gov/iris/subst/0457.htm>.

-
15. Deutsch-Wenzel R, Brune H, Grimmer G, Dettbarn G, Misfeld J. Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons. *J Natl Cancer Inst* 1983;71(3):539–44.
 16. Lacassagne A, Buu-Hoi NP, Zajdela F, Lavit-Lamy D, Chalvet O. 1963. Activite cancerogene d'hydrocarbures aromatiques polycycliques a noyau flouranthene. *Un Int Cancer Acta* 19(3-4):490–6. (Fre.)
 17. Hoffmann D, Wynder EL, 1966. Beitrag zur carcinogen wirkung von dibenzopyrene. *Z Krebsforsch* 68(2):137–49. (Ger.)
 18. Rice JE, Coleman DT, Hosted TJ, LaVoie EJ, McCaustland DJ, Wiley JC. On the metabolism, mutagenicity, and tumor-initiating activity of indeno[1,2,3-cd]pyrene. In: M. Cooke M, and A.J. Dennis AJ, editors. *Polynuclear aromatic hydrocarbons: mechanism, methods and metabolism*. Columbus, Ohio: Batelle Press; 1985. p. 1097–1109.
 19. Rice JE, Hosted TJ Jr, DeFloria MC, LaVoie EJ, Fischer DL, Wiley JC Jr. 1986. Tumor-initiating activity of major in vivo metabolites of indeno[1,2,3-cd]pyrene on mouse skin. *Carcinogenesis* 1986;7(10):1761–4.

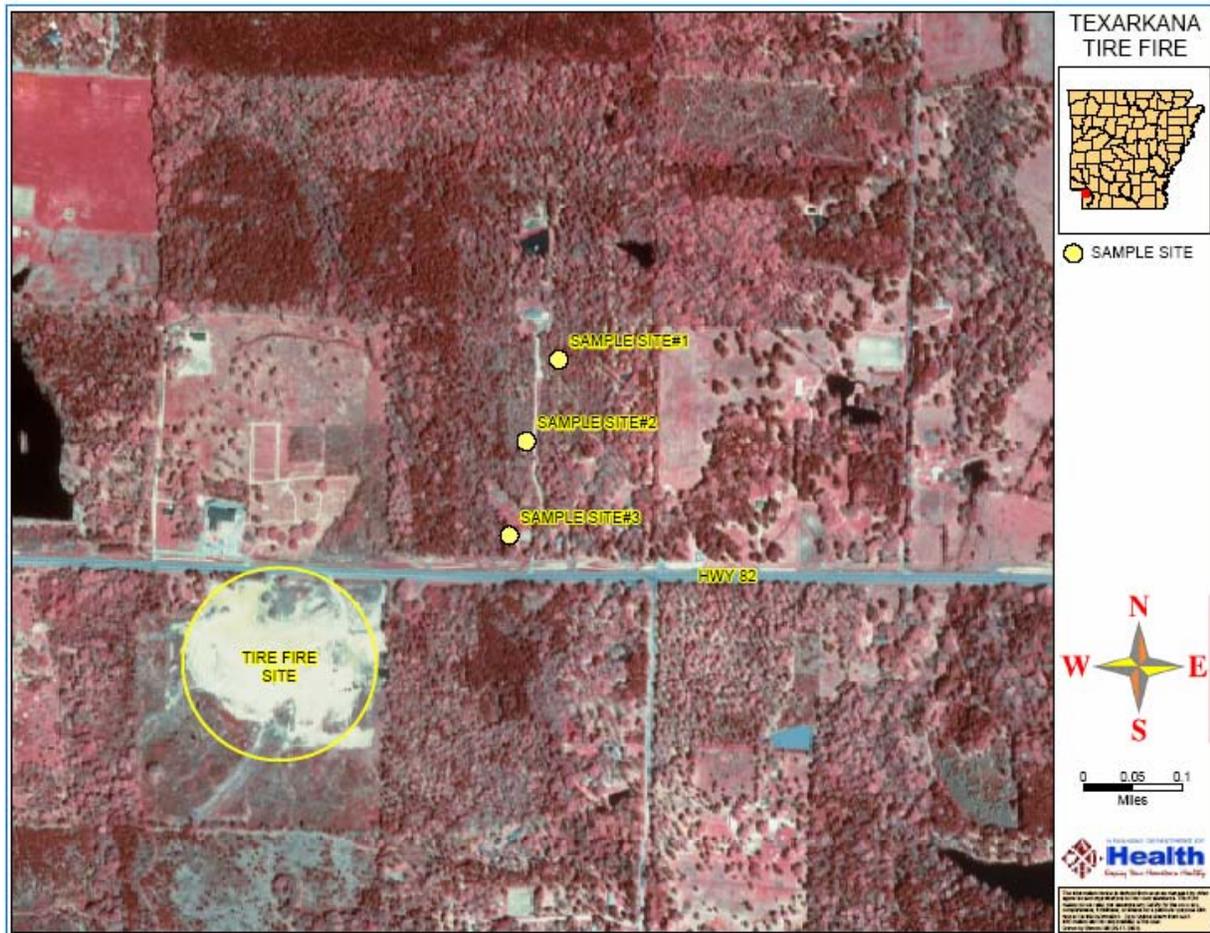
APPENDICES

(left blank)

Appendix A – Figure 1

(left blank)

Figure 1. Aerial Photo of Texarkana Tire Fire and Associated Sampling Sites



(left blank)

Appendix B – Tables

(left blank)

Table 2. 02/28/03 Surface Soil Sample Results

Parameter	Sample Site #1 N 33°26.026 W 093°54.874	Sample Site #2 N 33°25.968 W 093°54.906	Sample Site #3 N 33°25.877 W 093°54.917	Toxic Equivalency Factor (TEF)	†BaP _{eq}	‡Risk-Based Concentrations for Residential Soil
Acenaphthene	§ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	3700
Acenaphthylene	ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	None
Anthracene	ND (0.66)	ND (0.66)	ND (0.66)	0.01	0.0066	22000
Benz[a]anthracene	ND (0.66)	ND (0.66)	ND (0.66)	0.1	0.066	0.62
Benzo[a]pyrene	ND (0.66)	ND (0.66)	ND (0.66)	1.0	0.66	0.062
Benzo[b]fluoranthene	ND (0.66)	ND (0.66)	ND (0.66)	0.1	0.066	0.62
Benzo[ghi]perylene	ND (0.66)	ND (0.66)	ND (0.66)	0.01	0.0066	None
Benzo[k]fluoranthene	ND (0.66)	ND (0.66)	ND (0.66)	0.1	0.066	6.2
Chrysene	ND (0.66)	ND (0.66)	ND (0.66)	0.01	0.0066	62.0
Dibenz[ah]anthracene	ND (0.66)	ND (0.66)	ND (0.66)	5.0	3.3	0.062
Fluoranthene	ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	2300
Fluorene	ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	2600
Indeno[1,2,3-cd]pyrene	ND (0.66)	ND (0.66)	ND (0.66)	0.1	0.066	0.62
Naphthalene	ND (0.66)	ND (0.66)	ND (0.66)	None	None	1200
Phenanthrene	ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	None
Pyrene	ND (0.66)	ND (0.66)	ND (0.66)	0.001	0.00066	2300
Total BaP _{eq}	N/A	N/A	N/A	N/A	4.2	N/A

* All values are in mg/kg = milligram per kilogram or parts per million (ppm)

† BaP_{eq} = Benzo [a] pyrene equivalent concentration

‡ RBC = US EPA Region 3 Risk-Based Concentration values are concentration levels for individual chemicals that correspond to a specific cancer risk level of 10⁻⁶.

§ ND = No Detect. Actual analyte values were below the test equipment's detection limit of 0.66 mg/kg.

Note: the BaP_{eq} and Total BaP_{eq} values used in this table are being used simply to assess a worse case scenario using 0.66 mg/kg as the concentration level for each analyte.

Table 3. Polycyclic Aromatic Hydrocarbons (PAHs) Below Instruments Capability

PAHs	Health Assessment Information Summary
Benz[a]anthracene	<p>Based on no human data and sufficient data from animal bioassays, benz[a]anthracene is classified as a probable human carcinogen. Benz[a]anthracene produced tumors in mice exposed through the introduction of material into the stomach by a tube (gavage); injection; and topical application. Benz[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.</p> <p>Although there are no human data that specifically link exposure to benz[a]anthracene to human cancers, benz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke [6].</p>
Benzo[a]pyrene	<p>Human data specifically linking benzo[a]pyrene (BaP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BaP to be carcinogenic following administration by numerous routes.</p> <p>Studies have shown lung cancer to be induced in humans by various mixtures of PAHs known to contain BaP including cigarette smoke, roofing tar and coke oven emissions. BaP administered in the diet or by the introduction of material into the stomach by a tube to mice, rats and hamsters has produced increased incidences of stomach tumors. It is not possible, however, to conclude from this information that BaP is the responsible agent [7].</p>
Benzo[b]fluoranthene	<p>Although there are no human data that specifically link exposure to benzo[b]fluoranthene to human cancers, benzo[b]fluoranthene is a component of mixtures that have been associated with human cancer [8].</p>
Dibenz[a,h]anthracene	<p>Based on no human data and sufficient data from animal bioassays, dibenz[a,h]anthracene has been classified as a probable human carcinogen [9]. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene to human cancers, dibenz[a,h]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke. Dibenz[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route [10, 11]. Mice developed carcinomas following dermal exposure to dibenz[a,h]anthracene at concentrations of 0.001% or greater [12, 13].</p>
Indeno[1,2,3-cd]pyrene	<p>Based on no human data and sufficient data from animal bioassays, indeno[1,2,3-cd]pyrene has been classified as a probable human carcinogen [14]. Indeno[1,2,3-cd]pyrene produced tumors in mice following lung implants, subcutaneous injection and dermal exposure. Although there are no human data that specifically link exposure to indeno[1,2,3-cd]pyrene to human cancers, indeno[1,2,3-cd]pyrene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke. In carcinogen bioassays, indeno[1,2,3-cd]pyrene exposure resulted in increased incidences of epidermoid carcinomas in a lung implantation study [15], injection site sarcomas in a subcutaneous injection assay [16] and skin tumors in dermal application studies [17-19].</p>