Public Health Assessment

Final Release

LITTLE SCIOTO RIVER SITE

MARION COUNTY, OHIO

EPA FACILITY ID: OHN000509950

Prepared by the
Ohio Department of Health

JULY 11, 2012

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR's Cooperative Agreement Partner pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR's Cooperative Agreement Partner has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 60-day public comment period. Subsequent to the public comment period, ATSDR's Cooperative Agreement Partner addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by 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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CONTENTS

Little Scioto River Site

SUMMARY

Introduction The Little Scioto River site is a six and a half mile section of the Little Scioto River in Marion County, Ohio, just southwest of the City of Marion. The site begins slightly north of State Route 95 and extends to the Scioto River confluence south of Green Camp, Ohio. The sediment in this section of the river was grossly contaminated with coal-tar creosote from historic discharges from the Baker Wood Creosoting (Baker Wood Preserving Company) site (HAS, 2000). Creosote-contaminated sediments have been found in the Little Scioto River beginning downstream of Holland Road and extending to the mouth of the Little Scioto River at the confluence with the Scioto River, approximately six and a half miles to the south (Figures 1 and 2).

Documents from the Ohio Department of Health (ODH) indicate that ODH asked the Baker Wood Creosoting Company to cease operations and discharges that were affecting water quality of the Little Scioto River in the 1940's (ODH 1992).

The Baker Wood Creosoting site was the subject of a time-critical removal action (TCRA) by the United States Environmental Protection Agency (USEPA) in 2000 and 2001. This removal action addressed remnant creosote wastes and grossly contaminated soil in and around the former wood treatment area. Contaminated groundwater and some residual soil contamination remain at the site. The USEPA is conducting a Remedial Investigation/ Feasibility Study (RI/FS) of the Baker Wood Creosoting site along with the RI/FS of the Little Scioto River site to evaluate the cleanup of remaining contaminants.

Between 2002 and 2006, a one-mile section of the Little Scioto River and a half-mile stretch of the North Rockswale Ditch were drained and polycyclic aromatic hydrocarbon (PAH) contaminated sediment was dredged (Figure 2). The river was backfilled with clean soil, and excavated contaminated sediment was dried and removed off-site for disposal. Following completion of the cleanup, at least three and a half river miles of the Little Scioto River still contain gross visible sediment contamination downstream of the previous sediment removal actions (Ohio EPA, 2008b).

In 1992, the ODH issued a fish consumption advisory for all species due to PAH contamination in the four mile stretch of the Little Scioto River, from Holland Road south to State Route 739. A contact advisory was issued at the same time against swimming and wading in this section of the river due to the gross visible contamination of the river sediments. The river bank is not fenced and the public has unrestricted access to this section of the river, although "No Fishing, Swimming, or Wading" signs have been posted.

Conclusion 1

(sediment) The Health Assessment Section (HAS) concludes that frequent contact with PAHs in sediments for more than a year at the Little Scioto River site could harm people's health. This is a public health hazard.

Basis for Decision:

Swimmers and waders coming into contact with sediment contaminated with PAHs could have an increased risk of developing certain types of skin cancer. For more details on the evaluation of exposure to contaminants and the calculation of theoretical cancer risk, see Appendix A. Although the risk is low, the HAS considers it prudent to reduce or eliminate skin contact with PAH-contaminated sediments.

Next Steps: To protect people:

- The USEPA has started a RI/FS to evaluate the cleanup of the contaminated sediments. Results of sediment samples taken along the bank of the Little Scioto River and its tributaries will be included in the RI report.
- HAS will monitor the investigation and evaluate the cleanup levels to ensure that sampling objectives and results are protective of public health.
- HAS will re-assess the sediment contact advisory following the cleanup and review of the post remedial environmental monitoring data.

Conclusion 2

(fish) The HAS concludes that frequently eating fish contaminated with PAHs from sediments from the Little Scioto River site for more than a year is not likely to harm people's health.

Basis for Decision:

Although high levels of metabolites were found in fish bile, concentrations found in the edible portion of fish are low. In addition, there is a "do not eat" fish advisory for this section of the river, and bottom conditions in the river have largely eliminated the sport fish population along this section of the river. However, HAS considers it prudent to reduce or eliminate the amount of fish eaten that are caught in areas of the Little Scioto River with significant PAH contaminated sediments. For more details on the evaluation of exposure to contaminated fish and the estimation of theoretical cancer risk, see Appendix A.

Next Steps: To protect people:

- The USEPA has started a RI/FS to evaluate the cleanup of the contaminated sediments.
- HAS will re-assess the sediment contact advisory and the "do not eat" fish advisory following the cleanup and the provision of new fish data.

Conclusion 3

(water) The HAS cannot conclude whether people drinking well water or surface water

contaminated with PAHs from the Little Scioto River site could harm people's health in the future. The information we need to make this decision is not yet available. Data from public water supplies for groundwater and surface water indicate that current the levels of site-related contaminants are not a health concern.

Basis for Decision:

Well water: Sand and gravel deposits in the bed of the river were encountered during the cleanup of the upper portion of the Little Scioto River site. These sand and gravel deposits were saturated with creosote and petroleum contamination and were within about 600 feet of some residential wells. Although these creosote and petroleum constituents are not likely to be very mobile in the groundwater, residential wells in these sand and gravel areas need to be sampled and the extent of contamination of these deposits need to be defined. There is no evidence that anybody is using these perched sand and gravel layers as a drinking water supply. None were identified from area well logs (ODNR webpage). What few people live in the area along the river between the Holland Road bridge and Green Camp, obtain their water from wells in the underlying limestone bedrock aquifer which likely has not been impacted by the PAH contamination in the river (separated from the surface by 40 feet of mostly impermeable clay).The limestone bedrock underlying the Baker Wood Creosoting site may have fractures that could provide a conduit for contaminants in groundwater to be transported to public wells.

Surface water: Disturbing the sediments in the Little Scioto River could suspend PAHs in the water and could harm the health of people who use these surface waters as a source of drinking water. However, there is no information indicating that anyone is using surface water from the Little Scioto River downstream of the Holland Road bridge as a drinking water supply. There are no drinking water intakes or municipal wells downstream of the Holland Road bridge that use the surface waters of the LSR as a drinking water supply. The closest downstream municipality is Green Camp – 2 miles downstream of the State Route 739 bridge. It is possible that swimmers could be exposed to suspended PAHs through the incidental ingestion of water during recreational use.

Next Steps: To protect people:

- The USEPA has started a RI/FS to evaluate the cleanup of the contaminated sediments. Results from groundwater and sediment samples will be included in USEPA's remedial investigation report.
- During the RI/FS, any data from surface water intakes or residential wells will be evaluated to determine if drinking water from these sources could harm people's health.

For More Information

If you have any concerns about your health, as it relates to exposure to PAHs you should contact your health care provider. You can also call the HAS at (614) 466-1390 and ask for information on the Little Scioto River site.

For updates on the USEPA cleanup activities at this site, see the EPA Region 5 web page at: http://www.epa.gov/region5/cleanup/littlescioto/index.htm.

STATEMENT OF ISSUES

Sediments in a three and a half mile stretch of Little Scioto River are contaminated with PAHs from coal-tar creosote waste from the former Baker Wood Creosoting facility. Baker Wood Creosoting operated from the 1890's to the 1960's (Ohio EPA, 1994). Their operations allowed waste to flow through a combined sewer overflow to North Rockswale Ditch and then to the Little Scioto River, entering the river just south of Holland Road bridge (Figures 1 and 2)(Table 1).

The Ohio Department of Health (ODH) issued two health advisories in 1992. A contact advisory was issued in conjunction with Marion County Health Department for swimmers and waders due to the visibly contaminated sediment in the Little Scioto River from Holland Road downstream to State Route 739. The ODH, in cooperation with the Ohio Environmental Protection Agency (Ohio EPA) and the Ohio Department of Natural Resources (ODNR), issued a fish consumption advisory for all species of fish for the same section of the river due to PAHs (ODH, 1992). Significant levels of abnormalities were documented in fish in the Little Scioto River downstream of Holland Road to the mouth at the confluence with the Scioto River in surveys conducted in 1992 and 1998 (USEPA, 2009a). The fish abnormalities documented by the Ohio EPA in 1994 included tissue anomalies such as deformities, fin erosions, lesions, ulcers, and tumors.

PAH-contaminated sediments may continue to pose a risk to downstream water quality. When the contaminated sediment was disturbed during sampling for the Expanded Site Inspection, an extensive sheen and a strong smell of creosote was present at locations where the highest PAH concentrations were detected. Blobs of oil from heavily contaminated sediment routinely came to the surface throughout the project site and created oil sheens. Sediment erosion during significant flood events is believed to release oils and PAHs into the water column.

During sediment cleanup in the upstream portion of the site, some sand and gravel deposits were saturated with creosote and petroleum contamination (Ohio EPA, 2008b). The distance from the creosote-contaminated sediment to the closest private water wells is approximately 600 feet (Ohio EPA, 2007). These contaminated sediments may pose a risk to the shallow groundwater quality and the water quality of nearby private wells. Additional investigation is planned during the Remedial Investigation (RI) to look into the contaminated sand and gravel layers discovered during the cleanup to determine if the contaminants from the sediment are migrating to area groundwater.

BACKGROUND

The Health Assessment Section (HAS) of the ODH has had a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) since 1990. Under that agreement,

the HAS undertook the lead in completing this public health assessment. The assessment evaluates the environmental data collected by the Ohio EPA and the USEPA as part of their investigations at this site. The HAS makes conclusions and recommendations for additional actions that may be necessary to protect public health.

In support of a USEPA Time-critical removal action, the HAS completed a Public Health Consultation for the Baker Wood Creosoting site in 2000. Creosote waste from the Baker Wood Creosoting site flowed through the combined sewer overflow into the North Rockswale Ditch and then into the Little Scioto River by the Holland Road Bridge. In 1992, the Marion County Health Department and the ODH issued the contact advisory, advising swimmers and waders to avoid physical contact with the contaminated sediments. The ODH, ODNR, and Ohio EPA also issued the "Do Not Eat" fish advisory for the Little Scioto River site from 1992 to the present.

Site Location

The Little Scioto River site is located west of Marion in Marion County, Ohio. The site begins just upstream (about 400 feet) of State Route 95 and extends to the mouth of the Little Scioto River at the confluence with Scioto River, just south of Green Camp (Figures 1 and 2). The Baker Wood Creosoting site, about one mile east of the Little Scioto River and Holland Road bridge (Figure 3), is being evaluated through the Remedial Investigation/Feasibility Study (RI/FS) process. Contaminated sediment was removed from a mile section of the Little Scioto River between 2002 and 2006 and from the contaminated section of North Rockswale Ditch (Figure 2). The three-quarter mile section of the Little Scioto River begins upstream of the Holland Road Bridge and extends south to a location approximately 400 feet upstream of the bridge at State Route 95.

Demographics

Marion County has: a total area of about 404 square miles; a total population of approximately 66,500; and an average population density of 164 people per square mile. Marion is the largest city in the county with a population of over 36,800 people living in the city limits (Census, 2010)

Approximately 91 percent of the people in Marion County are white, 5.7 percent African-American and about 2 percent other races (Census, 2010). At the time of the 2010 Census, 78 percent of the people were 18 years of age or older and 14 percent were 65 years of age or older. There were a total of 27,834 housing units in Marion County with an average of 2.45 persons per household. At the time of the 2010 Census, 68.7 percent of the 24,691 occupied housing units were owner-occupied and 31.3 percent were rented. Also from the 2010 Census, but based on 2006-2010 income, 17.3 percent of the people (of all ages) living in the Marion County were living with incomes below the poverty level (Census, 2010).

Land Use

The site is in a sparsely-populated rural area 1.5 miles west and southwest of the City of Marion. The area along the Little Scioto River site is a mixture of agricultural croplands and wetland properties. There are some urban areas, primarily industrial with some commercial and

residential areas east of the river near the City of Marion.

Several sections of the site are adjacent to or run through portions of the Big Island Wildlife Area (ODNR, 2010). The Big Island Wildlife Area is managed by the Ohio Department of Natural Resources (ODNR, 2010). Marion County Park District is building a bike path on an abandoned railway just west of the site. The Park District has established a parking area for the bike path on the west bank of the river just south of Holland Road (USEPA, 2009a). Contaminated sediments were recently removed from this area.
There are several other potential sources of contamination along the site; however, none of these sources are thought to be significant sources of PAHs. The Marion Wastewater Treatment Plant and the Marion Landfill are located along either side of the river south of Holland Road (Ohio EPA, 1994) (Figure 3). There are also numerous industrial sources along Rockswale Ditch east of the site, including facilities belonging to Whirlpool and Dana Corporation.

REGIONAL GEOLOGY AND HYDROLOGY

Geology

Soils in the area of the site were formed from receding glaciers and are generally dense silty clay loams or clay loams (Ohio State University, 2010). These glacial soils also contain occasional thin inter-bedded sand layers. These sand layers can extend from beneath the surface soil to the underlying limestone and dolomite bedrock, at depths of approximately 13 to 25 feet below ground surface. The clayey overburden is approximately five feet thick at the Baker Wood Creosoting site and up to 40-50 feet thick to the west at the Ohio American Water Company (OAWC) well field just north of the Holland Road bridge (ODNR well logs).

The uppermost bedrock in central Marion County is limestone and is typically over 100 feet thick. Karst features (bedrock solution features like caves and sinkholes) can be found in some of these limestone layers. Dolomite bedrock is found underlying the limestone with several layers exceeding 100 feet thickness. Fractures can be found in these limestone and dolomite bedrock units and when encountered in wells can provide groundwater yields greater than 100 gallons per minute (gpm) (ODNR, 2003).

Water and Groundwater Resource Use

Private wells supply drinking water to approximately 15 percent of the population of Marion County. Public water systems supply water to the remaining 85 percent. These public water systems receive 86 percent of their water from surface water sources (Ohio State University, 2010). About five public water supply wells are in close proximity to the Little Scioto River site. Of these, four are Ohio American Water Company drinking water wells with depths reported to range from 183 feet to over 500 feet (Ohio EPA, 2007). The Ohio American Water Company serves about 48,000 people in the City of Marion and in the surrounding suburban areas (Ohio EPA, 2007). Surface water from the Scioto and Little Scioto Rivers normally supplies 75 to 80 percent of the supply needs for the Ohio American Water Company (Ohio State University, 2010). The surface water intake in the Little Scioto River is located approximately 1,000 feet upstream of the Holland Road Bridge and the Little Scioto River Site (see Figure 3). The raw

water intake is upstream of a low-head dam constructed for the intake system. This surface water supply is augmented by groundwater pumped from 16 production wells that obtain their water from limestone aquifers 20-60 feet below the ground surface (Ohio EPA, 2008; ODNR well logs). In central and western Marion County, wells obtain groundwater from the underlying limestone and dolomite aquifers. The upper aquifer yields about 5 to 100 gpm and deeper limestone aquifer yields over 100 gpm (Ohio EPA, 2008b). The regional groundwater flow in the area of the Little Scioto River site is believed to be influenced by a quarry northeast of the Baker Wood Creosoting site and the public water supply wells in the OAWC well field, upstream and along the Little Scioto River. The closest municipal well to the creosote contamination in the Little Scioto River is approximately 2,700 feet to the north and the closest private well is approximately 600 feet to the west (Ohio EPA, 2008b). The generalized groundwater flow at the bedrock-till interface and in the bedrock itself is to the west (Ohio EPA, 2008b). Actually, in the vicinity of the LSR at Holland Road bridge, groundwater flow in the bedrock aquifer appears to be to the north paralleling the LSR [K.S. Crowell. 1979. Ground-Water Resources of Marion County. ODNR Division of Water. (map)].

Hydrology

The Little Scioto River is 27.2 miles long (OCAFS, 2001). It begins in Crawford County and flows south into the Scioto River at Green Camp, draining 113 square miles (Ohio State University, 2010) (See Figure 1). The Scioto River also flows generally from north to south and is a major tributary of the Ohio River. The channel of the Little Scioto River was re-routed sometime between 1909 and 1927 (Ohio EPA, 2008b) likely in response to statewide flooding in 1913. Old channels and oxbows were abandoned when the river was channelized and straightened (Ohio EPA, 2008b). At the Holland Road bridge, the Little Scioto River is little more than a creek with the channel roughly 50-60 feet wide and water depths during the summer months of no more than 2 feet.

Marion County receives an average of 34 inches of precipitation per year, of which 9 inches becomes runoff moving to streams and lakes. The county is generally level, with only 235 feet of relief (Ohio State University, 2010). Drainage is a major concern for management of croplands and locating suitable residential sites (Ohio State University, 2010). Prior to the arrival of European settlers, this area was once part of a large wetlands prairie (ODNR, 2010).

SITE HISTORY

Baker Wood Creosoting Site

The Baker Wood Creosoting site is located in an industrial area about half a mile west of downtown Marion at the northwest corner of Holland Road and Kenton Street (State Route 309) (see Figure 2).

The facility operated for approximately 70 years, from 1890 through the late 1960's. It produced railroad ties treated with coal-tar creosote. The waste generated was likely a mixture of chemicals used in the railroad tie preservation process—coal-tar creosote, petroleum, and other solvents. The western portion of this 60 acre facility was used to stack and dry the railroad ties,

while the creosoting process took place on the eastern portion. A combined sanitary and storm sewer ran along the southern border of the facility and provided likely transport of creosote waste discharges to Rockswale Ditch and the Little Scioto River.

On September 4, 1946, the ODH cited Baker Wood Creosoting as a contributor of contamination to Little Scioto River surface water. Coal-tar creosote was being discharged from Baker Wood Creosoting to combined sewers that drain into North Rockswale Ditch and the Little Scioto River approximately one mile west of the site by the Holland Road bridge. ODH recommended that Baker Wood Creosoting install a waste treatment system to reduce off-site release of the creosote waste. A treatment system was not installed until 1953 (Ohio EPA, 2008b). The ODH documented that Baker Wood Creosoting continued to discharge creosote materials from their property. The ODH urged the company to cease any operations affecting the water quality in Little Scioto River. Baker Wood Creosoting ceased operations sometime in the late 1960's.

This site came to the Ohio EPA's attention from historical aerial photographs while investigating the neighboring Union Tank Car site. At the time, Baker Wood Creosoting site was being used as scrap metal salvage yard. In 1991, the Ohio EPA determined that PAHs were present at the Baker Wood Creosoting site; however, data was inconclusive that PAHs were migrating off-site (Ohio EPA, 2008b).

Little Scioto River Site

From August 1992 to February 1993, the Ohio EPA Division of Surface Water conducted a biological and water quality study of the Little Scioto River. Sampling included fish community, macroinvertebrate community, fish tissue, biomarker, sediment and surface water. Sediment samples were collected in North Rockswale Ditch, Rockswale Ditch, and Columbia Ditch. The Ohio EPA concluded a severe biological and water quality degradation existed in the lower Little Scioto River, a one-half mile portion of North Rockswale Ditch, all of Rockswale Ditch, and the lower mile of Columbia Ditch. Very high levels of a number of polycyclic aromatic hydrocarbons (PAHs) were found in these sediments (Ohio EPA, 2008b).

On March 20, 1992, in light of the concentrations of PAHs in the sediment and fish detected in 1988 and 1991 (Ohio EPA, 1992), the ODH in cooperation with the Ohio EPA and ODNR issued an advisory against swimming, wading and eating fish caught in the four-mile length of the Little Scioto River west of the city of Marion, from Holland Road south to State Route 739 (ODH, 1992). The Ohio EPA sampled biota and sediment again in 1992 and 1993 and determined that highly elevated levels of PAHs were in the sediments in North Rockswale Ditch and the outfall to Little Scioto River. There was a creosote odor from the black sediment and fish were being exposed to carcinogenic PAH compounds (Ohio EPA, 1994).

In 1996, the Ohio EPA did an Integrated Assessment of the Baker Wood Creosoting site. At that time, the Ohio EPA sampled the combined sewer on the southern edge of Baker Wood Creosoting site. Their analytical data confirmed a direct relationship between the compounds detected in the water in the sewer and the sediment in the ditch with the coal tar and creosote compounds from the Baker Wood Creosoting site (Ohio EPA, 2008b).

The Ohio EPA, Division of Surface Water, produced a report "Biological and Water Quality Study of Marion Area Streams 1998." This study confirmed that the sediment in the four mile length of lower Little Scioto River remained severely contaminated with PAHs and several heavy metals. A comparison of the 1998 data to the 1992 data indicated that there was no improvement in sediment quality in the lower sections of the Little Scioto River. Abnormalities in fish (DELTs – deformities, fin erosions, lesions, ulcers, and tumors) were prevalent in this section of the river. The high number of pollution-tolerant species and a high number of abnormalities in the fish suggested toxic conditions continued to exist in the lower section of the Little Scioto River (Ohio EPA, 2008a).

USEPA – Baker Wood Creosoting Site

In 1999, the USEPA Region 5 Office requested HAS to evaluate environmental sampling data for the Baker Wood Creosoting site. The HAS concluded that the Baker Wood Creosoting site posed a public health hazard because of the potential for exposure to elevated levels of creosote compounds and arsenic in on-site soils. These conclusions and recommendations to fence the site and identify, contain, mitigate, or remove contaminants from soils on the site were provided to the USEPA in 1999 (HAS, 2000).

In April 1999 the USEPA began a time-critical removal action. Grossly contaminated surface and shallow subsurface soils were removed for disposal off-site. Additional contaminated soils were excavated and treated by bio-remediation on-site. The remediated soils were then shipped off-site for disposal as non-hazardous waste in 2001 (Ohio EPA, 2008b). The site has been vacant since the early 1990's, and there are currently no workers on-site (Ohio EPA, 2008b).

An Expanded Site Inspection (ESI) was conducted by the Ohio EPA in 2003 and concluded that the Baker Wood Creosoting facility no longer poses a threat to the Little Scioto River. Creosote contaminants were detected in low concentrations in shallow groundwater but did not pose a threat to municipal water supply wells. Five private wells in close proximity to the site were sampled in 2007 and did not show any detectable organic compounds or metals above federal drinking water standards. Analysis included the following parameters: volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), pesticides, and polychlorinated biphenyls (PCBs), and 23 targeted metals plus cyanide (Ohio EPA, 2008b).

USEPA – Little Scioto River Site

From May 1999 through May 2000, the USEPA conducted a site assessment of contaminated sediments from the Little Scioto River and North Rockswale Ditch as related to Baker Wood Creosoting contamination (E & E, 2000; Ohio EPA, 2008b). The assessment determined that the sewer on the south side of the Baker Wood Creosoting site was linked to North Rockswale Ditch and the Little Scioto River. A majority of the creosote contamination was in the sediment in the upper two and a half mile section of the Little Scioto River site. However, there was detectable contamination in the sediment in four miles of the river and a half mile of North Rockswale Ditch with creosote (E & E, 2000). Access to the Little Scioto River is unrestricted. It can be reached from nearby farm fields, bridges, state designated recreation areas, and wildlife areas (Ohio EPA, 2008b).

Phase 1 Clean-up
From June 10, 2002, until December 18, 2002, the USEPA mobilized their Emergency Rapid Response Services and their Superfund Technical Assessment and Response Team for Phase 1 removal activities. Contaminated sediments were removed from 2,800 linear feet of North Rockswale Ditch (from the storm sewer overflow to the Little Scioto River) and 2,900 linear feet of the Little Scioto River starting from approximately 800 feet upstream of the Holland Road Bridge and working south (Figure 2). Contaminated sediments were temporarily stored on-site. From May 27 through July 17, 2003, contaminated sediments were shipped off-site for disposal at a landfill (Ohio EPA, 2008b).

Phase 2 Clean-up
The USEPA Phase 2 clean up began on May 22, 2006, and continued through September 28, 2006. Sediment was removed from an additional 2,800 linear feet of the Little Scioto River and shipped off-site for disposal (Ohio EPA, 2008b). Work ended approximately 400 feet upstream of the intersection of State Route 95 in November 2006 (Figure 2) (USEPA, 2009a).

In April 2007, the Ohio EPA collected data for a Site Inspection (SI) report for the Little Scioto River site. The Ohio EPA stated in this report that "*portions of LSR* (Little Scioto River) *pose a substantial threat to human health and the environment from highly contaminated creosote laden sediments. Sediments discharge oil sheens and PAHs (polycyclic aromatic hydrocarbons) contaminants into surface waters that migrate downstream.*" The Ohio EPA also stated that impacts from the contaminated sediment were noted downstream of the mouth of the Little Scioto River in the Scioto River. Additionally, the report concluded that contamination had entered the shallow groundwater from sand and gravel layers that are interconnected with North Rockswale Ditch and Little Scioto River (Ohio EPA, 2007). The Ohio EPA's biological and water quality studies have also documented severe degradation in the Little Scioto River site over last two decades (Ohio EPA, 2008a).

The Ohio EPA, in an ESI report, confirmed that highly contaminated creosote-laden sediments had originated from the Baker Wood Creosoting site and posed a threat to human health and the environment. They documented levels of PAHs in the sediments (Figure 4) and surface waters and PAH metabolites in fish (suckers and carp) (Ohio EPA, 2008b). The Ohio EPA sampled the shallow groundwater monitoring wells at the Baker Wood Creosoting site in 2007 and all PAHs were below levels of concern (below the drinking water quality standards – MCLs or maximum contaminant levels) (Ohio EPA, 2008b). The biological data collected indicates that fish and other aquatic life have improved in the upper section of the impacted Little Scioto River site over the past 20 years likely due to upgrades in the Marion Wastewater Treatment Plant and removal of PAH-laden sediments from North Rockswale Ditch and the Little Scioto River. However, the report cautions, "conditions could easily degrade in a stream undergoing continuous natural changes that may re-suspend and re-distribute contaminated sediments." The Little Scioto River site was listed on the National Priorities List (NPL) of Superfund sites on September 23, 2009. The site was defined geographically spanning from State Route 95 to the confluence with the Scioto River at Green Camp (USEPA, 2009a).

EXPOSURE PATHWAYS

In order for the public to be exposed to chemical contaminants in and around the Little Scioto River site, they must first come into contact with the contaminated groundwater, surface water, soils, sediment, or air. To come into contact with the contaminated media there must be a *completed exposure pathway*. A completed exposure pathway consists of *five main parts,* which must be present for a chemical exposure to occur. These include:

- A Source of the Toxic Chemicals of concern;
- A method of Environmental Transport, which allows the chemical contaminant to move from its source (soil, air, groundwater, surface water, sediment);
- A Point of Exposure where the residents come into direct physical contact with the chemical (on-site, off-site);
- A Route of Exposure, which is how the residents come into physical contact with the chemical (drinking, eating, touching); and
- A Population at Risk which are the people who could possibly come into physical contact with site-related chemicals.

Exposure pathways can also be characterized as to when the exposure occurred or might occur in the *Past, Present, or Future*.

Physical contact with a chemical contaminant, in and by itself, does not necessarily result in adverse health effects. A chemical's ability to affect a resident's health is also controlled by a number of factors, including:

- How much of the chemical a person is exposed to (the dose).
- How long a person is exposed to the chemical (duration of exposure).
- How often a person is exposed to the chemical (frequency).
- The toxicity of chemicals the person is exposed to (how chemicals can make people sick).

Other factors affecting a chemical's likelihood of causing adverse health effects upon contact include the resident's:

- Personal habits
- Diet
- Age and sex
- Current health status
- Other exposures to toxic chemicals (occupational, hobbies, etc.)

The site-related chemicals of concern found in the sediment at the Little Scioto River site are primarily Polycyclic Aromatic Hydrocarbons (PAHs). Table 3 identifies pathways for potential human exposure to contaminants at this site.

Sediment

The ODH and Marion County Health Department have had a contact advisory for sediments for waders and swimmers for the Little Scioto River site from Holland Road Bridge downstream to State Route 739 since 1992 (ODH, 1992). The 1992 contact advisory was based on the presence of gross, visible contamination in the river plus data from an Ohio EPA evaluation (Ohio EPA, 1992) which found elevated levels of PAHs, heavy metals and solvents in sediments in the river. The evaluation found the banks and bottom of the Little Scioto heavily saturated with black material that has a creosote odor and, when disturbed, releases a rainbow sheen on the surface. The foul smell and tar-like consistency of the grossly contaminated sediments likely limited exposures of residents to contaminants even before the "No Wading or Swimming" signs were posted in this section of the river. People who ignore the advisory could come into dermal or skin contact with PAH-contaminated sediments. There is no information indicating whether people came into contact with contaminated sediment prior or subsequent to the 1992 contact advisory. ODH documents indicate that Baker Wood Creosoting affected water quality in the Little Scioto River as far back as the 1940's. Therefore, it is likely that residents might have come into contact with PAHs in river sediment and fish from the river since then. People having regular contact with contaminated sediments when swimming and wading could be exposed to contaminants at levels that may cause harm to their health. The range of concentrations of carcinogenic PAHs in contaminated sediment is shown in Table 1. For more details on the evaluation of exposure to sediment and the estimated theoretical cancer risk, see Appendix A.

Food Chain (Fish)

Due to the gross contamination of the sediments in the river, the ODH, ODNR, and Ohio EPA issued the "Do Not Eat" fish advisory in 1992 for the Little Scioto River site from Holland Road bridge downstream to State Route 739. There is no information indicating whether or not people caught and ate contaminated fish from the Little Scioto River before or after the fish advisory was issued. Bottom conditions in the river have largely eliminated the sport fish population along this section of the river—the fish population in this section of the river is severely reduced. Therefore, it is unlikely that residents would be catching and eating large amounts of contaminated fish from this area. Past investigations showed anomalies, such as deformities, fin erosions, lesions, ulcers, and tumors (DELT anomalies) in a high percentage of fish in the contaminated section of river (Ohio EPA, 1994). High levels of metabolites were found in fish bile, indicating that fish were exposed to polycyclic aromatic hydrocarbons (PAHs) [Ohio EPA 1994; 2008a]. Although high levels of metabolites were found in fish bile, limited whole-body fish data from the Ohio EPA historically show that either PAHs were not detected or were very low (Ohio EPA, 1994). More recent fish samples that were collected by the U.S. EPA during its remedial investigation indicate low levels of PAHs in fish tissue (personal communication, U.S. EPA, June 2012). Based on the U.S. EPA data, the estimated theoretical cancer risk from eating PAH-contaminated fish from the Little Scioto River is very low. However, HAS considers it prudent to reduce or eliminate the amount of fish eaten that are caught in areas of the Little Scioto River with significant PAH contaminated sediments. For more details on the evaluation of exposure to contaminated fish and the estimation of theoretical cancer risk, see Appendix A.

Groundwater

The Marion public water supply has 16 wells that provide water for the Marion water supply system, which uses both groundwater (approximately 51%) and surface water (approximately 49%) (Ohio EPA, 2008b). The closest municipal well to the contamination is approximately 2,700 feet to the north (Figure 3). Sampling data from the public wells indicates that no contaminants of concern, i.e., PAHs and heavy metals, have been detected in the Marion City public water supply. Surface water and groundwater data from the city indicate that people are not drinking water contaminated with site-related contaminants. The monitoring wells around the Baker Wood Creosoting site indicate that the levels of PAHs do not pose a contamination threat to the public water supply (Ohio EPA, 2008b).

The distance from the contamination to the closest private residential well is approximately 600 feet (Ohio EPA, 2007). Five residential (private) wells were chosen to be sampled during the ESI based on the proximity to the Little Scioto River. The analysis included VOCs, SVOCs (e.g., PAHs), pesticides, PCBs, metals (e.g., arsenic), and cyanide. Results indicate that the contaminants from the sediment have not migrated to residential water supplies (Ohio EPA, 2008). It is unknown whether the contaminated sediments from the Little Scioto River site pose a threat of exposure to area residents via the groundwater pathway. There are no data to determine if the groundwater from the contaminated sand and gravel deposits has impacted the quality of water from private wells. These contaminated sediments may pose a risk to the shallow groundwater quality and the water quality of nearby private wells. Well logs (ODNR, 2011) indicate that most all area private wells obtain their water from the deeper limestone bedrock aquifer which is separated from the surface by up to 50 feet of mostly impermeable clay. Additional investigation is planned during the Remedial Investigation (RI) to look into the contaminated sand and gravel layers discovered during the Phase 1 and 2 clean-up to determine if the contaminants from the sediment are migrating to area groundwater and potentially impacting nearby wells.

Surface Water

In November 2006, the Ohio EPA collected six surface water samples as part of a SI report for the Little Scioto River site. No significant levels of contamination were detected in the surface water samples collected in the Little Scioto River or Scioto River. The Ohio EPA also stated in this report that "*Portions of LSR pose a substantial threat to human health and the environment from highly contaminated creosote laden sediments. These sediments continually discharge oil sheen and PAH contaminants into surface waters that migrate downstream*" (Ohio EPA, 2007). Disturbing the sediments in the Little Scioto River could suspend PAHs in the water and could harm the health of those using these surface waters as a source of drinking water. However, currently there is no information indicating that anyone is using surface water from the LSR downstream of the Holland Road bridge as a drinking water supply (Figure 3).

CONTAMINANTS OF CONCERN

The primary contaminant of concern found in the sediment at the Little Scioto River site is

creosote. The major chemicals in coal tar creosote that can cause harmful health effects are polycyclic aromatic hydrocarbons (PAHs). Therefore, this discussion will focus mainly on PAHs. The following information on the toxicological effects of creosote and PAHs has been derived from occupational exposures and laboratory studies in which people and animals were exposed to high levels of these compounds. For the lower level, periodic environmental exposures from the contaminants at this site, we would not expect the same health effects. The following sections on creosote and PAHs present general summaries of health effects. Public health implications of exposure to these contaminants from sediments and fish are discussed later.

Creosote

Creosote is the name used for a variety of products: wood creosote, coal tar creosote, coal tar, coal tar pitch, and coal tar pitch volatiles. The chemical components in creosote mixtures are rarely consistent in their type or concentrations. Coal tar creosote is the creosote fraction that is specifically used for wood preservation and at least 75 percent of the coal tar creosote mixture is PAHs. Coal tar creosote is a thick, oily liquid typically amber to black in color. It is a mixture of various organic hydrocarbon compounds. Components of creosote that do not dissolve in water will remain in place as a tar–like mass. Some compounds of coal tar creosote dissolve in water and may move through the soil to groundwater. Once in groundwater, these compounds may take years to break down. Coal tar creosote can build up in plants and animals (ATSDR, 2002).

Brief direct contact with large amounts of coal tar creosote may result in a rash or severe irritation of the skin, chemical burns of the surface of the eyes, convulsions and mental confusion, kidney and liver problems, unconsciousness, and even death. Direct skin contact over a long time to low levels of creosote mixtures or their vapors can result in increased light sensitivity, damage to the cornea, and skin damage. Extended exposure to creosote vapors can cause irritation of the respiratory tract (ATSDR, 2002).

Skin cancer and cancer of the scrotum are health effects that have occurred after many years of exposures to creosote. Cancer of the scrotum in chimney sweeps has been associated with long-term skin exposure to soot and coal tar creosotes. Long-term exposure to creosote in the workplace, especially direct contact with the skin during wood treatment or manufacture of coal tar creosote-treated products, has resulted in skin cancer and cancer of the scrotum. Laboratory animal studies have also shown skin cancer from skin exposure to coal tar products (ATSDR, 2002). Children exposed to creosote will probably experience the same health effects seen in adults exposed to creosote. We do not know whether children differ from adults in their susceptibility to health effects from creosote (ATSDR, 2002).

Little Scioto River Exposure Scenario

The toxicity characteristics of the creosote wastes that were dumped into the Little Scioto River via the North Rockswale Ditch are not known. It is unknown if exposure to the sediment contaminated with the creosote wastes could cause or could have caused any of the previously described health effects. During the many years that these wastes have been in the environment, some of the creosote compounds may have broken down. However, previous (2007)

observations indicate that when the sediment is disturbed, an oil sheen floats to the surface of the water and a strong odor of creosote can be detected. It is likely that swimmers and waders will have contact with the Little Scioto River sediments only during the few warm months of the year and would not have the same regular, long-term exposures to low levels of creosote that resulted in skin cancer and cancer of the scrotum. Considering the uncertainty of the toxicity characteristics of the creosote in the sediment, it would be prudent to avoid skin contact with contaminated Little Scioto River sediments.

Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different compounds that typically occur in the environment as a mixture of two or more of these compounds. PAHs are found in creosote, coal tar, crude oil, driveway sealer, and roofing tar, but are also formed by incomplete combustion of coal, oil and gas, garbage, and other organic substances like tobacco smoke and charbroiled meat (ATSDR, 1995). The bulk of the PAHs in the environment consist of heavier molecular weight compounds that form solids that tend to be rather persistent in most environmental settings.

In general, due to their low solubility in water and their high rate of adsorption to organic particles, PAHs released to aquatic environments tend to concentrate in the sediments near the site of deposition. Most low molecular weight PAHs, compounds with two to three rings (naphthalene, acenaphthene, acenaphthylene, anthracene, fluorene, phenanthene), tend to be removed from the aquatic environment through volatilization and biodegradation (ATSDR, 1995). PAHs that volatilize to the air typically break down by reacting with sunlight and other chemicals over a period of days to weeks (ATSDR, 1995). Medium and high molecular weight PAHs, such as, those that have four and five or more rings (fluoranthene, pyrene, chrysene, benzo[a]anthracene and benzo[a]pyrene, benzo[g,h,i]perylene), tend to be removed from the aquatic environments through volatilization and adsorption to suspended organic particles with subsequent deposition to sediment (ATSDR, 1995). At the Little Scioto River site, the concentrations of PAHs in the sediments are so high that they are believed to be toxic to microorganisms that would ordinarily biodegrade PAHs in sediment (ATSDR, 1995).

PAHs can bio-accumulate in fish from contaminated sediment and water. PAHs can accumulate in fish tissues at rate of 10 to 10,000 times the concentration found in water, sediment, and food. In general, these accumulations in fish tissues were greater for the higher molecular weight compounds than for the lower molecular weight compounds. When fish take PAHs into their bodies, carcinogenic and mutagenic compounds can be formed and fish exposed to PAHs have developed tumors. Tissue samples taken from fish from heavily PAH-contaminated environments frequently had no benzo[a]pyrene or very low levels detected. This is attributed to the ability of fish to rapidly metabolize and eliminate PAHs (ATSDR, 1995).

For most people living in the U.S., the greatest sources of exposure to PAHs are inhalation of the compounds in tobacco smoke, wood smoke, and contaminated air, and ingestion of the compounds in grilled or smoked foods. It has been estimated that the general U.S. population has an average total daily intake of PAHs 0.207 micrograms per day (µg/day) from air, 0.027 µg/day

from water, and 16-1.6 µg/day from food. It is important to note that although the exposure estimates are relatively high for ingestion of contaminated food, these levels are uncertain because the levels of PAHs in food are not well monitored (ATSDR, 1995).

Pregnant mice had difficulty reproducing after they were fed high levels of one PAH compound during pregnancy and so did their offspring. Higher rates of birth defects and lower body weights also occurred in these offspring. It is not known whether these effects occur in people. Short-term and long term exposures to PAHs have been shown to cause harmful effects on the skin, body fluids, and ability to fight disease in laboratory animal studies. These effects, however, have not been seen in people (ATSDR, 1995).

The Department of Health and Human Services (DHHS) has determined that some PAHs may reasonably be expected to be carcinogenic. The U.S. EPA has determined that benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-c,d)pyrene are probable human carcinogens (ATSDR, 1995). Some people, particularly workers exposed to mixtures containing PAHs (e.g., coal tar, roofing tar, soot, coke oven emissions), have developed lung or skin cancer after breathing or touching mixtures of PAHs and other chemicals for long periods of time. Laboratory animals have developed lung cancer after breathing air, stomach cancer after ingesting food, or skin cancer after certain PAHs were applied to their skin (ATSDR, 1995).

Little Scioto River Exposure Scenario

Swimmers and waders coming into contact with sediment contaminated with PAHs could have an increased risk of developing certain types of skin cancer. For more details on the evaluation of exposure to PAHs and the estimated theoretical cancer risk, see Appendix A. Although the risk is low, the HAS considers it prudent to reduce or eliminate skin contact with PAH-contaminated sediments.

Metals

Numerous metals were detected at elevated levels in the sediment sample collected just south of the State Route 95 bridge during the ESI. The area most recently dredged where contaminated sediments were removed at Holland Road (RM 6.5) exhibited minimally elevated levels of several heavy metals, such as arsenic, cadmium, copper and nickel. Further downstream sampling sites (RMs 5.7–2.7) had slightly higher sediment levels of metals. At State Route 739 (RM 2.7), the metals that were the most elevated were cadmium, chromium and nickel. Analytical results for private well water samples did not show any elevated metals contamination above drinking water quality standards (Ohio EPA, 2008b).

DISCUSSION

Some workers who have had skin contact with mixtures of PAHs and other chemicals for long periods of time have developed skin cancer. Occupational studies have indicated that workers who have had dermal exposures to mixtures of PAHs have developed skin cancer (ATSDR, 1995). Certain PAHs are known to cause cancer in laboratory animals when applied to their skin

(ATSDR, 1995). At the Little Scioto River site, the PAH-contaminated sediments have been determined to be present in layers up to 48 inches deep. These sediments are visibly contaminated and when disturbed, an oily sheen floats to the surface of the water and a strong creosote odor is released to the air. Currently, at least 3.5 miles of the Little Scioto River contain gross, visible sediment contamination (Figure 4). This section is not fenced and public access is unrestricted. The ODH and Marion County Health Department have had a contact advisory for sediments in this section of the river since 1992. Although, "No Wading or Swimming" signs have been posted in this section of the river, the foul smell and tar-like consistency of the grossly contaminated sediments has likely limited exposures to residents to contaminants even before the signs were posted. People who ignore this "No Wading or Swimming" advisory could have brief skin contact with PAH contaminated sediments, however it is unlikely that they would have frequent skin contact. The theoretical cancer risk for people having dermal contact with the sediment or inadvertent ingestion of sediment contaminated with PAHs from the Little Scioto River for 90 days per year for 30 years is higher than the cancer risk range suggested by the USEPA (see Appendix A). Theoretical cancer risk is an estimate of a population's risk of developing cancer following exposure to the chemical. Typically used ranges of cancer risk are from one extra individual with cancer in 10,000 (10^{-4}) to one extra individual in 1,000,000 (10^{-6}).

The chemical components in creosote mixtures are rarely consistent in their type or concentrations (ATSDR, 2002). It is likely that the creosote waste from Baker Wood Creosoting was less concentrated and less consistent than the creosote mixtures used in treating wood. Therefore, the past toxicity characteristics of the creosote wastes dumped into the Little Scioto River via the North Rockswale Ditch are not known. Because the levels of environmental exposures from the contaminants at this site are low, it is unlikely that exposure to the sediment contaminated with the creosote wastes could currently cause or could have caused in the past the same acute health effects as exposure to high levels of creosote would cause, such as, rash or severe irritation of the skin or chemical burns, chemical burns of the surface of the eyes, convulsions and mental confusion, kidney and liver problems, unconsciousness, and even death. During the many years that these wastes have been in the environment, some of the creosote compounds may have broken down. However, considering the uncertainty of the toxicity characteristics of the creosote waste in the sediment, it would be prudent to avoid skin contact or inadvertent ingestion of the sediment.

In a 2007 study conducted by the Ohio EPA, it was determined that in areas of sediment contamination in the Little Scioto River the levels of metabolites in fish for the PAH compounds naphthalene and benzo[a]pyrene were highly elevated above background and these levels confirm the fishes' extensive exposure to PAHs (Ohio EPA, 2008b). This section of the Little Scioto River is the same area of sediment contamination that is posted with a "Do Not Eat" fish advisory. In general, recreational and subsistence fishers that consume appreciably higher amounts of locally-caught fish from contaminated water bodies may be exposed to higher levels of PAHs associated with dietary intake (ATSDR, 1995). Infrequent fishers are not expected to be exposed to appreciable levels of PAHs.

Sites in Ohio that contain high levels of PAHs in the sediments appear to also have a high percentage of fish with tissue anomalies, such as deformities, fin erosions, lesions, ulcers, and tumors (DELT anomalies) (Ohio EPA, 1994). In 1994, in the Little Scioto River, an upstream

background location had DELT anomalies in only 1.6 percent of the fish population. A substantial increase in DELT anomalies, with values ranging from 9.9 to 22.3 percent, occurred at downstream locations where high levels of PAHs were detected in the sediment (Ohio EPA, 1994). Some fish from these downstream locations were also found to have chemicals in their tissues that indicated they were exposed to high levels of PAHs. The high levels of chemicals in fish tissues, the significant increases in DELT anomalies in fish, and the high levels of PAHs in the sediment were found in the section of the Little Scioto River site contaminated by Baker Wood Creosoting (Ohio EPA, 1994).

DELT anomalies and sediment were again investigated in 2007 and the new data indicated that fish communities made a substantial improvement in the areas where contaminated sediments were removed (from Holland Road to just upstream of State Route 95) (Ohio EPA, 2008b). However, there are still elevated numbers of fish with DELT anomalies in downstream locations with contaminated sediments. With high levels of PAHs in sediments and with a high percentage of fish with DELT anomalies in the lower section of the Little Scioto River, there is still a concern that people may ingest fish with tumors. These fish have likely lived in contaminated waterways and would have elevated levels of PAHs or their metabolites in their tissues (Baumann and Black, 1991).

The ODH, ODNR, and Ohio EPA have issued the "Do Not Eat" fish advisory for the Little Scioto River site from 1992 to the present. The low number of fish in this section of the river has likely limited the opportunities for residents to catch fish let alone eat large amounts of contaminated fish. Due to the reduced fish populations, the PAH metabolites are not found in fish tissues normally eaten by fishermen, the high percentage of fish in the contaminated section of river having DELT anomalies, and the "Do Not Eat" fish advisory, the risk of exposure is expected to be low. Therefore, stomach cancers are not expected to be significantly above the incidence rates for the State of Ohio and the United States. In fact, stomach cancer rates for Marion County are below Ohio and U.S. expected rates (ODH, 2008). Also, since PAHs can be quickly metabolized by fish, contaminants in fish are not expected to reach high concentrations in edible fish tissue. Laboratory test results of common carp and white sucker fish tissue samples did not detect the presence of semivolatile organic compounds—only one northern pike sample had detects of PAHs (Ohio EPA, 1994).

Although not very soluble in water, PAHs have been detected in groundwater at other sites either as a result of migration directly from contaminated surface water or through the soil (ATSDR, 1995). Five residential wells were sampled during the Expanded Site Inspection (ESI) in 2007 and five public water supply wells were sampled during Site Inspection in 2006 for the Little Scioto River site. No PAHs were detected in these groundwater samples and all inorganic (metals) were below levels of concern (MCLs) (Ohio EPA, 2008b and Ohio EPA, 2007). The Ohio American Water Company wells are east and west of the Little Scioto River and north of Holland Road. The closest public well is approximately 2,700 feet north of the nearest known creosote contamination (Ohio EPA, 2008b). The closest residential well is approximately 600 feet from the nearest known creosote contamination (Ohio EPA, 2008b).

In 2001, the USEPA installed five shallow groundwater monitoring wells around Baker Wood Creosoting site, and in 2002, another four monitoring wells were installed in the deeper

limestone bedrock due to the shallow depth to bedrock and the concern that contaminants may have reached the bedrock. In 2003, the Expanded Site Inspection for Baker Wood Creosoting site concluded that the Baker Wood Creosoting site no longer poses a threat to the Little Scioto River site (Ohio EPA, 2008b). The ESI further stated, "Groundwater analysis in the vicinity of the creosote treatment area indicated that creosote contaminants from the site operations were present at relatively low concentrations in the shallow groundwater at the site" (Ohio EPA, 2008b).

There are some concerns that the sand and gravel deposits encountered during the previous cleanup of river sediments may provide a conduit for the groundwater transport of contaminants to private wells. However, given PAH's low solubility in water and tendency to attach to soil particles, it is unlikely there has been much contaminant groundwater migration toward residential wells. There is also a concern that fractures in the limestone bedrock may provide a conduit for the transport of contaminants toward public wells from the Baker Wood Creosoting site. Current water quality data indicates no additional risk of health effects from drinking water from the public water supply. However, additional groundwater data is needed to assess these potential future risk pathways. The USEPA will investigate these potential pathways during the Remedial Investigation.

COMMUNITY HEALTH CONCERNS

In 1997, a school nurse compiled a list of 15 cancer cases in graduates of River Valley High School on the east side of Marion. When it was brought to the attention of the ODH and the Ohio EPA, an environmental investigation was initiated by the Ohio EPA, focusing on identifying potential sources of environmental contamination in the Marion area. The Baker Wood Creosoting site and the contaminated sediments in the Little Scioto River were initially identified as potential contaminant sources; however, they were later determined not to be linked to the River Valley Schools cancer cases. A new middle and high school was built at another location as a result of the environmental investigation and contamination adjacent to the old school, a former Army Depot, was removed or capped in place.

Currently, much of the river bank is private property and access to the river is limited such that the river evidently is not heavily used by residents for recreation or fishing. According to USEPA's Community Involvement Plan for the site, there is not a lot of concern from residents and local officials about the Little Scioto River site. Some of the area residents interviewed stated that though the area is not used and the river has always been "dirty." (USEPA, 2009b).

Local officials that have interacted with the Ohio EPA over the years have remained concerned and involved in the ongoing investigation and cleanup processes at the site, which has been in the public eye for over 10 years. There are other interested parties that believe the poor health of this river detracts from their community (Ohio EPA, personal communication, 2012). The president of the Marion Area Chamber of Commerce commented: "…the business community is pleased that this project is in the USEPA Superfund program and look forward to the day that the area can be returned to a viable community resource" (Pam Hall, personal communication, 2012).

HEALTH OUTCOME DATA

Specific health outcome data is not available for the area around the Little Scioto River site; however, cancer incidence rates are available for Marion County. The Marion County cancer incidence rate for all cancer sites/types combined is lower than the rates for the State of Ohio and the United States (ODH, 2008). For cancers associated with exposure to PAHs through ingestion of contaminated fish or groundwater, stomach cancer incidence rates, are lower in Marion County than the rates in the State of Ohio and the United States (ODH, 2008). For cancers associated with exposures to PAHs through skin contact through wading and swimming, skin cancer incidence rates in Marion County fell slightly above rates in the State of Ohio and slightly below rates for the United States (ODH, 2008). Without site specific exposure data, we are not able to make an approximate identification of the exposed population and therefore cannot separate it from the unexposed population. Also, the number of people exposed is expected to have been too small in number compared to the expected rate of disease, so that it will not provide useful interpretation.

CHILD HEALTH CONSIDERATIONS

Both the ATSDR and the HAS recognize the unique vulnerabilities of children exposed to environmental contamination and hazards. As part of this public health assessment, the HAS considered the greater sensitivity of the children who live in the area of the Little Scioto River site when drawing conclusions and making recommendations regarding health effects from exposure to chemicals related to the Little Scioto River site.

CONCLUSIONS

1. The HAS concludes that frequent contact with PAHs in sediment via swimming or wading in the Little Scioto River for more than a year could harm people's health. This is currently a public health hazard. People coming into frequent contact with the sediments contaminated with elevated levels of PAHs could have an increased risk of developing certain types of skin cancer. The contaminants found in the sediment are at levels of health concern. Although the likelihood of prolonged exposure to these compounds in the Little Scioto River sediments may be low, as a public health agency, the HAS considers it prudent to reduce or eliminate skin contact with PAH contaminated sediments.

2. The HAS concludes that frequently eating fish contaminated with PAHs from sediments from the Little Scioto River site for more than a year is not likely to harm people's health. Due to the "*No Fishing*" signs and the anomalies, it is not likely that people are eating many fish from this area. Although, the likelihood of prolonged exposure to elevated levels of PAHs from eating fish caught in the Little Scioto River may be low, the HAS considers it prudent to reduce or eliminate the amount of fish eaten that are caught in areas of PAH contaminated sediments in the Little Scioto River.

3. The HAS cannot conclude whether people drinking well water or surface water contaminated with PAHs from the Little Scioto River site over the course a lifetime could harm people's health in the future. It is not certain whether untested private wells are impacted by the Little

Scioto River site or if contaminant levels will increase in the future. Data from public water supplies for groundwater and surface water indicate that current the levels of site-related contaminants are not a health concern.

RECOMMENDATIONS

1. Isolate and contain or remove the highly contaminated sediments that pose a threat to exposure via dermal contact and/or through people eating fish contaminated with PAHs.

2. Further investigate the threat of contamination of the public and residential water supply wells via the groundwater pathway.

3. Collect downstream surface water samples at any nearby water intakes to assess any potential contamination.

4. Continue to monitor fish and biota to determine if the "Do Not Eat" fish advisory should be modified to reflect current conditions.

5. Re-evaluate the "Do Not Wade or Swim" advisory after the site is remediated.

6. Determine whether contaminants from the Rockswale Ditch or the Baker Wood Creosoting site have the potential to re-contaminate downstream portions of the Little Scioto River that may be cleaned-up.

PUBLIC HEALTH ACTION PLAN

Completed Actions

The ODH in cooperation with the Ohio EPA and ODNR issued a "Do Not Eat" fish advisory and a "No Swimming or Wading" advisory since 1992 for the Little Scioto River site.

In September 2000, the HAS completed the Public Health Consultation for the Baker Wood Creosoting, Marion, Marion County, Ohio.

The USEPA conducted a time-critical removal action at the Baker Wood Creosoting site in 2000, eventually removing 6,565 tons of creosote-contaminated soil off site for disposal. The USEPA removed contaminated sediments in the upper section of Little Scioto River site and North Rockswale Ditch in 2002 and 2003. The USEPA listed the Little Scioto River site on the NPL in September 2009.

The Ohio EPA completed a SI and an ESI for the Little Scioto River site in 2007 and 2008.

The Little Scioto River site was placed on the USEPA NPL of Superfund hazardous waste sites in September 2009.

Ongoing Actions

The USEPA Superfund program is currently conducting a RI/FS on both the Little Scioto River site and the Baker Wood Creosoting site.

The ODH, in conjunction with the Ohio EPA and ODNR, continues the "Do Not Eat" fish and "Do Not Wade or Swim" advisories for the contaminated section of the Little Scioto River.

The HAS is providing fact sheets for PAHs, Exposure to Toxic Chemicals, and Cancer in the Appendix of this document (Appendix C) and on the ODH web page http://www.odh.ohio.gov/odhPrograms/eh/hlth_as/chemfs1.aspx .

Planned Actions

Concurrent with the Remedial Investigation (RI), the USEPA will begin a Feasibility Study (FS) for the evaluation of remedial action alternatives to address the remaining site contaminants. A remedy will be selected after the FS and the review of all remedial alternatives.

The ODH, in conjunction with the Ohio EPA and ODNR, will review new environmental data after contaminated sediments have been removed or isolated to determine if the "Do Not Eat" fish and "Do Not Wade or Swim" advisories should be removed and if swimming, wading, and eating fish continues to pose a health concern. It is reported that area residents do not currently use the site for fishing, swimming, or wading.

The HAS will evaluate additional data as it becomes available to determine if there are any other potential health threats.

REPORT PREPARATION

This Public Health Assessment/Health Consultation for the Little Scioto River Site was prepared by the Ohio Department of Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

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TABLES

Table 1. Little Scioto River Site - Carcinogenic PAHs in Contaminated Sediment

Carcinogenic PAHs	*Range* (ppm)*	*Comparison Value (ppm)*	*Comparison Value Reference*	*EPA Cancer Class*	*Background Levels (ppm)*
Benzo(a)anthracene[1,2,4]	1.47 – 214	0.15	U.S. EPA RSL	B2	<0.146
Benzo(b)fluoranthene[1,3,4]	1.3 – 179	0.15	U.S. EPA RSL	B2	<0.146
Benzo(a)pyrene[1,2,4]	1.24 – 176	0.1	ATSDR CREG	B2	0.149
Chrysene[1,4]	1.68 – 314	15	U.S. EPA RSL	B2	<0.146
Dibenzo(a,h)anthracene[1,4]	0.19 – 44.6	0.015	U.S. EPA RSL	B2	<0.146
Indeno[1,2,3-c,d]pyrene[1,4]	0.734 – 89.8	0.15	U.S. EPA RSL	B2	<0.146
Benzo[k]fluoranthene[3,4]	0.965 – 164	1.5	U.S. EPA RSL	B2	0.160

Source: Ohio EPA Expanded Site Inspection Report 2008

ppm = parts per million
PAHs = polycyclic aromatic hydrocarbons
RSL = Regional Screening Level (U.S. EPA November 2011)
CREG = Cancer Risk Evaluation Guide (ATSDR)

[1]The Department of Health and Human Services (DHHS) has determined that these PAHs are known animal carcinogens.
[2]The International Agency for Research on Cancer (IARC) has determined that these PAHs are probable human carcinogens.
[3]The International Agency for Research on Cancer (IARC) has determined that these PAHs are possible human carcinogens.
[4]The USEPA has determined that these PAHs are probable human carcinogens.
*Range includes the following sediment sample locations on the Little Scioto River: ELSR-SE-03, ELSR-SE-05, ELSR-SE-06, ELSR-SE-07, ELSR-SE-09, ELSR-SE-10, ELSR-SE-11, and ELSR-SE-22 (Ohio EPA, 2008b).

Table 2. Toxicity Equivalent Benzo(a)pyrene Concentration in Sediment

Carcinogenic PAHs	*Toxicity Equivalency Factor*[a]	*Maximum Detected Concentration (ppm)*	*Equivalent B(a)P Concentration (ppm)*
Benzo(a)anthracene	0.1	214	21.4
Benzo(b)fluoranthene	0.1	179	17.9
Benzo(a)pyrene	1	176	176
Chrysene	0.01	314	3.1
Dibenzo(a,h)anthracene	5	44.6	223
Indeno(1,2,3-cd)pyrene	0.1	89.8	9.0
Benzo(k)fluoranthene	0.1	164	16.4
Total Benzo(a)pyrene Equivalent Concentration			**467**

Source: Ohio EPA 2008

ppm = parts per million

[a] ATSDR Toxicological Profile for Polycyclic Aromatic Hydrocarbons, August 1995.

Equation for Total Toxicity Equivalency Factor Concentration:

$$TEQ = \sum [C_i] \times TEF_i$$

TEQ = Toxicity Equivalent of a mixture
C_i = concentration of individual compound
TEF_i = relative potency (as based on carcinogenicity) to benzo(a)pyrene

Example:

$$TEQ = (5 \times 44.6\ ppm) + (0.1 \times 179\ ppm) + (0.1 \times 214\ ppm) + (1 \times 176\ ppm) + (0.1 \times 89.8\ ppm) + (0.01 \times 314\ ppm) + (0.1 \times 164\ ppm) = 467\ ppm$$

Table 3. Little Scioto River - Exposure Pathways						
Pathway Name	**Exposure Pathway Elements**					**Time Frame for Exposure**
	Sources of Contamination	**Environmental Medium**	**Points of Exposure**	**Routes of Exposure**	**Potentially Exposed Population**	
Completed Pathways						
Sediment Waders & Swimmers	Contaminated sediments in **lower** LSR	Sediment	Wading & Swimming in LSR	Dermal Contact & Incidental Ingestion	Waders & Swimmers	Past Current Future
Food Chain Fish	Contaminated sediments in **lower** LSR	Fish	Fishing	Ingestion	Recreational & Subsistence Fishers	Past Current Future
Potential Pathways						
Groundwater Nearby Private Wells	Sand lenses intersecting LSR sediments	Groundwater	Private Wells	Ingestion	Residents with private wells near the site	Current Future
Surface Water	Disturbed Sediment in **lower** LSR	Surface Water	Surface Water Intakes	Ingestion	Downstream Residents using surface water	Past Current Future
Eliminated Pathways						
Surface & Groundwater	Baker Woods & Sediments in **upper** LSR	Surface Water & Groundwater	Marion Municipal Water Supplies	Ingestion	Marion residents	Past
Sediment Waders & Swimmers	Contaminated sediments in **upper** LSR	Sediment	Wading & Swimming in LSR	Dermal Contact & Incidental Ingestion	Waders & Swimmers	Past

FIGURES

Figure 1. Little Scioto River ESI Site Location Map

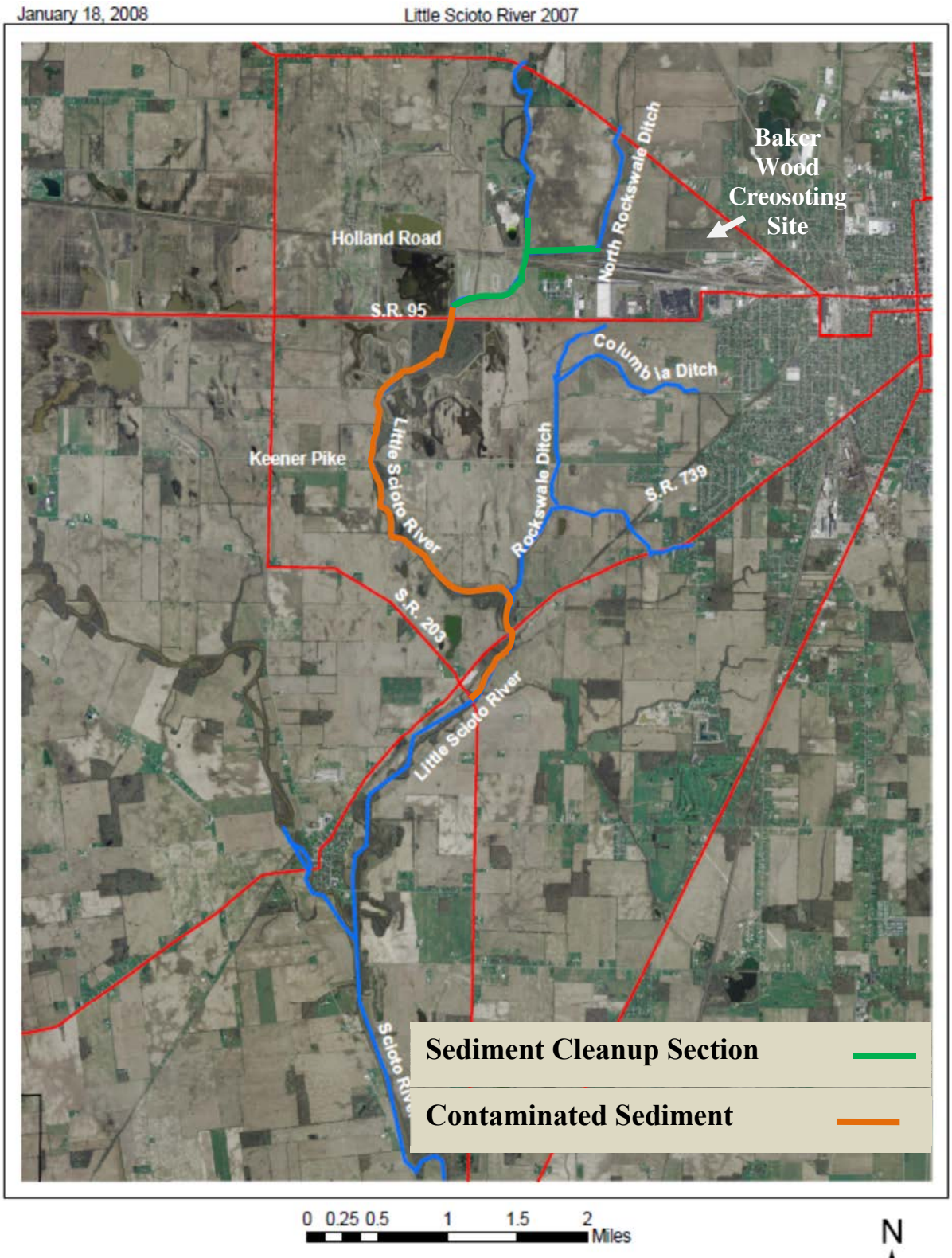

Figure 2. Little Scioto River & Tributaries & Baker Wood Creosoting site: from Ohio EPA ESI 2008

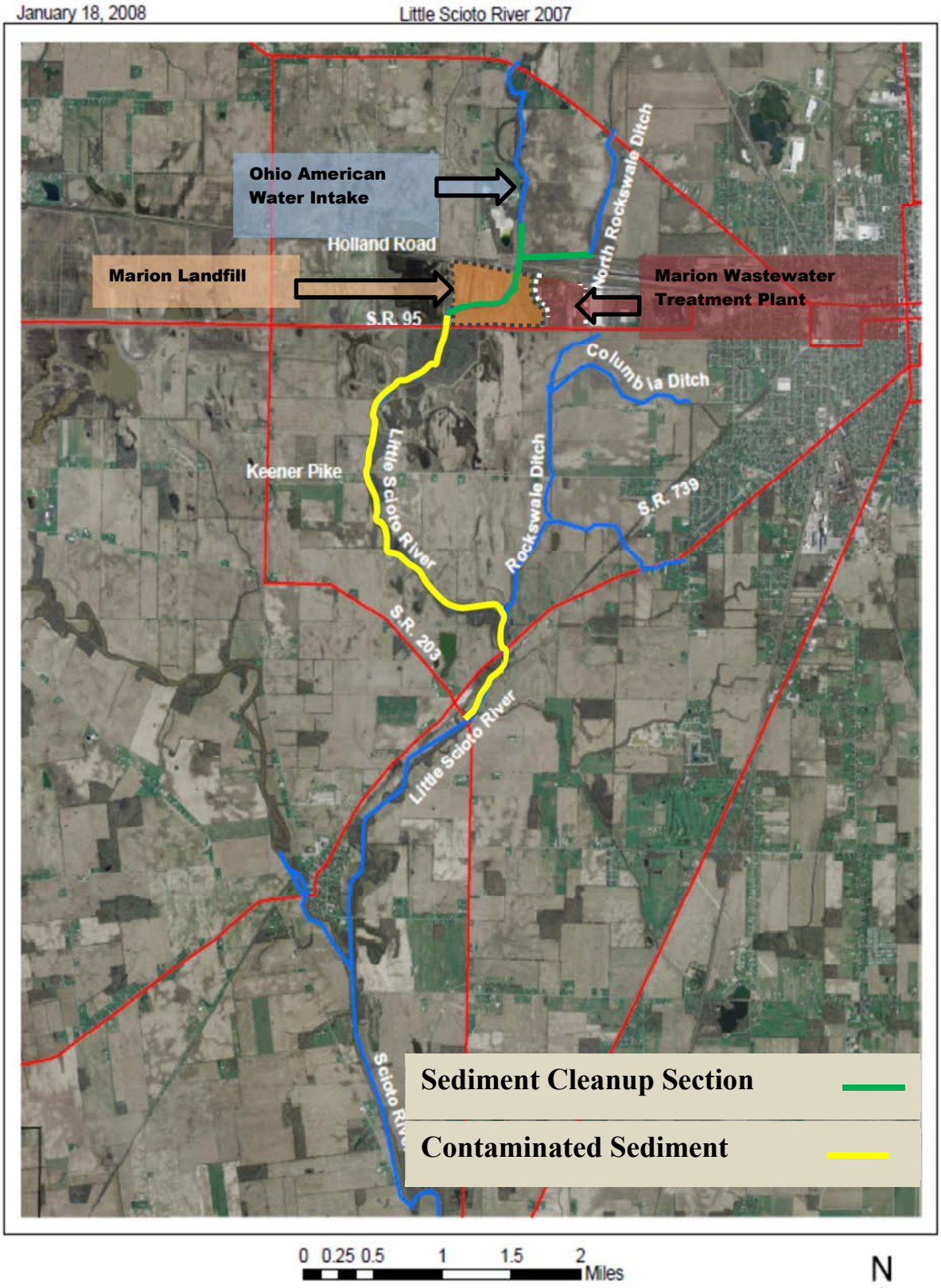

Figure 3. Location of Wastewater Treatment Plant, Landfill and Water Intake

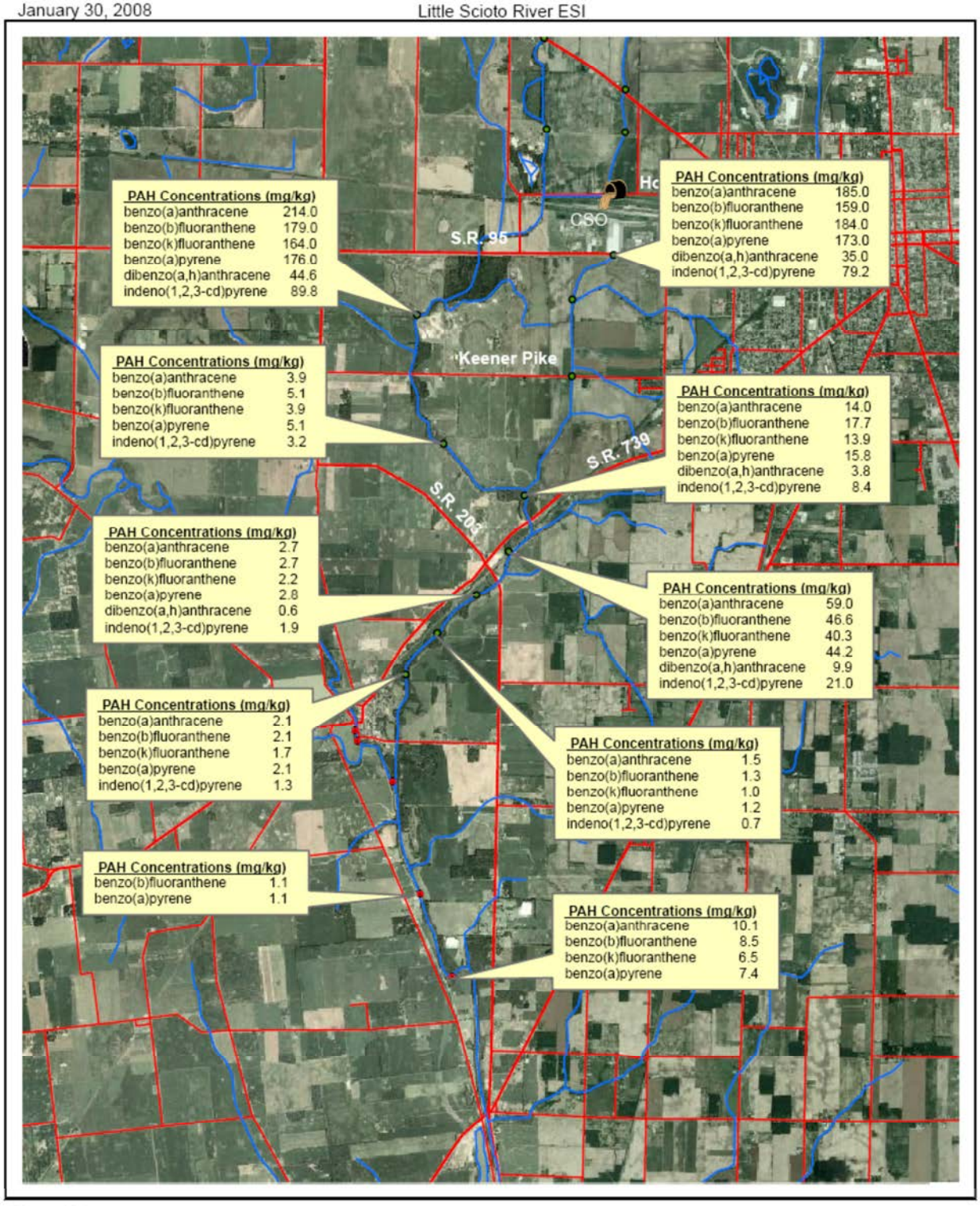

Figure 4. Little Scioto River & Scioto River Carcinogenic PAH Concentrations in Sediment Samples

APPENDICES

Appendix A. Estimates of Exposure Doses and Theoretical Cancer Risks
(Little Scioto River site)

The Health Assessment Section (HAS) of the Ohio Department of Health (ODH) evaluated exposures to Polycyclic Aromatic Hydrocarbons (PAHs) at the Little Scioto River site in Marion, Ohio. For the evaluation of exposures to PAHs in sediment, benzo(a)pyrene (B(a)P) was used as a surrogate to assess the relative toxicity of seven carcinogenic PAHs (cPAHs) classified by the U.S. EPA as probable human carcinogens. To determine the toxicity of the mixture of PAHs, the maximum detected concentration of each cPAH was multiplied by a Toxic Equivalency Factor (TEF) which relates its toxicity to that of B(a)P. The maximum PAH concentrations were found in sediment sample ELSR-SE-03, which was collected in the Little Scioto River downstream of the 2006 sediment removal area, approximately half way between State Route 95 and Keener Pike (Ohio EPA 2008). The sum of the weighted concentrations, the benzo(a)pyrene toxicity equivalent concentration, was calculated to be 467 parts per million (ppm) (see the table at the end of this appendix). Exposure doses and estimated theoretical cancer risks were calculated for skin contact (dermal exposure) with PAH-contaminated sediment and for inadvertent ingestion of sediment. For fish ingestion, exposure doses and cancer risks were estimated using more recent fish tissue data from the U.S. EPA, where 50 samples were collected during their remedial investigation of the Little Scioto River in 2010 (personal communication, US. EPA, June 2012).

Exposure to a cancer-causing compound, even at low concentrations, is assumed to be associated with some increased risk. The estimated theoretical cancer risk from exposure to contaminants associated with this site was calculated by multiplying the estimated exposure dose for each age group with the Cancer Slope Factor (CSF) for benzo(a)pyrene. This calculation estimates the theoretical excess cancer risk expressed as a portion of the population that may be affected by a carcinogen during a lifetime of exposure. An estimated risk of 1×10^{-6} predicts the probability of one additional cancer, over background, in an exposed population of 1 million. An increase in the lifetime cancer risk is not an estimate of expected additional cancer cases. Rather, it is an estimate of the increase in the probability that a person may develop cancer sometime in his or her lifetime following exposure to a particular contaminant.

Because of conservative safety factors used to calculate the CSFs, using these values provides only a theoretical estimate of risk; the true or actual risk is unknown and could be close to zero. Risk estimates are generated using mathematical models applied to epidemiologic or experimental data for carcinogenic effects. These models extrapolate from higher experimental doses to lower experimental doses. Often, even the lower experimental doses represent exposures to chemicals that are at concentrations orders of magnitude higher than the concentrations found in the environment. These models also assume that there are no thresholds to carcinogenic effects; a single molecule of a carcinogen is assumed to be capable of causing cancer. The doses associated with these estimated hypothetical risks may be orders of magnitude lower than doses reported in toxicology literature to cause carcinogenic effects. A low cancer risk estimate (less than 10^{-6}= less than 1 in one million) may indicate that the toxicology literature support a finding that no excess cancer risk is likely. A cancer risk estimate (greater than 10^{-6} = greater than 1 in one million), however, indicates that a careful review of toxicology literature before making conclusions about cancer risks is in order.

DERMAL CONTACT TO SEDIMENT
EXPOSURE DOSES AND ESTIMATED THEORETICAL CANCER RISK

1. Assumptions, Limitations, and Default Values Used in Calculations

Appendix A Table 1 - Standard Default Dermal Exposure Values

Age (years)	Body Weight (kg)	Soil Adherence Concentration	Feet[a] (cm^2)	Legs[a] (cm^2)	Hands[a] (cm^2)	Total Exposed Area[a] (cm^2)	Total Soil Adhered[a] (mg)
1-11	30	0.2	620	2401	455	3478	696
12-17	50	0.2	1166	4956	807	6930	1386
18-70	70	0.07	1344	6062	1008	8415	589

Source: EPA 2001; EPA 1997

[a] default values from ATSDR Public Health Assessment Guidance Manual 2005. Total soil adhered (A) is estimated by multiplying the exposed area by the default soil adherence concentration of 0.07 mg/cm^2 for adults and 0.2 mg/cm^2 for children.
kg = kilogram
cm^2 = square centimeter
mg = milligram

Limitations of applying dermal toxicological data to site-specific scenarios:

- Much of the data depends on animal studies with repeated application of relatively high doses of pure substances directly to the skin of the test animal
- This data may not be directly applicable to short or infrequent periods of human contact with soil or sediment

Assumptions:

- Exposed Skin Areas limited to feet, legs, hands
- Daily Exposure During 3 Summer Months - 90 days/year exposure
- Bioavailability Factor = 0.1
- 0-1 age group was not evaluated because dermal contact exposure to sediment is not expected to occur for this age group.

2. Calculated Exposure Doses and Estimated Theoretical Cancer Risks

Appendix A Table 2 - Sediment Dermal Contact Exposure Doses and Estimated Theoretical Cancer Risk								
Age (years)	**Body Weight (kg)**	**Exposed Skin Area (cm^2)**	**Sediment Adhered (mg/cm^2)**	**Total Sediment Adhered (mg)**	**Exposure Dose (mg/kg/day)**	**Daily Intake (mg/day)**	**Exposure Duration (years)**	**Estimated Theoretical Cancer Risk***
1-11	30	3478	0.2	696	2.67×10^{-4}	8.01×10^{-3}	12	3.34×10^{-4}
12-17	50	6930	0.2	1386	3.19×10^{-4}	1.60×10^{-2}	6	2.00×10^{-4}
18-70	70	8415	0.07	589	9.69×10^{-5}	6.78×10^{-3}	30	3.03×10^{-4}

kg = kilogram
cm^2 = square centimeter
mg = milligram

* Theoretical cancer risk can be defined as the number of additional cases of cancer in a population due to exposure to a toxic substance, usually written as a negative power of 10. For example, one additional case of cancer per 10,000 individuals is written as 1×10^{-4}. The estimated cancer risks are compared to guidelines for cancer risk as suggested by the U.S. EPA which range from 1×10^{-6} to 1×10^{-4}.

The estimated theoretical cancer risks for the Little Scioto River site tabulated above are low but greater than 10^{-4} or 1 in 10,000 – the high end of the range of cancer risk guidelines suggested by the U.S. EPA. The actual risk is likely to be less when considering that exposure is likely to be less than a daily exposure throughout all summer months at the highest concentration.

3. **Equation for Estimated Exposure Dose from Dermal Contact with Sediment*:**

$$dose = \frac{concentration\ x\ sediment\ adhered\ x\ bioavailability\ x\ exposure\ factor\ x\ conversion\ factor}{body\ weight}$$

dose	= estimated exposure dose (mg/kg/day)
concentration	= contaminant concentration (mg/kg)
sediment adhered	= total sediment adhered (mg)
bioavailability	= bioavailability factor (unitless)
exposure factor	= exposure factor (unitless)
conversion factor	= conversion factor (10^{-6} kg/mg)
body weight	= body weight (kg)

Example:

$$dose = \frac{\frac{467mg}{kg}\ x\ 696\ mg\ x\ 0.1\ x\ (\frac{90}{365})\ x\ 10^{-6}\ \frac{kg}{mg}}{30\ kg} = 2.67\ x\ 10^{-4}\ mg/kg/day$$

4. **Equation for Cancer Risk*;**

Cancer Risk = Exposure Dose x Cancer Slope Factor

Cancer Risk	= estimated theoretical cancer risk (unitless)
Exposure Dose	= estimated exposure dose (mg/kg/day)
Cancer Slope Factor	= cancer slope factor $(mg/kg/day)^{-1}$; 7.3 $(mg/kg/day)^{-1}$ for B(a)P

*Equations from ATSDR Public Health Assessment Guidance Manual 2005. Contaminant concentration was derived from ELSR-SE-03, the sediment sample location on the Little Scioto River with the maximum PAH concentrations (OHIO EPA 2008), converted to a benzo(a)pyrene toxicity equivalent concentration.

INADVERTENT INGESTION OF CONTAMINANTS IN SEDIMENT
EXPOSURE DOSES AND ESTIMATED THEORETICAL CANCER RISK

The estimated exposure doses for inadvertent ingestion of contaminants in sediment were calculated using the maximum detected concentrations of carcinogenic PAHs adjusted to benzo(a)pyrene toxicity using Toxicity Equivalent Factor (TEF) (see Table 2, page 33).

1. Default Values Used in Calculations

Appendix A Table 3 - Standard Default Values	
Body Weight (BW):	
70 kilogram	Adult, approximate average
16 kilogram	Children 1 through 6 years old, 50th percentile
10 kilogram	Infant (6 to 11 months) approximate average
Exposure Duration (ED):	
70 years	Lifetime; by convention
30 years	National upper-bound time (90th percentile) at one residence
9 years	National median time (50th percentile) at one residence
6 years	Children 1 through 6 years old

Appendix A Table 4 - Default Soil / Sediment Intake Rates	
100 milligrams/day	Adult, average soil ingestion rate
200 milligrams/day	Child, average soil ingestion rate

2. Calculated Exposure Doses and Estimated Theoretical Cancer Risks

Appendix A Table 5 - Sediment Inadvertent Ingestion Exposure Doses and Estimated Theoretical Cancer Risk

Age (years)	Body Weight (kg)	Intake Rate (mg/day)	Exposure Dose (mg/kg/day)	Daily Intake (mg/day)	Exposure Duration (years)	Estimated Theoretical Cancer Risk*
1-6 child	16	200	1.44×10^{-3}	2.30×10^{-2}	6	9.00×10^{-4}
18-70 adult	70	100	1.65×10^{-4}	1.15×10^{-2}	30	5.15×10^{-4}

*A cancer risk estimate greater than 10^{-4} (1 in one 10,000) is above acceptable cancer risk guidelines defined by the U.S. EPA. The actual risk is likely to be less, because conservative assumptions and maximum concentrations were used to estimate cancer risk.

kg = kilogram
mg = milligram

3. **Equation for Sediment Inadvertent Ingestion Exposure Dose*;**

$$dose = \frac{concentration\ x\ intake\ rate\ x\ exposure\ factor\ x\ conversion\ factor}{body\ weight}$$

dose	= estimated exposure dose (mg/kg/day)
concentration	= contaminant concentration (mg/kg)
intake rate	= intake rate of sediment (mg/day)
exposure factor	= exposure factor (unitless)
conversion factor	= conversion factor (10^{-6} kg/mg)
body weight	= body weight (kg)

Example:

$$dose = \frac{\frac{467mg}{kg} x \frac{200\ mg}{day} x \left(\frac{90}{365}\right) x\ (10^{-6}\ kg/mg)}{16\ kg} = 1.44\ x\ 10^{-3} \frac{mg}{kg}/day$$

*Equations from ATSDR Public Health Assessment Guidance Manual 2005. Contaminant concentration was derived from ELSR-SE-03, the sediment sample location on the Little Scioto River with the maximum PAH concentrations (from *Expanded Site Inspection Report Little Scioto River*, Ohio EPA 2008), converted to a benzo(a)pyrene toxicity equivalent concentration.

FISH INGESTION
EXPOSURE DOSES AND ESTIMATED THEORETICAL CANCER RISK

Benzo(a)pyrene [B(a)P] is seldom found in fish tissue samples due to the ability of fish to rapidly metabolize and eliminate PAHs. Ohio EPA typically analyzes bile extracted from fish gall bladders for PAH metabolites to determine if the fish are being impacted. In 2007, Ohio EPA collected fish bile from white sucker and common carp at five locations in the Little Scioto River. The bile metabolite data from fish samples collected by the Ohio EPA in 2007 indicate that common carp and white sucker have been exposed to benzo(a)pyrene and naphthalene. The highest median concentration of the B(a)P metabolite detected in fish bile was 11 ng/ug or 11,000 mg/kg (Ohio EPA 2008a). ODH and ATSDR reviewed the fish analytical data from the Ohio EPA and determined that they were not adequate to make a reliable estimate of potential health effects from eating fish from the Little Scioto River, because the sample results focused on fish bile and not the edible portions of fish. It is unlikely that fishers are eating fish bile or fish tissue with contaminant concentrations equal to those found in the fish bile.

In 1992, the Ohio EPA collected whole body fish samples from the Little Scioto River. At that time, all common carp and white sucker test results (nine samples) were reported as not detected for semivolatile organic compounds, including PAHs. One northern pike sample in the Scioto River upstream from the Little Scioto confluence had detectable concentrations of benzo(b)fluoranthene at 0.160 ppm and chrysene at 0.170 ppm, reported as estimated values (Ohio EPA 1994).

In October 2010, the U.S. EPA collected 50 fish samples (25 fish fillet and 25 fish carcass) from the Little Scioto River as part of its remedial investigation. The fish tissue samples were analyzed for semivolatile organic compounds (including PAHs), metals, and lipid content by a subcontracted laboratory. Results for the seven carcinogenic PAHs are summarized in the table below using the data received from the U.S. EPA (personal communication, U.S. EPA, June 2012). The total toxicity of the mixture of PAHs was determined using the average detected concentration of each cPAH and its respective Toxic Equivalency Factor (TEF) which relates its toxicity to that of B(a)P. The total benzo(a)pyrene equivalent concentration in fish was calculated to be 0.0210 ppm. An exposure dose can then be estimated, along with its associated theoretical cancer risk using the calculations given below. In summary, the estimated theoretical cancer risk from eating PAH-contaminated fish from the Little Scioto River is very low. The actual risk is also likely to be very low, because of the low sport fish population and the “do not eat” fish advisory for this section of the river.

Appendix A Table 6 – Toxicity Equivalent Benzo(a)pyrene Concentration in Fish					
Contaminants	**Toxicity Equivalency Factor[a]**	**Minimum Detected Concentration (ppm)**	**Maximum Detected Concentration (ppm)**	**Average Detected Concentration (ppm)**	**Equivalent Concentration of B(a)P (ppm)**
Benzo(a)anthracene	0.1	0.0019	0.0286	0.0073	0.00073
Benzo(b)fluoranthene	0.1	0.0017	0.0219	0.0083	0.00083
Benzo(a)pyrene	1	0.0021	0.0182	0.0072	0.0072
Chrysene	0.01	0.0021	0.0536	0.0105	0.000105
Dibenzo(a,h)anthracene	5	0.0018	0.0025	0.0022	0.011
Indeno(1,2,3-cd)pyrene	0.1	0.0017	0.0078	0.0039	0.00039
Benzo(k)fluoranthene	0.1	0.002	0.0198	0.0079	0.00079
Total Benzo(a)pyrene Equivalent Concentration in Fish (ppm)					**0.0210**

Source: U.S. EPA 2012

[a] ATSDR Toxicological Profile for Polycyclic Aromatic Hydrocarbons, August 1995.

1. Calculated Exposure Doses

$$dose = \frac{concentration\ x\ intake\ rate\ x\ bioavailability\ factor\ x\ exposure\ factor\ x\ conversion\ factor}{body\ weight}$$

dose = estimated exposure dose (mg/kg/day)
concentration = contaminant concentration (mg/kg)
intake rate = intake rate of contaminated medium (mg/day)
bioavailability factor = bioavailability factor (unitless)
exposure factor = exposure factor (unitless)
conversion factor = conversion factor (10^{-6} kg/mg)
body weight = body weight (kg)

Example:

$$dose = \frac{0.021\ \frac{mg}{kg}\ x\ 20{,}100\ mg/day\ x\ 0.1\ x\ 1.0\ x\ (10^{-6}\ kg/mg)}{70\ kg} = 6.0\ x\ 10^{-7}\ \frac{mg}{kg}/day$$

kg = kilogram
mg = milligram
*Equations from ATSDR Public Health Assessment Guidance Manual 2005.

2. Estimated Theoretical Cancer Risks

Cancer Risk = Exposure Dose x Cancer Slope Factor

Cancer Risk = estimated theoretical cancer risk (unitless)
Exposure Dose = estimated exposure dose (mg/kg/day)
Cancer Slope Factor = cancer slope factor $(mg/kg/day)^{-1}$; 7.3 $(mg/kg/day)^{-1}$ for B(a)P

Example:

Cancer Risk = 6.0 x 10^{-7} mg/kg/day x 7.3 $(mg/kg/day)^{-1}$ = 4.4 x 10^{-6}

Appendix B. Response to Agency and Public Comments

Government agencies and the general public were asked to review this Public Health Assessment for the Little Scioto River site and provide comments and questions. The Initial/Public Comment Release, dated December 5, 2011, was made available for public comment until January 18, 2012. The document was available for public review on the Ohio Department of Health web page at http://www.odh.ohio.gov. Copies of the assessment were also available at the Marion Public Library, 445 E. Church St., Marion, OH 43302.

Comments were received from the U.S. Environmental Protection Agency (USEPA) Remedial Project Manager (RPM) for the Little Scioto River site, from the Ohio EPA Northwest District Office Site Coordinator, and from the Director of Environmental Health of the Marion Public Health Department. No comments or questions were received from the community regarding suspected exposures or health effects from exposures from the Little Scioto River site. However, the president of the local chamber of commerce expressed the business community's support of USEPA's Superfund program.

Comments from USEPA RPM:

1. Page 27, rewrite first sentence (Planned Actions Section): Concurrent with the RI, the USEPA will begin a FS for the evaluation of remedial action alternatives to address the remaining site contaminants.The FS always includes a "no action alternative." US/EPA/Superfund cannot preselect a remedy. The remedy will be selected after the FS and the review of all remedial alternatives.
   Response: The first sentence in Planned Actions was changed to read: "Concurrent with the Remedial Investigation (RI), the USEPA will begin a Feasibility Study (FS) for the evaluation of remedial action alternatives to address the remaining site contaminants. A remedy will be selected after the FS and the review of all remedial alternatives."

2. Other comments (12) were grammatical in nature.
   Response: Corrections to the text were adopted as suggested.

Comments from Ohio EPA:

1. I briefly reviewed the narrative sections of the report and have no comments of a technical nature. However, on page 24, last paragraph of section "Community Health Concerns", I believe the first sentence is not very accurate. Local officials that I have interacted with over the years have remained concerned and involved in the ongoing investigation and cleanup processes. This project has been in the public eye for over 10 years. There are other interested parties too that believe the poor health of this river detracts from their community. Pam Hall was the president of the local chamber of commerce and can provide you with more detailed information regarding community involvement. She can be reached at 740-382-2181.
   Response: Changes were made to the Community Health Concerns section of the PHA to better match the original wording found in USEPA's Community Involvement Plan (2009), which indicated "There is not a lot of concern from residents and local officials about the site." Steve Snyder's experience with local officials and interested parties indicates more

interest and involvement than that indicated by USEPA. This was added to the PHA as a personal communication.

Comments from Marion Public Health Department:

1. I just started reading through the report and asked the Ohio American Water Treatment Plant Superintendent, Scott Ballenger, if he draws water from the river in the contaminated area. He said they draw water before the contaminated site. Is this correct? It appears from what I have read so far, that the municipal water used to be potentially contaminated.
   Response: The closest public well is approximately 2,700 feet north and upstream of the nearest known creosote contamination (Ohio EPA Expanded Site Inspection Report, 2008). Mr. Ballenger is correct. A low head dam separates the part of the river that the OAWC obtains its water from and the contaminated portion of the river, which is downstream of the dam.

3. (Comment to Roger Baldinger) I was reviewing an old Case Study about the Baker Wood site and found that they stated there was a combined sanitary/storm sewer thought to be a direct link to the surface water contaminant migration pathway leading to the North Rockswale Ditch and that the drawings indicate the old sewer tie-ins from the facility may still be in use. Do you know if the tie ins are still there? The plant was at the Northwest corner of 309 and Holland Rd. One of the reports indicated that there is still minimal soil contamination on the site.
   Response from Roger Baldinger: After review with the personnel here at the Plant, it would be assumed that the taps on the combined sewer going out Holland Rd. are still there. EPA has done remediation on the Baker Woods site, but we do not know the extent of their project. Perhaps they disconnected/cut all laterals leaving the area? At the time of the EPA investigation, the atmospheric conditions in the Holland Rd. sewer would not allow the confined space entry to proceed.
   Response from ODH HAS: I think that Margaret Gielniewski, the U.S. EPA Remedial Project Manager for the site, would be better able to answer your question about the combined sanitary/storm sewer that you describe below and its current connection condition with regard to North Rockswale Ditch. The U.S. EPA has taken some additional groundwater and sediment samples as part of their remedial investigation, and they focused on pathways that may exist for contamination to move from Baker Wood toward the river through North Rockswale Ditch. We do not have those results at this time, but we would like to update our public health assessment with this information. (The U.S. EPA's RI report may not be available until the middle of 2012.) Her contact information is: Margaret Gielniewski (gielniewski.margaret@epa.gov)
   312-886-6244 or 800-621-8431, ext. 66244
   Response from Margaret Gielniewski: US EPA did not disconnect or cut laterals or tie-ins leaving the Baker Wood property. The taps on the combined sanitary/storm sewer going out Holland Road are still there.

   In 1999, US EPA performed a removal action at the Baker Wood site, excavating and disposing of nearly 5,500 tons of contaminated soil. During the excavation, clay tile segments that were encountered and were plugged with creosote were removed. These tiles were no longer able to fulfill their function of draining the property because they were

plugged with creosote.

In 2002, US EPA removed 40,000 cubic yards of contaminated sediment in the North Rockswale Ditch and the Little Scioto River.

From 2009-2011, US EPA investigated the drainage tiles on the Baker Wood site and found a parallel drainage tile that is connected to the sanitary/storm sewer. However, this tile is also not draining into the sewer due to tree and other plant roots blocking the pathway.

In 2010-2011, US EPA collected samples along the Little Scioto River, its tributaries, and at the Baker Wood site. We are in the process of analyzing those results. As John stated, we hope to have the Remedial Investigation report stating the findings of our sampling, completed by late 2012. Once issued, the report will be available for review at the Marion Public Library.

If you have any further questions, please do not hesitate to contact me or Howard Caine (caine.howard@epa.gov), the current project manager on the site.

Comments from Marion Area Chamber of Commerce:

1. I have no questions or concerns; my only comment would be that the business community is pleased that this project is in the USEPA Superfund program and look forward to the day that the area can be returned to a viable community resource.
   Response: Pam Hall's comments regarding the local community were added to the Community Health Concerns section of the PHA as a personal communication.

Appendix C. Fact Sheets

Bureau of Environmental Health
Health Assessment Section

"To protect and improve the health of all Ohioans"

Polycyclic Aromatic Hydrocarbons (PAHs)

What are Polycyclic Aromatic Hydrocarbons (PAHs)?

PAHs are a group of chemicals naturally found in coal, coal tars, oil, wood, tobacco and other organic materials. PAHs are released into the environment as the result of the incomplete burning of these materials.

There are more than 100 different PAHs. PAHs are the waxy solids found in asphalt, crude oil, coal, coal tar pitch, creosote and roofing tar. Some types of PAHs are used in medicines and to make dyes, plastics and pesticides.

PAHs are ubiquitous (are everywhere) throughout the world and can be found in every type of environment. Urban environments (cities) tend to have higher levels of PAHs due to the increased amounts of gas and oil burned as well as the increased use of asphalt and tars on roads and shingles on roofs.

What happens to PAHs when they enter the environment?

PAHs can enter the environment in the air from volcanoes, forest fires, residential wood burning and exhaust from cars and trucks.

In urban (city) environments, PAHs can enter creek and river sediments (soils) from water running off asphalt roads, parking lots and driveways. PAHs are also found in roofing shingles and tars and can run off roofs to be carried to downspouts and drainage systems during rain events.

Some of the PAHs are lighter (or a lower molecular weight) and can volatize (evaporate) into the air. These PAHs break down by reacting with sunlight and other chemicals in the air. This generally takes days to weeks. The more sunlight, the quicker these PAHs will breakdown. These lighter (low molecular weight) PAHs are less toxic to humans and <u>are not</u> carcinogenic (cancer causing).

Heavier (or a higher molecular weight) PAHs do not dissolve in water, but stick to solid particles and settle to the sediments in bottoms of lakes, rivers or streams. These "fat" PAHs stick to soils and sediments and will generally take weeks to months to break down in the environment. Microorganisms in soils and sediments are the main cause of breakdown. These heavy PAHs <u>are</u> carcinogenic (cancer causing) to lab animals and <u>may be</u> carcinogenic to humans.

How might I be exposed to PAHs?

For most of the U.S. population, the primary sources of exposure to PAHs are inhalation of compounds in tobacco smoke, wood smoke and the ambient (outside) air. Smoke may contain both light (vapors) and heavy (soot or ash) PAHs.

You may also be exposed to PAHs by incidental (minor or casual) contact to lake, river or creek sediments or by eating smoked or charbroiled foods.

Overall exposure to PAHs will increase if persons come in contact with PAHs in their workplace. PAHs have been found in industries such as coal tar production plants, smoke houses, coking plants, aluminum production plants, coal tarring facilities and municipal trash incinerators. Also, PAHs can be found in industries such as mining, oil refining, metalworking, chemical production, transportation and the electrical industry. PAHs have also been found in other facilities where petroleum and petroleum products are used or where coal, oil, wood or cellulose is burned.

PAHs are present throughout the environment and you may be exposed to these substances at home, outside or at the workplace. Typically, you will not be exposed to an individual PAH, but to a mixture of PAHs.

How do PAHs enter and leave my body?

PAHs can enter your body through your lungs when you breathe air. However, it is not known how rapidly or completely your lungs absorb PAHs.

PAHs can enter your body through drinking water and swallowing food, soil or dust particles that contain PAHs. But absorption is generally slow when PAHs are swallowed and generally you will not be ingesting (swallowing) large amounts of PAHs.

Under normal conditions of environmental exposure, PAHs could enter your body if your skin comes into contact with soil that contains <u>high levels</u> of PAHs. Studies have shown that low molecular weight (lighter) PAHs can be absorbed through the skin but the absorption of high molecular weight (heavy) PAHs is quite limited.

Once in the human body, PAHs are changed into different substances and stored in tissue and fat cells.

Results from animal studies show that PAHs do not tend to be stored in your body for a long time. Most PAHs that enter the body leave within a few days, primarily in the feces and urine.

Can PAHs make you sick?

Yes, you can get sick from PAHs. But getting sick will depend on:

- How much you were exposed to (dose).
- How long you were exposed (duration).
- How often you were exposed (frequency).
- Route of exposure: Ingesting (eating) and inhaling (breathing) is more of a risk than dermal (skin) exposure.
- General Health, age, lifestyle: Young children, the elderly and people with chronic (on going) health problems are more at risk to chemical exposures.

PAH's have a low acute toxicity. What this means is that if you were exposed to high levels of PAH's for a short period of time, you will most likely not experience harmful health effects.

Chemicals with high acute toxicity are chemicals that would cause immediate harmful health effects or even death if you came in contact with a high dose. Examples of chemicals with a high acute toxicity are cyanide or arsenic. If you were to come in contact with high levels of arsenic or cyanide, you could die. This is not the case with PAHs.

Do PAHs cause cancer?

It is uncertain if PAHs are carcinogenic (cancer causing) to humans.

Several studies have shown that PAHs have caused tumors in laboratory animals when they breathed these substances in the air, when they ate them or when they had long periods of skin contact with them. Studies in animals have also shown that PAHs can cause harmful effects on skin and the body's system for fighting disease after both short and long-term exposure. But these effects have not been reported in humans.

Studies of people show that individuals exposed by breathing or skin contact for long periods to mixtures that contain PAHs and other compounds may develop cancer. But the studies were uncertain if the cancer was caused by PAHs or the other associated chemicals.

The U.S. Department of Health and Human Services (HHS) has determined some PAHs are known animal carcinogens.

The International Agency for Research on Cancer (IARC) has determined some PAHs are probably carcinogenic to humans, some PAHs are possibly carcinogenic to humans and some PAHs are not classifiable as to their carcinogenicity to humans.

The U.S. Environmental Protection Agency (EPA) has determined some PAHs are probable human carcinogens and some PAHs are not classifiable as to human carcinogenicity.

Is there a medical test to determine whether I have been exposed to PAHs?

Yes. Many PAHs can be measured in the blood or urine soon after exposure. Although these tests can show that you have been exposed to PAHs, these tests cannot be used to predict whether any health effects will occur or to determine the extent or source of your exposure to the PAHs. It is not known how effective or informative the tests are after exposure has stopped. The medical tests used to identify PAHs or their products are not routinely available at a doctor's office because special equipment is required to detect these chemicals. Seek medical advice if you have any symptoms you think may be related to chemical exposure.

What recommendations has the federal government made to protect human health?

Water: Drinking Water MCL (Maximum Contaminant Level) for *Benzo (a) pyrene* is 0.2 ppb (parts per billion). *Benzo (a) pyrene* is a heavy (or a higher molecular weight) PAH.

Air: No standards exist for the amount of PAHs allowed in the air of private homes. However, air standards have been set for occupational (work) settings.

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^3). The OSHA Permissible Exposure Limit (PEL) for mineral oil mist that contains PAHs is 5 mg/m^3 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^3 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.

For more information about PAHs:

For detailed information about PAHs, visit the Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for PAHs.
Web Site: http://www.atsdr.cdc.gov/toxprofiles/tp69.html
E-mail: ATSDRIC@cdc.gov
Toll-free: 1-888-422-8737

References:

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

ATSDR. 1990. Polynuclear Aromatic Hydrocarbon (PAH) Toxicity. Case Studies in Environmental Health Medicine #13. U.S. Department of Health and Human Services. 19p.

Wisconsin Department of Health and Family Services, Division of Public Health, Bureau of Environmental Health, Chemical Fact Sheet, PAHs, 2004.

Comprehensive Cancer Program
Division of Prevention
"To protect and improve the health of all Ohioans"

Exposure to Toxic Chemicals and Cancer

How are we exposed to chemicals in our environment?

We come in contact with many different chemicals every day that generally do not cause health problems. But any chemical can become toxic if a person comes into contact with large enough doses. For example: aspirin will cure a headache, but too much aspirin becomes toxic and can cause serious health problems. Contact with toxic chemicals does not always cause adverse (negative) health effects. Whether you get sick as a result of a chemical exposure depends on:

- How toxic the chemicals are;
- How much you were exposed to (dose);
- How long you were exposed (duration);
- How often you were exposed (frequency);
- Your general health, age and lifestyle. Young children, the elderly and people with chronic (ongoing) health problems are more at risk to health problems following exposures to chemicals.

What is a completed exposure pathway?

Chemicals must have a way to get into a person's body in order to cause health problems. Environmental scientists work to show the five links between a chemical source and the people who are exposed to a chemical. In order for a person to get sick from contact with chemicals, a "Completed Exposure Pathway" must be present.

The five links that make a completed exposure pathway include:

(1) Source (where the chemical came from);
(2) Environmental Transport (the way the chemical moves from the source to the public. This can take place through the soil, air, underground drinking water or surface water);
(3) Point of Exposure (where contact with the chemical is made. This could be where chemical contamination occurred or off-site if the contamination has moved);
(4) Route of Exposure (how people came into physical contact with the chemical. This could occur by drinking, eating, breathing or touching the chemical);
(5) People Who Might be Exposed (those who are most likely to come into physical contact with a chemical).

Documenting a completed exposure pathway:

Documenting a completed exposure pathway can link a chemical exposure with a health problem such as cancer. But it is difficult to study communities living near chemical contamination sites and link their health problems with exposure to a chemical. A few of the difficulties include:

- Not knowing the exact level of a person's exposure to a cancer-causing chemical. This is especially true if the exposure to chemicals occurred years ago and there is no information to prove the exposure;
- Chemical contamination sites often contain more than one chemical. This makes it difficult to link a health problem to a single exposure or chemical;
- Scientists who study communities will also look at other factors before linking a disease to an exposure from a site. Cancer often takes a long time to develop and getting information on the type of past behaviors that increase the risk of getting cancer (such as tobacco use, alcohol consumption and diet) are often difficult or sometimes impossible to collect.

Do toxic chemicals cause cancer?

Yes, some chemicals are known to be carcinogenic (cause cancer). But it is important to know that less than 5% of all cancers are believed to be due to factors in the environment such as environmental pollution (2%), industrial products (1%) or food additives (1%).

Toxic chemicals are cancer risk factors. A risk factor is anything that could increase a person's chance of getting a disease. Cancer risk factors, such as tobacco use, drinking a lot of alcohol, having a poor diet, lack of physical activity and unprotected exposure to the sun, can be changed. Other cancer risk factors such as a person's age, sex and family medical history (genetics) cannot be changed.

The Ohio Department of Health works closely with the Agency for Toxic Substance and Disease Registry (ATSDR), the U.S. EPA and Ohio EPA, local health departments and concerned communities to investigate and prevent harmful exposures and disease related to toxic substance in the environment.

IMPORTANT FACTS: Cigarette smoke contains 43 known cancer-causing chemicals. In 2003, the U.S. EPA Superfund program prepared a list of the 275 chemicals found at chemical contamination sites throughout the nation. Six of the top 10 chemicals found at chemical contamination sites are also found in cigarette smoke.

A List of Known[1] and Possible[2] Human Carcinogenic (cancer-causing) Agents by Organ		
Organ	**Known Human Carcinogen**	**Possible Human Carcinogen**
Bladder	* Arsenic * Benzidine * Cadmium * Tobacco smoke	* Tetrachloroethylene (PERC or PCE)
Blood Diseases (leukemia, lymphoma)	* Benzene * Ionizing Radiation	* Trichloroethylene (TCE) * Vinyl chloride
Brain		* Vinyl chloride
Colon	* Arsenic	
Kidney	* Arsenic * Coke oven emissions	* Tetrachloroethylene (PERC or PCE) * Chloroform * Trichloroethylene (TCE)
Liver	* Alcoholic drinks * Vinyl chloride	* Chlordane * Chloroform * Dieldrin * Polychlorinated Biphenyls (PCBs) * Trichloroethylene (TCE)
Lung	* Arsenic * Asbestos * Beryllium * Cadmium * Chromium (Hexavalent) * Coke oven emissions * Tobacco smoking * Uranium - Radon	* Benzo(a)pyrene * Polycyclic aromatic hydrocarbons (PAHs) * Vinyl chloride
Mouth, Pharynx, Larynx, Esophagus	* Alcoholic drinks * Chewing tobacco (mouth only) * Tobacco smoke	
Skin	* Arsenic * Overexposure to the sun	* Benzo(a)pyrene * Polycyclic aromatic hydrocarbons (PAHs) * Tetrachloroethylene (PERC or PCE)

[1] The category *"known human carcinogen"* requires evidence from human studies.

[2] The category *"possible human carcinogen"* gathers evidence mainly from animal studies. There may be limited human studies or there may be no human or animal study evidence to support carcinogenicity; but the agent, substance or mixture belongs to a well-defined class of substances that are known to be carcinogenic.

Note: Due to limited space, the above table is not a complete listing of all the known and possible human carcinogens. The top 20 chemicals listed in this table can be found in the 2003 U.S. EPA Superfund, Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) listing of chemicals found at chemical contamination sites placed on the National Priorities List (NPL).

- To see a full listing of known and possible carcinogens, you can review the National Toxicology Program, Report on Carcinogens, Eleventh Edition or visit online (see below reference).
- To see a full listing of the chemicals found at National Priorities List (NPL) sites, you can review the 2003 CERCLA Priority List of Hazardous Substances report or visit online (see below reference).

References:

- American Cancer Society, 2004.
- Ohio Department of Health, Comprehensive Cancer Program, 2004.
- Agency for Toxic Substances and Disease Registry, 2003 CERCLA Priority List of Hazardous Substances (2005 electronic at **www.atsdr.cdc.gov/cercla/**).
- Report on Carcinogens, Eleventh Edition; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, 2005 (2005 electronic at **http://ntp.niehs.nih.gov/ntp/roc/toc11.html**).

For more information contact:

Ohio Department of Health
Bureau of Environmental Health
Health Assessment Section
(614) 466-1390

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Agency for Toxic Substances and Disease Registry (ATSDR)
Toll-free at 1-888-422-8737

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What is cancer?

Cancer is the irregular growth of abnormal cells. In the human body, normal cells grow, divide and die in a normal process. Cancer cells outlive normal cells and continue to grow and make new abnormal cells.

Cancer cells will clump together and form tumors. These tumors can invade and destroy normal cells and tissue. Tumors can be malignant (cancerous) or benign (non-cancerous).

Cancer cells can travel (metastasize) through the blood or the lymph system to other areas of the body where they can settle and form new tumors. Some cancers, such as leukemia, do not form tumors but invade the blood and blood-forming organs. Benign (non-cancerous) tumors do not spread to other parts of the body and are usually not life-threatening.

In many cases, the exact cause of cancer is not known. We know certain changes in our cells can cause cancer to start but we don't yet know exactly how this happens. Many scientists and health professionals study cancer in the hope they can discover the causes and a cure. But, there are a lot things we **do** know about cancer.

Who gets cancer?

Cancer may strike at any age. However, cancer is mostly a disease of middle and old age. In Ohio, about 86% of cancers were diagnosed in people age 50 and older in 2000.

Cancer is the second-leading cause of death in the United States. It is estimated that half of all men and one-third of all women in the United States will develop cancer during their lifetimes.

In 2003, about 60,300 Ohioans – or 165 Ohioans per day – were diagnosed with cancer. More than 25,200 Ohioans – or about 69 people each day – died from it.

What are cancer risk factors?

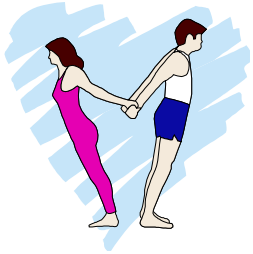
A risk factor is anything that increases a person's chance of getting a disease. Some risk factors, such as tobacco use, can be changed, and others, such as age, cannot.

Having a risk factor for cancer means a person is more likely to develop the disease at some point in his or her life. However, having one or more risk factors does not always mean a person will get cancer. Some people with one or more risk factors never develop the disease, while other people who develop cancer have no apparent risk factors. Even when a person who has a risk factor is diagnosed with cancer, there is no way to prove the risk factor actually caused the cancer. In reality, getting cancer is probably due to the combination of risk factors rather than one single factor.

Risk factors for cancer include a person's age, sex and family medical history (genetics). Other major factors are related to lifestyle choices such as using tobacco, drinking a lot of alcohol, eating a poor diet, lack of physical activity and unprotected exposure to the sun. Occupational (work) exposures can be another risk factor.

Using tobacco products, a poor diet and lack of physical activity account for about 65% of cancer deaths. Less than 5% of cancers are believed to be due to factors in the environment such as environmental pollution (2%), industrial products (1%) or food additives (1%).

The risk of developing most types of cancers can be reduced by changes in a person's lifestyle. By quitting smoking, eating healthier and exercising, you can reduce your risk of developing cancer.



Risk factors (continued)

Different kinds of cancer have different risk factors. Some of the common cancers and their risk factors include the following:

- Lung cancer: **Tobacco smoking is responsible for 80 to 85 percent of lung cancers.** Note: Tobacco use (including cigarettes, cigars, chewing tobacco and snuff) is also related to cancers of the mouth, larynx, cervix, bladder, kidney, esophagus and pancreas. Other important risk factors for lung cancer include exposure to radon and asbestos; a history of tuberculosis and some types of pneumonia; and family history.
- Breast cancer risk factors include: Increasing age; hormone-related factors such as early age at first menstruation, fewer number of pregnancies and late age at menopause; obesity; and lack of physical activity. Also, women with a mother or sister who have had breast cancer are more likely to develop the disease themselves (genetics). **All women 40 years and older should get a yearly mammogram and perform monthly self-examinations.**
- Prostate cancer: All men are at risk for prostate cancer. Prostate cancer is more common among African-American men compared to white men. Also, men with a father or brother who have had prostate cancer are more likely to get prostate cancer themselves (genetics). **All men 50 years and older should talk with their doctor about being tested.**
- Colon and Rectum cancer risk factors include: Increasing age (persons 50 years and older); a diet high in animal fat; lack of exercise; and obesity. **Women and men should be screened for colorectal cancer beginning at age 50.**
- Skin cancer is related to unprotected exposure to strong sunlight and severe sunburns as a child**. To protect against skin cancer use sunscreen, wear protective clothing and avoid direct sunlight between 10 a.m. and 4 p.m.**
- Cervical cancer risk factors include: infection with a certain sexually transmitted disease (STD) called the Human Papilloma Virus (HPV); smoking; and being HIV positive. **It is important for women to receive regular Pap tests because they can detect HPV and pre-cancerous cells.**

How is cancer treated?

Cancer is a group of diseases that behave very differently. For example, lung cancer and breast cancer develop and grow at different rates and respond to different treatments. That is why people with cancer need treatment that is aimed at their particular kind of cancer.

The patient is a vital part of his or her cancer care team. Patients and families should talk with their health care providers about which treatment choices are best. Today, millions of people are living with cancer or have been cured of the disease. **The sooner a cancer is found and the sooner treatment begins, the better a patient's chances are of a cure.** That is why early detection is such an important weapon in the fight against cancer.

Learn more about cancer:

Cancer is the second-leading cause of death among adults in Ohio following heart disease.

According to a survey released at the 11th Annual Research Conference of the American Institute for Cancer Research (AICR), cancer is the No. 1 day-to-day health concern in America. Additionally, half of all Americans believe it is impossible or next to impossible to prevent cancer. But this is not true and in many cases, cancer can be prevented.

The Ohio Department of Health wants to help Ohioans learn more about cancer, including how to prevent it, how to find it early and how to get treatment if needed.

Through coordination and working together we will make a difference in the health and quality of life in our state.

References:

American Cancer Society, http://www.cancer.org, 2003.

Winauer SJ, Shike M. Cancer Free: The Comprehensive Cancer Prevention Program. New York: Simon and Shuster, 1995.

Ohio Department of Health, Comprehensive Cancer Program, 2003.

American Institute for Cancer Research, July, 2001.

For more information:

If you have questions or if you need information that is not available on this fact sheet, please contact one of the following organizations:

Ohio Department of Health
(614) 728-7418
American Cancer Society
1-800-ACS-2345 or 1-800-227-2345
Ohio Radon Program
1-800-523-4439
National Cancer Institute
1-800-422-6237