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Appendix H added 6/3/98

Index of Appendices 6/3/98 updated

BASELINE HUMAN HEALTH RISK ASSESSMENT

Former UGI Columbia
Manufactured Gas Plant
Columbia, Pennsylvania

April 1998

Prepared for:

Clean Sites, Inc.
1199 North Fairfax Street
Suite 400
Alexandria, Virginia 22314

Prepared by:

Menzie-Cura & Associates, Inc.
One Courthouse Lane, Suite Two
Chelmsford, MA 01824

ORIGINAL (Red)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

Office of Superfund
Steven J. Donohue

Direct Dial (215) 597-3166
Mail Code (3HW22)

Douglas C. Ammon, P.E.
Project Manager
Clean Sites Environmental Services, Inc.
635 Slaters Lane, Suite 130
Alexandria, VA 22314

June 1, 1998

Re: UGI Columbia Gas MGP Site
Approval of Final Baseline Human Health Risk Assessment Report

Dear Doug:

The United States Environmental Protection Agency ("EPA") has received and reviewed the April 1998 Baseline Human Health Risk Assessment ("RA") for the UGI Columbia Manufactured Gas Plant Site ("Site"). The RA was submitted by Menzie Cura & Associates, Inc. and received by EPA on April 16, 1998. EPA has reviewed the RA to ensure revisions made to the text and appendices were responsive to comments made by EPA in a January 6, 1998 letter and subsequent communications.

Enclosed please find a copy of an internal EPA memorandum from Lynn Flowers, Ph.D., the toxicologist for the Site, to me dated May 19, 1998. EPA will consider the RA final and approved provided the memo is included as an appendix in the Final RA. EPA recommends the table of contents be revised, a tab be made for the additional appendix and the items forwarded to EPA for inclusion in the previously submitted RA. If you have any questions or would like to discuss the contents of the memo please contact me.

I have contacted Tony Martinelli, the Pennsylvania Department of Environmental Protection ("PADEP") project manager for the Site. Mr. Martinelli indicated that PADEP would not have any additional comments on the RA. Therefore, satisfaction of the EPA comments would make the RA final.

ORIGINAL (Red)

As indicated in my April 8, 1998 letter approving the Remedial Investigation report, please contact EPA and PADEP as soon as possible to schedule a meeting to discuss the Feasibility Study for the Site. If you have any questions on the above comments please contact me at the number above.

Sincerely,

Steven J. Donohue
Remedial Project Manager

cc: Anthony Martinelli, PADEP

ORIGINAL (Red)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Former UGI Columbia Manufactured Gas Plant
Baseline Human Health Risk Assessment
April 1998

FROM: Lynn Flowers, Ph.D., Toxicologist
Technical Support Section (3HS41) Lynn Flowers

TO: Steve Donohue, RPM
Eastern Pennsylvania Section (3HS22)

May 19, 1998

I have reviewed the document and the accompanying responses to EPA comments and have the following comments for your consideration:

Responses to January 6, 1998 Comments

(1) Comment 3: "worker" should be "resident."

(2) Comment 4: The sediment data for the Susquehanna River has not been included in Appendix A as indicated.

(3) Comment 11: Section 3.2.6 indicates that residential exposure to on-site subsurface soil was not included in the risk assessment (see last sentence of second paragraph). This exposure pathway should have been included in the risk assessment as the soil would have to be re-worked in order to build a residence on the Site.

(4) Comment 13: The statement that the PEF units do not cancel properly is true. However, the actual calculation is correct. The manipulation of the equation in order to incorporate a more appropriate Q/C value is entirely acceptable.

(5) Comment 24: The reference should be to aluminum and beryllium.

(6) Comment 39: Total skin surface areas and soil adherence factors that were actually inputted into the risk equations were requested and are not found in Appendix A.

(7) Comment 40: The response is incorrect. It was stated that the 95% UCL of the mean would be used for both RME and CTE calculations. This has been misinterpreted. All other CTE exposure factors should be average values. The actual CTE values that were used are not shown in the document.

ORIGINAL (Red)

(8) Comment 41: The final exposure frequency that was used in the risk assessment was needed in order to cross-check the calculations.

Responses to March 11, 1998 Comments

None

Comments on Risk Assessment Document

(1) ES; page viii; summary table:

(a). The risks for soil and ground water for future residents either on-site or off-site have been combined. It would be beneficial for these risks to be separated for risk management purposes. The values can be determined from Appendices E and G.
(b). Only surface soil was considered for an on-site residential scenario. Both surface and subsurface soil should have been included as the soil would require re-working in order for a home to be built on the Site. Residents would then be exposed to both surface and subsurface soils. Both surface and subsurface soils were considered in the assessment of risk to a potential off-site resident.
(c). The fraction ingested from source (Fi) should be 0.5 for off-site (both near the S. River and south of the Site) soil exposure scenarios, i.e., for construction workers and residents. It is presumed that the surface soil is uncontaminated. The risk values shown for these scenarios should be divided by two.
(d). The RME concentrations for inorganic compounds found in subsurface soils south of the Site (includes sample TP-A) are incorrect. It appears that they correspond to the subsurface soil concentrations found near the S. River. After correcting for this error, the noncancer hazard index for a construction worker who would be exposed to subsurface soils south of the Site is 0.6 and the increased cancer risk is 1.1E-5.

(2) ES; conclusions: No reference is made to the dermal exposure risk characterizations that were included in the risk assessment.

(3) Page 10: "A-6" should be "A-4" and "A-7" should be "A-5."

(4) Tables: A number of the Tables contain footnotes which incorrectly state that benzo[ghi]perylene and acenaphthylene will be evaluated "quantitatively" as opposed to "qualitatively."

(5) The qualitative discussion on the toxicity of benzo[ghi]perylene and acenaphthylene is lacking in information. The on-site and off-site concentrations of acenaphthylene ranged from 3.8 to 160 mg/kg. The concentration of benzo[ghi]perylene ranged from 4.4 to 11 mg/kg.

Additional Comments

(1) The effect of including all PAHs detected in soils in the risk assessment was determined. The risk values were increased but the outcome of the risk assessment was unchanged.

(2) Taking all comments into consideration, the possibility of slightly varying input variables being used in the EPA analysis, and rounding differences, the following is a corrected summary of the risks found at the Site for all scenarios, including those where no errors were found.

Ground water human health risks (on-site and off-site) are unacceptable and can be found in Appendix E.

Conclusions

(1) None of the above-mentioned corrections would lead to a change in the overall outcome of the risk assessment.

(2) On-site and off-site ground water human health risks were found to be unacceptable.

(3) On-site soils (surface and subsurface) would pose an unacceptable risk to potential future residents.

(4) The Hazard Quotients for the on-site construction worker scenario and the off-site child-resident scenario were found to be 1.2 and 1.8, respectively. These risks are associated with several target organs, i.e., skin effects, central nervous system effects, and iron overload, with each individual systemic effect having a hazard quotient of less than 1.0. Therefore, the on-site soils do not pose a health threat to construction workers and the off-site soils do not pose a risk to a potential future child resident.

(5) Dermal risk to PAHs present in soils was not considered in this risk assessment. Currently, toxicity values for the determination of risk through dermal exposure to PAHs are not available. EPA Region III has been advised (National Center for Environmental Assessment, Cincinnati, OH) to not include an assessment of this risk as the choice of toxicity values would be inappropriate. It should be noted that the non-cancer risk (due to skin irritation from PAHs) and the cancer risks would be increased if it were possible to quantitate this facet of the risk assessment. It is not possible to estimate the impact of this uncertainty.

.UGI Columbia MGP risks by scenario		
	Total Risk	
On-site	Hazard Index	Cancer Risk
Industrial worker (surface soil only)	0.12	1.3E-5
Construction worker (surface and subsurface soil)	**1.2**	2.3E-5
Trespasser (surface soil and sediment)	0.05	1.3E-6
Child resident (surface and subsurface soil)	**4.2**	**4E-4**
Adult resident (surface and subsurface soil)	0.4	**1.6E-4**
Off-site (subsurface soil near Susquehanna River)		
Construction worker	0.4	3.3E-6
Child resident	**1.8**	6.2E-5
Adult resident	0.12	2.4E-5
Off-site (subsurface soil south of the Site)		
Construction worker	0.6	1.1E-5

If I can be of further assistance please contact me at 6-3115.

cc: E. Johnson (3HS41)
ugi6

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TABLES

FIGURES

APPENDICES

ACRONYMS

ADD	Average daily dose
ADD(life)	Average daily dose (lifetime)
ADD(year)	Average daily dose (year)
AT	Averaging time
BW_{avg}	Average body weight
CNS	Central nervous system
COPC	Chemicals of potential concern
COC	Contaminant of concern
CSF	Cancer slope factor
CTE	Central Tendency Exposure
ED	Exposure duration
EF	Exposure frequency
EP	Exposure period
EPC	Exposure point concentration
GRI	Gas Research Institute
HEAST	Health Effects Assessment Summary Tables
IEU/BK	Integrated Exposure Uptake/Biokinetic Model
IR	Daily soil ingestion rate
IRIS	Integrated Risk Information System
MGP	Manufactured gas plant
NA	Not applicable (as specified in tables)
ND	Not detected (as specified in tables)
NRC	National Research Council
PAH	Polycyclic aromatic hydrocarbons
PEF	Particulate emission factor
PP&L	Pennsylvania Power and Light Company
RAF	Relative absorption factor
RBC	Risk-based concentration
RfC	Reference concentration
RfD	Reference dose
RME	Reasonable maximum exposure
SVOC	Semi-volatile organic compound
TEF	Toxicity equivalency factors
USEPA	United States Environmental Protection Agency
UCL_{95}	95% upper confidence limit
VF	Volatilization factor
VOC	Volatile organic compound

EXECUTIVE SUMMARY

This report provides a baseline human health risk assessment for the Former UGI Columbia Manufactured Gas Plant (MGP) site near the Susquehanna River in Columbia, Pennsylvania (the site). The objectives of the baseline human health risk assessment are to determine the potential for health effects to individuals who may be exposed to MGP-related chemicals both on site and off site for current and potential future land uses. In many cases, exposures would occur only if a particular set of conditions exist. The likelihood of these conditions occurring is a consideration in the risk analysis. This risk assessment follows U.S. Environmental Protection Agency (USEPA) guidance (*e.g., Risk Assessment Guidance for of Superfund. Volume I - Human Health Evaluation Manual (Part A) Interim Final*, 1989a). The risk assessment has been modified from previous drafts (April, 1997, November, 1997) based on several comment letters (August, 1996, January, 1998; March, 1998) and discussions with EPA Region III personnel.

Site Background

The site is approximately 0.8 acres in size and is located in an area of mixed residential and industrial land use. Currently, the site is vacant and fenced. Nearby land use includes a wastewater treatment facility, a borough garage, a boat sales and repair shop, residences, and a surface water supply facility. The local geology consists of shallow subsurface soils consisting mainly of fill overlying alluvial deposits. Weathered bedrock (saprolite) is present in some areas as well. The underlying bedrock consists of limestone which exhibits fracture zones oriented roughly east to west (Atlantic Environmental Services, Inc., 1996).

Surface and subsurface soil samples, sediment samples, surface water samples and groundwater samples indicate the presence of MGP-related wastes, including polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs). Physical MGP-related impacts, such as tar, odor and sheen, are present in soils (overburden and bedrock) on site and near the downgradient trend of the bedrock fracture zone to the east bank of Shawnee Creek. Tar wastes are also present in the overburden to the northwest of the wastewater treatment plant near the Susquehanna River shoreline (Figure 1).

Identification of Chemicals of Potential Concern

Based on the screening methodology, chemicals of potential concern (COPCs) include volatile organic compounds, *e.g.*, benzene, toluene, ethylbenzene, and xylenes (BTEX); semi-volatile organic compounds, *e.g.*, polycyclic aromatic hydrocarbons (PAHs); and, inorganic compounds, *e.g.*, metals and cyanide.

Exposure Assessment

This risk assessment evaluates exposure scenarios based on individuals potentially exposed to site-related contaminants, either presently or in the future, assuming no remediation or removal or capping will occur, even if the site were to be redeveloped. Some of these scenarios, most notably the hypothetical resident scenario involving ingestion of

contaminated groundwater, are unlikely to occur, but are evaluated for completeness as requested in USEPA Region III comments (USEPA, 1996a,1997a, 1998).

Several residences located approximately one quarter mile to the northwest of the site currently have private wells installed. Exposure and risks to current residents are not evaluated quantitatively because these locations are not hydrogeologically downgradient of contaminated groundwater, and because organic MGP-related contaminants were not detected in these wells. Based on the location of the private wells, the low mobility of the MGP-related chemicals, and the nature and direction of the bedrock fractures, these residences do not appear to be at risk due to MGP-related contaminants under current or future conditions.

Exposure scenarios include:

- a trespasser (current scenario), exposed to surface soil and fugitive dust on site and off site, and sediment in Shawnee Creek. There were no COPCs detected in Shawnee Creek surface water.

- an industrial worker (future scenario), exposed to on-site surface soil, fugitive dust, and soil vapors;

- a construction worker (future scenario), exposed to on-site surface and subsurface soil, fugitive dust, and soil vapors;

- a construction worker (future scenario), exposed to subsurface soil, fugitive dust, and soil vapors at two off-site areas: near the Susquehanna River, and near Front Street;

- a hypothetical resident (future scenario), exposed to off-site subsurface soils, fugitive dust, and groundwater from a hypothetical private well adjacent to the Susquehanna River; and,

- a hypothetical resident (future scenario), exposed to on-site surface and subsurface soils, fugitive dust, and groundwater from a hypothetical private well on the site.

Risks are estimated using both central tendency exposure (CTE) and reasonable maximum exposure (RME) exposure point concentrations for the COPCs. RMEs are based on the 95th percent upper confidence limit of the mean concentration (or the maximum concentration if the upper confidence limit exceeds the maximum concentration) and are intended to provide conservative upper bound concentrations. CTE estimates are calculated, per agreement with EPA, by using the mean exposure point concentration, and RME exposure assumptions.

Toxicity Assessment
The toxicity of the COPCs are described for potential non-carcinogenic and carcinogenic effects. The USEPA's Integrated Risk Information System (IRIS) is the primary source of this information.

Risk Characterization Summary
Individual or summed Hazard Index values greater than 1 are considered to indicate the potential for non-cancer health effects. A Hazard Index less than 1 indicates that it is unlikely for even sensitive sub-populations to experience adverse health effects.

Total cancer risk estimates greater than 1×10^{-4} are generally considered unacceptable, and risks less than 1×10^{-6} are generally considered *de minimis*. Estimates that are between these levels require regulatory action based on a combination of factors, such as the size of the exposed population or cost-effectiveness considerations (EPA, 1990).

The following are the risk estimates for the scenarios considered in the baseline human health risk assessment:

	Hazard Index		Cancer Risk	
	CTE	RME	CTE	RME
Current Scenarios				
Trespasser	0.06	0.08	1×10^{-6}	2×10^{-6}
Future Scenarios				
Industrial worker	0.06	0.14	8×10^{-6}	2×10^{-5}
On-site construction worker	0.7	1.2	5×10^{-6}	2×10^{-5}
Off-site construction worker				
Near River	0.8	0.8	5×10^{-6}	8×10^{-6}
Near Front Street	0.6	0.8	7×10^{-6}	2×10^{-5}
Hypothetical resident (off-site)				
Child (0-6 yrs)	78	920	6×10^{-4}	7×10^{-3}
Adult (7-30 yrs)	31	370	9×10^{-4}	1×10^{-2}
Hypothetical resident (on-site)				
Child (0-6 yrs)	230	580	4×10^{-3}	1×10^{-2}
Adult (7-30 yrs)	93	230	7×10^{-3}	2×10^{-2}

Trespasser(current)
The total Hazard Indices for the CTE exposure case and RME cases are less than 1. The total cancer risks are less than 1×10^{-6} for the CTE exposure (9.6×10^{-7}) and greater than 1×10^{-6} for the RME case (1.5×10^{-6}). The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in on-site surface soils. The evaluation conservatively assumes that trespassers regularly contact on-site soils, even though the site is surrounded by a chain link fence. If exposure to on-site surface soils is eliminated (and

exposure to off-site areas persisted), the cancer risk estimates for this scenario would be less than 1×10^{-6}.

Industrial worker (future)
The total Hazard Indices for the CTE exposure case and RME case are less than 1. The total cancer risk for the CTE exposure case (8×10^{-6}) is greater than 1×10^{-6}; for the RME case, the risk estimate (2×10^{-5}) is greater than 1×10^{-5}. The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in on-site surface soil.

On-site construction worker (future)
The total subchronic Hazard Index for the CTE case is less than 1 (0.7) but exceeds 1 for the RME case (1.2). The total cancer risks are greater than 1×10^{-6} for the CTE exposure case (5×10^{-6}) and the RME case (2×10^{-5}). The cancer risk estimate for this scenario is driven by potential exposure to carcinogenic PAHs in on-site soils, and assumes no personal protective equipment is used.

Off-site construction worker (future)
Two discrete locations were evaluated for this scenario: near the Susquehanna River and the wastewater treatment plant, and between Front Street and the railroad tracks adjacent to the site.. The total Hazard Indices for the CTE exposure case and RME cases are less than 1 for both exposure points. The total cancer risks are greater than 1×10^{-6} for the CTE exposure case (5×10^{-6}; 6.6×10^{-6}) and for the RME case (8×10^{-6}; 2×10^{-5}). The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in both areas of soil contamination.

Hypothetical resident (off-site; future)
Currently, no residents are located south and southwest of the site, however the risk assessment evaluates the potential exposure of a hypothetical resident located near the Susquehanna River and the wastewater treatment plant. Risks for this scenario are likely overstated, considering the nature of the exposures evaluated. Exposure to contaminants in groundwater is assumed to occur regularly via use of a private well. These exposures are evaluated assuming an exposure duration of 30 years. We did not evaluate the inhalation and dermal pathways quantitatively. The risks associated with residential exposure to groundwater by ingestion already exceed acceptable risk levels, and would be even higher if we considered risks from inhalation and dermal contact with groundwater. Because the Total Hazard Index represents risks to different target organs, we separate Hazard Indices specific to different target organs.

Total cancer risks for this scenario are greater than 1×10^{-4} and are driven by potential exposure to groundwater as drinking water; exposure to off-site soil also contributes to the total cancer risk. It is our opinion that a future residence in the area (near the Susquehanna River, wastewater treatment plant, and water intake facility) is highly unlikely based on zoning regulations and surrounding land use.

Hypothetical resident (on-site; future)
The risk assessment evaluates potential exposure of a hypothetical resident located on the site. Risks for this scenario are likely overstated, considering the nature of the exposures evaluated. This scenario is considered highly unlikely, and is evaluated per request of EPA (1997a). Exposure to contaminants in groundwater is assumed to occur regularly via use of a private well. These exposures are evaluated assuming an exposure duration of 30 years. Risks from inhalation and dermal contact with groundwater are not calculated because the risks associated with a hypothetical residential drinking water exposure to groundwater exceed the acceptable risk range of 1×10^{-4}, these pathways would also contribute to cancer risk from groundwater. Because the Total Hazard Index represents risks to different target organs, we separate Hazard Indices specific to different target organs.

Total cancer risks for this scenario are greater than 1×10^{-4} and are driven by potential exposure to groundwater as drinking water; exposure to on-site soil also contributes to the total cancer risk (9×10^{-5}). It is our opinion that a future on-site resident is highly unlikely based on zoning regulations and surrounding land use.

Conclusions
The conclusions of this baseline risk assessment are intended to provide risk managers with insight on how to evaluate the potential risks associated with the Former UGI Columbia MGP site. The results are discussed as they relate to media, locations, chemicals, and pathways with the intention of promoting an appropriate remedial strategy.

On-site soils
Contamination of on-site soil has been a focus of the remedial investigation. Under existing conditions, wherein the site is vacant and inaccessible, there is the potential for surface soil to pose a low cancer risk to trespassers (between 1×10^{-7} and 2×10^{-6}). The cancer risk is due to incidental ingestion and dermal contact with carcinogenic PAHs in on-site soil; the risks associated with exposure to off-site surface soils are *de minimis*, *i.e.*, less than 1×10^{-6}.

Under future conditions, assuming the site is being redeveloped, there is the potential for soil to pose a low to moderate cancer risk to construction workers as a result of direct contact with subsurface soils during excavation without personal protective equipment. The cancer risk estimate for the CTE exposure case (5×10^{-6}) is between 1×10^{-6} and 1×10^{-5}. The cancer risk estimate for the RME case (2×10^{-5}) is between 1×10^{-5} and 1×10^{-4}. The cancer risk is associated with incidental ingestion and dermal contact with carcinogenic PAHs in surface and subsurface soil while excavating at the site.

Under future conditions, assuming the site has been developed for industrial purposes, there is the potential for soil to pose a low to moderate cancer risk to industrial workers. The cancer risk estimate for the average CTE exposure case (8×10^{-6}) and the RME case (2×10^{-5}) is between 1×10^{-5} and 1×10^{-4}. The cancer risks are associated with incidental ingestion with carcinogenic PAHs in surface soil.

Under future conditions, although highly unlikely, the site could be redeveloped for residential purposes. There is potential for soils to pose a moderately high cancer risk (between 1 x 10^{-5} and 1 x 10^{-4}), and an elevated non-cancer risk. The cancer and non-cancer risk is associated with incidental ingestion and dermal contact with soil.

Off-site soils near the Susquehanna River and the wastewater treatment plant
Under future conditions, assuming the area is being redeveloped, there is the potential for soil to pose a relatively low cancer risk to construction workers by direct contact with subsurface soils (between 1 x 10^{-6} and 1 x 10^{-5}). The cancer risk is associated with incidental ingestion of carcinogenic PAHs in subsurface soil while excavating at the site.

Off-site soils between Front Street and the railroad tracks across from the site
Under future conditions, assuming the area is being redeveloped, there is the potential for soil to pose a moderate cancer risk to construction workers (between 1 x 10^{-6} and 2 x 10^{-5}). The cancer risk is associated with incidental ingestion of carcinogenic PAHs in subsurface soil while excavating at the site.

Sediment at near-shore reach of the Susquehanna River
MGP related compounds were present in the sediment at the near shore reach of the Susquehanna River. Removal of the impacted sediments occurred in January, 1998, which eliminated human health risk associated with exposure to MGP contaminants in these sediments.

Surface Water in Susquehanna River
There were no COPCs detected in Susquehanna River surface water.

Sediment in Shawnee Creek
Under current conditions, the potential risks associated with exposure (incidental ingestion and dermal contact) to Shawnee Creek sediment are *de minimis, i.e.,* less than 1 x 10^{-6}.

Surface Water in Shawnee Creek
There were no COPCs detected in Shawnee Creek surface water.

Groundwater
Under current conditions, residents located approximately one quarter mile to the northwest of the site use groundwater from private wells. Groundwater samples collected from these wells were analyzed for site-related contaminants; there were no organic COPCs detected. Transport of COPCs toward these residences appears unlikely based on the location of the wells, the low mobility of the MGP-related chemicals, and the nature and direction of the bedrock fractures (Atlantic Environmental Services, Inc., 1996).

Though unlikely, the site could be developed in the future for residential land use. If a well were placed in this area, the use of groundwater as drinking water could pose non-

carcinogenic risks greater than 1 and potential cancer risks greater than 1×10^{-4}. Currently, there are no residents at the site.

Though highly unlikely, the area between the site and river could be developed in the future for residential land use. If a well were placed in this area, the use of groundwater as drinking water could pose non-carcinogenic risks greater than 1 and potential cancer risks greater than 1×10^{-4}. Currently, there are no residents between the site and the river.

1.0 INTRODUCTION

This report provides a baseline human health risk assessment for the Former UGI Columbia Manufactured Gas Plant (MGP) site in Columbia, Pennsylvania (the site). The objective of the assessment is to evaluate the potential risk of harm to human populations that could have contact with site-related contaminants, in the absence of remediation. Human exposure is assumed to occur in on-site and off-site locations during current and future land uses. The risk of harm is discussed in terms of the potential for non-carcinogenic and carcinogenic effects.

The risk assessment has been modified from previous drafts (April, 1997, November, 1997) based on several comment letters (August, 1997, January, 1998; March, 1998) and discussions with EPA Region III personnel. The changes made as a result of discussions with EPA are summarized in the accompanying cover memorandum.

The baseline human health risk assessment follows U.S. Environmental Protection Agency (USEPA) guidance documents (*Risk Assessment Guidance for Superfund. Volume I - Human Health Evaluation Manual (Part A) Interim Final* (1989a), and related documents), which outline a four-step process for risk assessment:

- Hazard Identification, which identifies the extent to which contamination is present and specifies the chemicals of potential concern (COPCs);
- Exposure Assessment, which quantifies the exposures associated with current and future land uses, identifies potential receptors, exposure pathways, and exposure media, and estimates exposure point concentrations for receptor groups;
- Toxicity Assessment, which reviews the effects of the COPCs on humans and assigns quantitative estimates of toxicity;
- Risk Characterization, which integrates the exposure and toxicity assessments into quantitative estimates of human health risk for the selected exposure scenarios. The risk estimates are compared with target risk levels established by USEPA.

The results of the risk assessment are based on the outcome of this four-step process. The conclusions of this report provide an opinion as to whether site-related contaminants pose a potential risk to human health.

1.1 Previous Work

A preliminary human health risk assessment (PHHRA) was conducted for the site (Menzie-Cura & Associates, Inc., 1995) and submitted to the Pennsylvania Department of Environmental

Protection and USEPA Region III for review. The PHHRA provided a worst-case assessment of potential risk by using maximum detected concentrations in risk calculations. Scenarios considered in the PHHRA included:

- a child trespasser exposed to on-site surface soil and fugitive dusts;
- a future industrial worker exposed to on-site surface soil and fugitive dusts;
- a future construction worker exposed to on-site soil and fugitive dusts;
- a future recreational boater exposed to near-shore sediments; and
- a future resident exposed to deep groundwater for drinking water.

The results of the PHHRA indicate that on-site exposure to carcinogenic PAHs resulted in potential risks above a *de minimis* level of 1×10^{-6} but less than 1×10^{-4} (with the exception of the future resident scenario). Total cancer risks for the future resident exposed to benzene in groundwater were greater than 1×10^{-4}. All Hazard Indices were below a threshold level of 1.

The scenarios evaluated in a Draft Baseline Risk Assessment submitted to EPA in April, 1997 are similar to the exposure scenarios described in the PHHRA. The April report also included a future construction worker scenario at an area adjacent to the Susquehanna River, and a future residential scenario, as requested by USEPA Region III (Comments on Preliminary Human Health Risk Assessment, 1996a). We expanded the exposure pathways considered in the PHHRA to include dermal contact with soils, sediment, and surface water (USEPA, 1996a).

In this revision of the Baseline Risk Assessment, we eliminate the recreational boater scenario, due to the remediation of Susquehanna River sediment, and add a hypothetical on-site resident, per EPA's request (USEPA Region III (Comments on Baseline Human Health Risk Assessment, 1997), The risk estimates include the most recent rounds of data collected by Atlantic Environmental Services, Inc. This baseline risk assessment presents risk estimates based on Central Tendency Exposure (CTE) exposures and reasonable maximum exposures in accordance with prevailing USEPA guidance, modified as a result of discussions between Menzie-Cura & Associates, Inc. and USEPA Region III staff. For the CTE case, exposure point concentrations are the mean value, and exposure assumptions are default RME assumptions. No deviation is made in the RME case.

A preliminary ecological risk assessment (PERA) was conducted for the site (Menzie-Cura & Associates, Inc., 1994) and submitted to the Pennsylvania Department of Environmental Protection and USEPA Region III for review. The PERA provided a basis for determining if a Potential Risk of Harm exists, and identified data gaps that should be addressed in order to quantify risks. The PERA indicates that no habitat existed on the site, but that PAHs in Susquehanna River sediment indicated conditions pose a Potential Risk of Harm to the Environment. Contaminated sediment was removed in January 1998 in a remedial action as described in the *Engineering Evaluation Cost Analysis (EE/CA),* (Remediation Technologies, Inc. August, 1996).

1.2 Site Background

1.2.1 Site description

The 0.8-acre site is in an area of mixed residential and industrial land use approximately 400 feet northeast of the Susquehanna River in Columbia, Pennsylvania (Figure 1). Currently, the site is vacant and fenced. Nearby land use consists of the municipal wastewater treatment plant, a borough garage, a boat sales and repair shop, residences, railroad tracks, and a surface water supply facility. Shawnee Creek, a perennial tributary of the Susquehanna River, is approximately 400 feet northwest of the site.

Atlantic Environmental Services, Inc. conducted a local zoning study of the site and surrounding area by contacting the Columbia Tax Assessor's Office and Planning and Zoning Department (Atlantic Environmental Services, Inc., 1996). A map depicting zoning in the area (Figure 2) illustrates that the site and adjacent areas immediately to the south and west are zoned for industrial use only. Areas immediately adjacent to the creek and river are zoned as floodplain.

1.2.2 Local geology

The local geology consists of shallow subsurface soils consisting mainly of fill overlying alluvial deposits. The fill consists of silt, sand, gravel, crushed limestone, crushed brick, slag/cinders, wood chips, coal fines, crushed concrete, broken glass, and miscellaneous construction debris. The thickness of the fill ranges from 4 feet (on site) to 24 feet (adjacent to the Susquehanna River). Weathered bedrock (saprolite) is present in some areas as well. The underlying bedrock consists of limestone which exhibits fracture zones oriented roughly east to west. Karst topography (*i.e.*, solution of limestone) is not expressed to a significant degree (Atlantic Environmental Services, Inc., 1996).

1.2.3 General history

The Columbia Gas Company began production of gas from wood carbonization at the Columbia site in 1851. Sanborn Fire Insurance maps (1886-1904) depict two gas holders, an oil tank and a gas works building containing a water gas generator. In 1909, the gas works was rebuilt. In 1947, the relief holder failed and its foundation was used for tar separation. Operations at the MGP ceased in the 1950s, and the site was decommissioned sometime thereafter. The above ground structures were demolished and removed, and the holder foundations and tar separator were back-filled. In 1979, the property was sold to a local resident who began operation of a boatyard. In 1994, Pennsylvania Power and Light Company (PP&L) repurchased the eastern half of the property and the boat dealer moved his operation immediately northwest of the facility (Atlantic Environmental Services, Inc., 1996).

1.2.4 Site contamination

Surface (0-6") and subsurface soil samples, as well as groundwater samples, collected on site and off site indicate the presence of MGP-related wastes, including polycyclic aromatic hydrocarbons (PAHs). Susquehanna River near-shore sediments and surface water to the south of the site also contain MGP-related wastes. Some contaminants have also been detected in Shawnee Creek sediment and surface water, although it is unclear whether these compounds (mainly SVOCs and inorganic compounds) originate from the former MGP site or from other sources (the highest levels of contaminants were measured upgradient of the site).

Physical MGP-related impacts, such as tar, odor and sheen, have been observed in soils (overburden and bedrock) on site and near the downgradient trend of the bedrock fracture zone to the east bank of Shawnee Creek. Tar wastes have also been observed in the overburden to the northwest of the wastewater treatment plant near the Susquehanna River shoreline. Results of the Remedial Investigation (Atlantic Environmental Services, Inc., 1996) and previous studies suggest that migration of MGP-related wastes are constrained by the physical characteristics of the bedrock.

1.3 Site Conceptual Model

This section presents the site conceptual model, which provides a qualitative discussion of potential or suspected sources of MGP-related contaminants, the types of contaminants detected at the site, the contaminated media, and the potential exposure pathways and receptors. Some of these exposures are more likely to occur than others. In particular, the hypothetical resident exposure described in Sections 1.3.1 and 1.3.3 and is unlikely to occur based on the current land use and zoning.

There are four general areas where evidence of contamination has been documented: the former MGP facility, an area to the southwest of the site, a small area adjacent to the Susquehanna River, and sediments located along the near-shore area of the Susquehanna River. The baseline risk assessment evaluates potential exposure to MGP-related wastes in three of these areas. The fourth was recently remediated, and is not considered in the baseline risk assessment. These areas are delineated in Figure 3.

1.3.1 On-site areas of contamination

Contaminants in on-site surface and subsurface soils include volatile organic compounds, semi-volatile organic compounds, and inorganic compounds. Although the site is not currently in use, a trespasser could be exposed to on-site surface soils and fugitive dust. Under current zoning, the site could be used in the future for industrial or commercial purposes. A construction worker involved in a future excavation could be exposed to contaminants in surface and subsurface soil. A future industrial worker could be exposed to contaminants in surface soils.

A future on-site resident is not likely because the site is zoned industrial and the Columbia Borough zoning regulations (Article XI, Industrial District, Section 90.36) specifically prohibit residential use within an industrial district. However, we evaluated risks to a hypothetical future resident in the event local zoning regulations change. If residents were located in this area, they could potentially be exposed to chemicals in surface and subsurface soil (assuming reworking of soil). In the unlikely event that these residents installed a private drinking water well, they could be exposed to contaminants in groundwater.

1.3.2 Off-site areas of contamination (southwest of site)

Contaminated areas to the southwest of the site include a small area of land adjacent to the site and Shawnee Creek, located between Front Street and the rail lines south of the site, as shown in the dotted area of Figure 3. MGP-related chemicals detected in surface and subsurface soil in this area and in Shawnee Creek include volatile organic compounds, semi-volatile organic compounds, and inorganic compounds. It is possible that a trespasser could be exposed to surface soils, fugitive dust, sediment, and surface water in this general area. A smaller section of this area has elevated levels of semi-volatile organic compounds in the subsurface soil. A construction worker in this area could be exposed to subsurface and surface soils during subsurface construction activity.

1.3.3 Off-site areas of contamination (adjacent to the Susquehanna River)

Contaminants in this small area adjacent to the Susquehanna River include volatile organic compounds, semi-volatile organic compounds, and inorganic compounds in soil and groundwater. Exposure to chemicals in soil, fugitive dust, and vapor could occur in this area during construction activities. The area is zoned as "floodplain" and Columbia zoning regulations do not prohibit residential use within a floodplain district. It is possible, although unlikely, that a future resident could build a home and be present in this general area. If a resident were located in this area in the future, they could potentially be exposed to chemicals in surface and subsurface soil (assuming reworking of soil). In the unlikely event that these residents installed a private drinking water well, they could be exposed to contaminants in groundwater.

1.3.4 Off-site areas of contamination (near-shore reach of the Susquehanna River)

Volatile organic compounds, semi-volatile organic compounds and inorganic compounds were detected in sediment and surface water in a small (approximately 5,000 square feet) near shore area of the Susquehanna River. Exposure to these chemicals in sediment and surface water was previously considered possible during recreational boating or wading. However, removal of impacted sediments in the area occurred in January 1998. Because the source of contaminants has been removed, this exposure scenario is not quantitatively evaluated in the baseline risk assessment.

2.0 HAZARD IDENTIFICATION

The hazard identification section evaluates the quality and usability of the data for the Former UGI Columbia MGP site. The analytical data are described by media and a detailed screening process is conducted to select chemicals of potential concern.

2.1 Data Sources

The baseline human health risk assessment is based on site information and analytical data presented in the following reports:

- TRC Environmental Consultants, Inc. 1986. *Final Report of Investigations - Columbia Gas Plant Site*. Columbia, Pennsylvania. Volumes I & II;
- NUS Corporation. 1991. *Expanded Site Inspection of UGI Columbia Gas Plant.* Columbia, Pennsylvania. TDD No. F3-9011-56;
- Atlantic Environmental Services, Inc. 1995. *River Sediment Investigation - Former UGI Manufactured Gas Plant*. Columbia, Pennsylvania;
- Atlantic Environmental Services, a division of GEI Consulting, Inc. 1997. *Microscale Solvent Extraction Method Data Validation Reports and Analytical Data.* Columbia, Pennsylvania.
- META Environmental, Inc. December 1, 1997*Review of Remedial Investigation Data for the Former UGI Manufactured Gas Plant,* Columbia, Pennsylvania.
- Atlantic Environmental Services, Inc. 1998. *Remedial Investigation - Former UGI Manufactured Gas Plant*. Columbia, Pennsylvania. Volumes I through V;

The reports include analytical data for:

- soil (surface and subsurface; on site and off site);
- sediment (Susquehanna River and Shawnee Creek);
- surface water (Susquehanna River and Shawnee Creek); and
- groundwater (shallow wells and deep wells; on site and off site).

2.2 Analytical Data and Data Usability

The *Guidance for Data Useability in Risk Assessment* (USEPA, 1992a) provides guidance for the assessment and interpretation of environmental data for use in human health risk assessments.

The purpose of the data evaluation is to determine which data should be retained for consideration in the risk assessment.

The site investigation reports include site descriptions, sampling designs, the general characteristics of the environmental media and site environs. The documentation included in these reports ensures that the sample results are properly related to geographic locations. This includes information such as sampling and analysis plans, chain-of-custody records, standard operating procedures, and field/analytical records.

Accompanying the data is information about detection limits and analytical methods, which are reviewed as a part of the data usability process. Treatment of detection limits in the risk assessment is described in Section 3.3 of this report. Most of the analytical methods used are USEPA-approved methods; there are also data generated by microscale solvent extraction (MSE), and extraction procedures (EP). The EP extractable metals data are not used; however, the MSE data are used in the assessment (See Section 5.4). The MSE data was validated according to EPA protocols (Atlantic, 1997).

Review of the analytical data includes an evaluation of five data quality indicators:

- "U" qualifiers, which indicate that the compound was not detected at the reported detection limit;
- "R" qualifiers, which indicate that the value was rejected;
- "J" qualifiers, which indicates that the concentration is an estimated value;
- "B" qualifiers, which indicate that the concentration is a detected value between the instrument detection limit and the CRDL; and
- "C" qualifiers, which indicate that a compound co-eluted with another contaminant during analysis.

Data with the qualifiers "J", "B", and "C" are considered appropriate for characterizing conditions at the site and are retained for use in the risk assessment. Data with the qualifier "U" or the combination of "UJ" are considered non-detected values and are considered in the risk assessment. Values with the qualifier "R" are not used in the risk assessment.

2.3 Analytical Database for Risk Assessment

This section describes the data used in the risk assessment. Table 1 presents a summary of the compounds detected at least once in the sampled media. Appendix A presents the analytical data for the detected compounds.

2.3.1 Surface Soil

Appendix A-1 presents the analytical data for compounds detected in surface soil. Surface soil samples (0 to 6 inches deep) were collected from on-site and off-site locations during two site

investigations. All of the available surface soil data were used in the risk assessment because they met data useability requirements.

The NUS Corporation investigation (1991) of surface soil included the collection of four on-site samples and one off-site sample. The off-site sample was collected north of the site. Using USEPA methods, these five samples were analyzed for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), inorganic compounds (metals and cyanide), pesticides, and polychlorinated biphenyls (PCBs).

The Atlantic Environmental Services, Inc. investigation (1996) of surface soil included the collection of three on-site samples and five off-site samples. One off-site sample was collected northwest of the site, and the other four were collected south/southwest of the site between the site and the Susquehanna River. Using the MSE method, these eight samples were analyzed for monocyclic aromatic hydrocarbons (MAHs) and polycyclic aromatic hydrocarbons (PAHs). These samples were also analyzed for target analyte list (TAL) metals and cyanide.

Surface soil south of the railroad tracks was not analyzed because it consisted of fill and/or gravel and was assumed not to be a source of site-related contaminants (Clean Sites Environmental Services (CSES), personal communication, 1997).

2.3.2 Subsurface Soil

Appendix A-2 presents the analytical data for compounds detected in subsurface soil. Subsurface soil samples (deeper than 6 inches) were collected from on-site and off-site locations during three site investigations (TRC Environmental Consultants, Inc., 1986; NUS Corporation, 1991; Atlantic Environmental Services, Inc., 1998). To characterize on-site subsurface soil, the risk assessment uses all on-site samples collected at depths to 15 feet. To characterize off-site subsurface soil, the risk assessment uses samples that were collected within two off-site areas of elevated PAH concentrations and at depths to 15 feet. One off-site area is located at the bank of the Susquehanna River near the wastewater treatment plant, the other is located south of the site from Front Street, as shown in Figure 3. (Section 3.0 of this report describes the areas of exposure evaluated in the risk assessment.)

The TRC investigation of subsurface soil included the collection of two on-site samples. Using USEPA methods, the samples were analyzed for PAHs, cresols, phenols, sodium, EP extractable metals, and cyanide. These data have not been validated because they were conducted under voluntary action, and are not used in the risk assessment.

The following data sets were used to develop exposure point concentrations.

The NUS investigation of subsurface soil included the collection of one on-site sample. Using USEPA methods, the sample was analyzed for VOCs, SVOCs, inorganic compounds (metals and cyanide), pesticides, and PCBs.

The Atlantic investigation of subsurface soil included the collection of eight on-site samples and eight off-site samples. Five of the off-site samples were from soil borings and three were from test pits. Four off-site samples were located near the bank of the Susquehanna River, and four near Front Street and the railroad tracks south of the site. Using the MSE method, these 16 samples were analyzed for mono-cyclic aromatic hydrocarbons (MAHs) and PAHs. The samples were also analyzed for TAL metals and cyanide.

2.3.3 Sediment

Surface sediment samples (0 to 4 feet deep) were collected from the Susquehanna River and Shawnee Creek. The contamination in the Susquehanna River sediment was excavated during a removal action which occurred in January 1998, therefore the river surface water and sediment are not evaluated quantitatively in the risk assessment. To characterize the creek sediment, the risk assessment uses all samples collected from the creek.

Appendix A-3 presents the analytical data for compounds detected in Susquehanna River sediment. Ten surface sediment samples were collected from the river during three investigations (TRC Environmental Consultants, Inc., 1986; NUS Corporation, 1991; Atlantic Environmental Services, Inc., 1995). The TRC investigation included the collection of two sediment samples, which were analyzed for selected VOCs, SVOCs and inorganic compounds, using USEPA methods. The NUS investigation included the collection of three sediment samples, which were analyzed for VOCs, SVOCs and inorganic compounds, using USEPA methods. The Atlantic investigation included the collection of five sediment samples, which were analyzed for MAHs and PAHs using the MSE method.

Appendix A-4 presents the analytical data for compounds detected in Shawnee Creek sediment. Three sediment samples were collected from the creek during the Atlantic Environmental Services, Inc. investigation (1996). Using USEPA methods, these samples were analyzed for selected VOCs, SVOCs, TAL metals, and cyanide. Sample SED-2 had a field duplicate, also referred to as SED-4. We compared the duplicate analysis for this sample, and chose to use the duplicate results to represent sample COPC concentrations in the risk assessment because the concentrations were higher.

2.3.4 Surface Water

Surface water samples were collected from the Susquehanna River and Shawnee Creek. To characterize the creek surface water, the risk assessment uses all samples collected from the creek.

Appendix A-5 presents the analytical data for compounds detected in Susquehanna River surface water. Five surface water samples were collected from the river during two investigations (TRC Environmental Consultants, Inc., 1986; NUS Corporation, 1991). The TRC investigation

included the collection of two surface water samples, which were analyzed for selected VOCs, SVOCs, and inorganic compounds using USEPA methods. The NUS investigation included the collection of three surface water samples, which were analyzed for VOCs, SVOCs, and inorganic compounds using USEPA methods.

Appendix A-6 presents the analytical data for compounds detected in Shawnee Creek surface water. Three surface water samples were collected from the creek during the Atlantic Environmental Services, Inc. investigation (1996). Using USEPA methods, these samples were analyzed for selected VOCs, SVOCs, TAL metals, and cyanide.

2.3.5 Groundwater

Appendix A-7 presents the analytical data for compounds detected in groundwater. Groundwater samples were collected from on-site and off-site locations during three site investigations. This section describes the most recent data from each monitoring well, and is comprised of unfiltered samples. Section 3.0 of this report describes which sample locations are used in the risk assessment.

The TRC Environmental Consultants, Inc. investigation (1986) provides the most recent data for MW-2, an off-site monitoring well. Using USEPA methods, the sample was analyzed for base neutral, pesticide, phenolic, and inorganic compounds. Because the data are not validated, they are not used in the risk assessment.

The following data sets were used to derive exposure point concentrations.

The NUS Corporation investigation (1991) provides the most recent data for MW-1S (shallow), MW-1D (deep), and MW-3S (shallow). Well pair MW-1S/MW-1D is located off site in a direction hydrogeologically upgradient of the site (north of the site). Well MW-3S is located on site. Using USEPA methods, groundwater samples from these wells were analyzed for VOCs, SVOCs, and inorganic compounds.

The Atlantic Environmental Services, Inc. investigation (1996) of groundwater included the collection of a round of samples in March 1995 and a second round in July 1995. During each of these sampling rounds, a sample was collected from 11 off-site locations: MW-1SR (replacement well), MW-1DR (replacement well), MW-2R (replacement well), MW-4, MW-5, MW-6S, MW-6D, MW-7S, MW-7D, MW-8S, and MW-8D. Also collected were two samples from an on-site location (MW-3D), and one sample from each of two cooling water wells at the Lancaster Water Authority (LWA) pumping station (CWW-1, CWW-2). Using the MSE method, the 26 samples were analyzed for MAHs and PAHs. These samples were also analyzed for TAL metals and cyanide.

2.4 Screening for Chemicals of Potential Concern

Following an evaluation of the useability of the analytical data, the compounds detected in each medium were carried through a screening process to select chemicals of potential concern (COPCs). The screening was conducted according to the USEPA Region III Technical Guidance Manual, *Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening* (1993a). The 1993 document recommends that the process include four steps:

1. evaluating data quality (Section 2.1 above);
2. reducing the data set using risk-based concentrations (RBCs);
3. considering the re-inclusion of eliminated chemicals based on factors such as historical information, toxicity, etc.; and
4. making further data set reductions for contaminants that are present at concentrations consistent with background levels or essential nutrients and/or not likely to be toxic at the detected concentrations.

The screening process for selecting COPCs is conducted separately for organic and inorganic compounds in soil, sediment, surface water, and groundwater. Tables 2 through 5 present the screening for organic and inorganic compounds detected in soil. Tables 6 and 7 present the screening for organic and inorganic compounds detected in sediment. Table 8 presents the screening for organic and inorganic compounds detected in surface water. No organic constituents were detected in Shawnee Creek surface water. Tables 9 and 10 present the screening for organic and inorganic compounds detected in groundwater.

2.4.1 Risk-based screening

The risk-based screening was conducted using the USEPA Region III Risk-Based Concentration Table (1997b). The RBCs are available for compounds in air, drinking water, fish tissue, and soil. They are intended for the protection of human health and are derived using a systemic hazard quotient of 1 or a lifetime cancer risk of 1×10^{-6}. The maximum concentration of compounds detected in each medium is compared to the appropriate RBC. We multiplied each RBC based on non-cancer effects by a factor of 0.1 to derive a concentration equal to a hazard quotient of 0.1. Each compound is either excluded from further evaluation (if the compound was not detected at least once or if the maximum concentration is less than the RBC), or retained for use in the risk assessment (if the maximum concentration is greater than the RBC or if a RBC does not exist).

Compounds detected in soil or sediment are compared to residential RBCs for soil. Use of the soil RBCs for soil and sediment is appropriate because the RBCs are based on incidental ingestion, a pathway that is evaluated using the same methodology for both soil and sediment. Use of the residential RBCs rather than industrial RBCs provides a conservative comparison because residential use of the study area is unlikely due to zoning restrictions and surrounding

land use. Compounds detected in surface water and groundwater are compared to RBCs for drinking water.

The results of the risk based screening are discussed in Section 2.5.

2.4.2 Re-inclusion of eliminated compounds

The re-inclusion component of the screening process evaluates whether compounds eliminated during risk-based screening should still be included as COPCs in the assessment for other reasons. No compounds eliminated from the risk-based screening are re-incorporated in the risk assessment.

2.4.3 Further data set reductions

Background comparison: No data set reductions are made based on a comparison to background, because site-specific background data is insufficient to determine background concentrations in soils. Sediment sample SD-1 in Shawnee Creek appears to represent anthropogenic background, but can not be used to eliminate COPCs in Shawnee Creek because only one sample is available and it may not be statistically representative. Groundwater samples MW-01S and MW-01D are located upgradient of the site, and are also considered representative of site-specific background groundwater conditions. These samples were not included in the risk based screening, but were also not used to represent site-specific background concentrations to screen out contaminants in their respective media. Therefore all COPCs exceeding risk-based concentrations are carried through the risk assessment, even though they may be present at levels consistent with background.

Essential nutrients: The final component of the screening process is to exclude metals that are considered essential nutrients i.e. calcium, magnesium, potassium, and sodium, (EPA, 1989).

2.5 Chemicals of Potential Concern

Chemicals of potential concern are compounds that are retained for further evaluation in the risk assessment based on the results of the screening process described in the previous section. Table 12 summarizes the COPCs for each medium.

The list of COPCs in Surface Soils are: Acenaphthylene, Benzo(a)anthracene, Benzo(b) fluoranthene, Benzo(g,h,i)perylene, Benzo(a)pyrene, Dibenzo(a,h)anthracene, Indeno(1,2,3-cd) pyrene, Aluminum, Arsenic, Beryllium, Cadmium, Iron, Lead, and Manganese. Acenaphthylene and benzo(g,h,i)perylene will be evaluated qualitatively as recommended by NCEA (2/95) [cited by EPA, March 1998].

The list of COPCs in Subsurface Soil are: Acenaphthylene, Benzo(a)anthracene, Benzo(b) fluoranthene, Benzo(k)fluoranthene, Benzo(g,h,i)perylene, Benzo(a)pyrene, Dibenzo(a,h)

anthracene, Indeno(1,2,3-cd)pyrene, Aluminum, Arsenic, Beryllium, Iron. Manganese, and Thallium. Acenaphthylene and benzo(g,h,i)perylene will be evaluated qualitatively as recommended by NCEA (2/23/95).

The list of COPCs in Sediments are: Benzo(a)anthracene, Benzo(b)fluoranthene, Benzo(g,h,i)perylene, Benzo(a)pyrene, Aluminum, Arsenic, Copper, Iron. and Manganese. Benzo(g,h,i)perylene will be evaluated qualitatively as recommended by NCEA (2/23/95).

There are no COPCs in Surface Water.

The list of COPCs in Groundwater are: Benzene, Ethylbenzene, Tetrachloroethene, Toluene, Trichloroethene, 1,2,4-Trimethylbenzene, Xylenes, (total), Acenaphthene, Acenaphthylene, Benzo(a)anthracene, Benzo(b)fluoranthene, Benzo(a)pyrene, Chrysene, Fluoranthene, 1-Methylnaphthalene, 2-Methylnaphthalene, Naphthalene, Phenanthrene, Pyrene, Bis(2-ehtylhexyl)phthalate, Dibenzofuran, Aluminum, Barium, Cyanide, Iron, Lead, and Manganese. Acenaphthylene will be evaluated qualitatively (NCEA (2/23/95).

3.0 EXPOSURE ASSESSMENT

The objective of the exposure assessment is to evaluate potential exposures to COPCs. This is accomplished by analyzing contaminant releases, identifying exposed populations, identifying potential exposure pathways, estimating exposure concentrations for complete exposure pathways, and estimating contaminant doses for complete exposure pathways.

The USEPA has adopted an approach for exposure assessment based on reasonable maximum exposure (RME). An RME is the highest exposure that is reasonably expected to occur at a site, and is estimated for individual pathways and scenarios. Risks based on the RME are intended to represent upper bound risks for potential receptors. Actual risks are expected to be lower than estimated by the RME because of the conservatism in the analysis.

3.1 Potential Exposure Pathways

An exposure pathway is complete if there is a source or chemical release from a source, an exposure point where contact can occur, and an exposure route by which contact can occur. Exposure pathways are identified for potentially exposed populations by considering the source of contaminants, locations of contaminants or exposure points, and the likelihood of exposure to the contaminants at the exposure points. The primary exposure routes evaluated in this risk assessment are ingestion and dermal contact of contaminated soil and sediment, and inhalation of fugitive dusts and vapors. In addition, we also consider ingestion, inhalation, and dermal contact with contaminated groundwater under a hypothetical future use scenario.

3.2 Exposure Scenarios

Table 13 summarizes the current and future exposure scenarios evaluated in this assessment. Included in the table is a description of the exposure media, pathways, and sample locations associated with each scenario. The exposure scenarios considered in this report include one that evaluates current land use:

- a trespasser,

three that evaluate likely potential future land use:

- an on-site industrial worker,
- an on-site construction worker,
- an off-site construction worker,

and two that evaluate very unlikely but possible future land use:

- an, on-site hypothetical resident and
- an off-site hypothetical resident.

These scenarios address reasonable maximum exposures in three of the four areas where evidence of contamination has been documented, on-site, off-site to the south and southwest, and near the Susquehanna River. We did not evaluate exposure to contaminated sediments near the Susquehanna River because of the January 1998 remediation of contamination there. This section provides the rationale and description for each scenario.

3.2.1 Trespassers

Although the site is surrounded by a chain link fence, it is possible that a teenager (age 11 to 18) could trespass on the property. This activity is assumed to include exposure on the property, west and south of the property, and in Shawnee Creek during warm weather months. Exposure to surface soil (0 to 6 inches deep) is assumed to occur via incidental ingestion, dermal contact, and inhalation of fugitive dust. Exposure to Shawnee Creek sediment is assumed to occur via incidental ingestion and dermal contact. There were no COPCs detected in Shawnee Creek surface water. Table 14A presents the exposure assumptions for this scenario. Figure 4 illustrates the sampling locations used to quantify exposure for this scenario.

3.2.2 Future industrial workers

Based on the current zoning laws, land use of the site is restricted to industrial use. Therefore, industrial workers are the receptor group most likely to contact on-site contaminants in the future. The scenario considers activity such as maintenance work and/or site walkovers. Exposure to on-site surface soil is assumed to occur via incidental ingestion, dermal contact, and inhalation of fugitive dust. Exposure to subsurface soil occurs via inhalation of vapors. Direct contact with subsurface soils is not evaluated for the future industrial worker because these exposures are not likely to occur during routine work habits. Table 14B presents the exposure assumptions for this scenario. Figure 5 illustrates the sampling locations used to quantify exposure for this scenario.

3.2.3 Future on-site construction workers

Future activity at the site could involve construction activities. The scenario considers activity during an excavation project that could include exposure to surface and subsurface soil (depths to 15 feet). Exposure to soil is assumed to occur via incidental ingestion, dermal contact, and inhalation of fugitive dust. Inhalation of soil gas is also evaluated for volatile constituents detected in soil using the Soil Screening Guidance (EPA, 1996b). Table 14C presents the exposure assumptions for the construction worker scenarios. Figure 6 illustrates the on-site sampling locations used to quantify exposure for this scenario.

3.2.4 Future off-site construction workers

Future activity in off-site areas could involve construction activities. The off-site construction worker is assumed to contact subsurface soil (depths to 15 feet) near the Susquehanna River and in the area between Front Street and the Railroad tracks. These locations were selected based on the elevated concentrations of PAHs in the area. Because of the presence of fill or gravel, contamination is not likely to be located in surface soil, hence exposure to off-site surface soil is not evaluated (personal communication, CSES, October, 1997). Exposure to subsurface soil is assumed to occur via incidental ingestion, and dermal contact. Inhalation of soil gas is also evaluated for volatile constituents detected in soil using the Soil Screening Guidance (EPA, 1996b) according to equations presented in Appendix C. Table 14C presents the exposure assumptions for the construction worker scenarios. Figure 6 illustrates the off-site sampling locations used to develop the two exposure point concentrations for this scenario.

3.2.5 Current residents

Private wells: Several residences located approximately one quarter mile to the northwest of the site currently use groundwater from private wells. The private wells were sampled during the Atlantic Environmental Services, Inc. investigation (1996). The groundwater samples were analyzed for site-related contaminants; there were no COPCs detected in these wells. Transport of COPCs toward these residences appears unlikely based on the location of the wells, the low mobility of the MGP-related chemicals, the lack of COPCs in wells MW-1S and MW-1D between residents and the site, and the nature and direction of the fractures in bedrock (Atlantic Environmental Services, Inc., 1996).

LWA pumping station: Residents in the Lancaster, Pennsylvania area receive water drawn from the Susquehanna River at the LWA pumping station upstream of the site. The main intake structure is located approximately 400 feet off shore, and a second intake structure (used only during emergencies such as flooding, etc.) is located on the shoreline. The emergency intake structure is located near the apparent extension of a probable fracture zone (Atlantic Environmental Services, Inc., 1996). The river water receives initial treatment at the pumping station and is then pumped to the main treatment facility located off of 12th Street.

The NUS Corporation investigation (1991) included a surface water sample collected from the emergency intake structure; there were no PAHs detected. The Atlantic Environmental Services, Inc. report (1996) indicates that it is unlikely that tar is discharging to the river near the pumping station. Bathymetric data demonstrate that the elevation of the river bottom at that location is significantly higher than the elevations at which tar has been observed in the area (Atlantic Environmental Services, Inc., 1996). It is unlikely that residents receiving Susquehanna River water are being exposed to site-related COPCs.

3.2.6 Future hypothetical residents

Although no residences currently exist between the site and the Susquehanna River, EPA requested we evaluate risk to a future hypothetical resident at an off-site area, located south of the site near the Susquehanna River and the wastewater treatment plant. The area of exposure for this scenario is zoned as floodplain and contains elevated concentrations of PAHs in soil and groundwater. Evaluation of this scenario is conservative because it is unlikely that a residence would be built in this area due to the restrictive nature of floodplain regulations and surrounding land use (a municipal garage, wastewater treatment plant, and municipal water intake). For this scenario, we assume the off-site resident would be exposed to contaminants in subsurface soil through ingestion, dust inhalation, vapor inhalation, and dermal contact following soil reworking, and groundwater through ingestion, inhalation and dermal contact. Off-site surface soil was not evaluated because it is fill material or gravel, and no sampling data is available.

Based on the current zoning law, land use at the site is restricted to industrial use. While zoning and site conditions indicate the site would most likely be used for industrial purposes in the future, EPA requested we evaluate a future on-site residential scenario in the Baseline Risk Assessment, because there is no existing agreement to limit future site use (EPA, 1997a). This scenario is not consistent with the site conceptual model and is calculated based on EPA's request. For this scenario, we assume the on-site resident would be exposed to surface soil through ingestion, dust inhalation, and dermal contact, subsurface soils through vapor inhalation, and groundwater through ingestion, inhalation and dermal contact.

The predominant pathway for the residential scenario is ingestion of groundwater via daily use of a private well. Exposure to on-site or off-site surface soil (including depths to 15 feet) is evaluated via incidental ingestion, dermal contact, and inhalation of fugitive dust.

To evaluate the on-site and off-site resident scenario, it is conservatively assumed that the resident will live their first 30 years at the same location (USEPA, 1991). These 30 years are evaluated in two age ranges: 0 to 6 years, and 6 to 30 years. This provides a conservative assessment by evaluating separately the ages at which an individual is most sensitive. Table 16F presents the exposure assumptions for the off-site resident scenario. The on-site resident scenario is located in Appendix G. Figure 7 illustrates the sampling locations used to quantify exposure for this scenario. Table 15 presents the rationale for the selection of groundwater wells used in the future residential scenarios.

3.3 Estimating Exposure Point Concentrations

We use an arithmetic mean for the central tendency estimate (CTE) and the 95 percent upper confidence limit (UCL_{95}) on the mean for the reasonable maximum exposure (RME) to estimate exposure point concentrations. This approach was agreed upon by Menzie-Cura & Associates, Inc. and USEPA Region III staff at a meeting in December, 1996. The arithmetic mean represents a reasonable CTE estimate of the concentration likely to be contacted over time

because "assuming long-term contact with the maximum concentration is not reasonable" (EPA, 1992b). Use of the UCL_{95} is consistent with the RME concept, since it is unlikely that actual CTE concentrations are higher than this statistical estimate. In cases where the UCL_{95} is greater than the maximum detected concentration, the maximum value is used as the RME exposure point concentration. To calculate CTE concentrations for the CTE case, a value equal to one half of the detection limit is used for samples where the compound was not detected. As recommended by USEPA (1989a), cases in which high detection limits are greater than the maximum detected value, the non-detect concentration is excluded from the calculation.

3.3.1 Calculating the 95% upper confidence limit

The 95% upper confidence limit on the mean was calculated for log-normal distributions (analytical data for the site exhibit log-normal distributions when plotted). The formula to calculate the 95% upper confidence limit for log-normal distributions is (USEPA, 1992b):

$$UCL_{95} = e^{\left(\bar{x} + \frac{s^2}{2} + \frac{s\,H}{\sqrt{n-1}}\right)}$$

where:

e = Constant (base of the natural log)
$\bar{x}$ = Mean of the transformed data
s = Standard deviation of the transformed data
H = H-statistic (*e.g.*, from table published in Gilbert, 1987)
n = Number of samples

Appendix B contains the summary statistics for all detected compounds (*e.g.*, number of samples, number of detects, range of detected concentrations, CTE concentration, UCL_{95}, and RME concentration). Refer to Table 13 for sample locations used to calculate exposure point concentrations for each scenario. The list of COCs for each exposure point is summarized in Section 2.5 and Appendix E. Tables 16A-16F summarize the exposure point concentrations for each scenario.

3.3.2 Fugitive dust

Fugitive dust generated during excavation and other earth moving activities could be inhaled by potential receptors. Because concentrations of COPCs in fugitive dust were not measured at the site, we modeled these concentrations based on levels in soil.

Exposure point concentrations for fugitive dust are estimated by adapting an the equation from the Soil Screening Guidance (EPA, 1996b):

$$EPC_{dust} = \frac{EPC_{soil}}{PEF}$$

where:

EPC_{dust} = Exposure point concentration in fugitive dust (mg/m^3)
EPC_{soil} = Exposure point concentration in soil (mg/kg)
PEF = Particulate Emission Factor (m^3/kg)

We conservatively assume that the site contributes 100% of the particulate concentration, even though there are known to be multiple contributors to the total particulate concentration.

We calculated a PEF value of 1.044 E9 m^3/kg during construction activities in accordance with EPA guidance (1996b). To do this, we applied a Q/C factor for a 1 acre site in Harrisburg, PA of 71.87 g/m^2 per kg/m^3 rather than the default 90.87 g/m^2 per kg/m^3 Q/C value used to calculate the default PEF value for a one half acre site located in Minneapolis, MN. The equation presented in the Soil Screening Guidance is incorrect, so we are unable to show the calculation. Instead, we divided the default PEF by the default Q/C factor, and then multiplied by the Harrisburg Q/C factor. This calculation is shown in Appendix C.

3.3.3 Vapor

Potential receptors could breathe chemical vapors released from subsurface soil to ambient air. Because chemical vapor levels were not measured, we model these exposure point concentrations based on soil concentrations. Soil vapor concentrations were estimated by adapting the approach in the Soil Screening Guidance (EPA, 1996b) using the following equation:

$$EPC_{vapor} = \frac{EPC_{soil}}{VF}$$

where:

EPC_{soil} = RME or CTE concentration in subsurface soil (mg/kg)
VF = Volatilization Factor (m^3/kg)

The Volatilization Factor (VF) is calculated on a chemical specific basis using default soil properties as outlined in the Soil Screening Guidance (EPA, 1996b), according to the following equation:

$$VF = \frac{Q}{C} \times \frac{(3.14 \times D_a \times T)^{0.5}}{(2 \times \rho_b \times D_a)} x10^{-4} m^2/cm^2$$

where:

Q/C = the release rate,(g/m^2-s per kg/m^3)
T = exposure interval (sec)
ρ_b = dry soil bulk density , (g/cm^3)

$$\text{and } D_a = \frac{[(\theta_a^{10/3} D_i H' + \theta_w^{10/3} D_w)/n^2])}{\rho_b K_d + \theta_w + \theta_a H'}$$

where

θ_a = air filled porosity (L_{air}/L_{soil})
θ_w = water filled porosity (L_{water}/L_{soil})
D_i = diffusivity in air(cm^2/s)
D_w = diffusivity in water (cm^2/s)
H' = Henry's Law Constant (dimensionless)
n = total soil porosity (L_{pore}/L_{soil})

EPA (1996b) provides default parameters for many of the inputs. The following default factors were incorporated in the calculation of the volatilization factor:

Q/C = 71.87 g/m^2-s per kg/m^3, (one acre site near Harrisburg).
T = 9.5 x 10^8 s (30 years)
ρ_b = 1.5 g/cm^3
θ_a = 0.28
θ_w = 0.15
n = 0.43

The chemical and site-specific factors for the VF equations are presented in Appendix C.

3.4 Dermal Absorption

To evaluate the uptake of contaminants in soil by dermal contact, dermal absorption is estimated from the exposed skin area multiplied by the soil loading on skin, and a chemical specific absorption factor described in Section 3.5. Kissel *et al.*, (1996) found that soil loading rates on skin varied by the type of activity and the part of the body exposed. We incorporated Kissel's findings into the calculation of dermal absorption of contaminants in soil.

Each receptor in this risk assessment, except the child resident, was assigned a representative activity from the Kissel activity group for measured soil loading. The average of the reported maximum soil loading for each representative body part was multiplied by the average surface area for that body part. The total soil adhered to the skin was then divided by the total skin surface area exposed. This is shown in Appendix C. The dermal soil loading is weighted by

body part, using the following study population paired with each site receptor. The arithmetic average of the reported maximum adherence in each group was used as the adherence factor, except for Kissel's irrigation worker, where there was only one group. For the child resident, we use the default adherence factor of 1 mg/cm^2 per EPA's request (EPA, 3/11/98).

SCENARIO	KISSEL ACTIVITY GROUP
Trespasser	Soccer Player
Resident	Soccer Player
Industrial Worker	Groundskeeper
Construction worker	Irrigation Worker

Appendix C shows the source table from Kissel *et al.* (1996) used for calculations. In Table 16 E, exposure frequencies for dermal exposure are presented twice, to represent differing assumptions regarding the percentage of the body exposed.

3.5 Estimating Average Daily Doses

Average Daily Doses (ADDs) of the chemicals from each exposure route and exposure point are estimated using standard chemical intake equations and a combination of standard and site-specific exposure assumptions. The general form of the ADD equation is as follows:

$$ADD = \frac{Total\ Amount\ of\ Contaminant\ Intake}{(Body\ Weight_{average})\ (Averaging\ Period)} \times Absorption\ Factor$$

Two ADDs are calculated for each exposure route: the ADD(year) and the ADD(life). The ADD(year) is used to evaluate non-carcinogenic effects; it represents the chemical dose during the exposure period and is calculated as the average daily dose over an appropriate averaging period. The ADD(life) is used to evaluate carcinogenic effects; it represents the chemical dose averaged over a lifetime and is calculated as the average daily dose over a 70-year lifetime.

Duration of the averaging period is significant because different effects may be manifested at different dose levels, and over different durations. The averaging period is important for effects for which there may be thresholds; thresholds are defined as the exposure dose below which deleterious effects are not likely for even the most sensitive populations. Exposures to probable carcinogens are expressed as lifetime average daily doses. Probable carcinogens are not considered to have thresholds because any exposure is assumed to present some risk.

Absorption factors describe the bioavailability of a compound. When chemicals are present in a soil matrix, their bioavailability is reduced relative to administration of a pure chemical. For the ingestion of soil, sediment, and water, 100% absorption is conservatively assumed. The assessment also assumes 100% absorption for inhalation of fugitive dust and vapors. For the

dermal absorption of contaminants in soil and sediment, values suggested by USEPA Region III (1995a) are used; the guidance includes values for benzene (0.05%), other VOCs (3%), SVOCs (10%), arsenic (3.2%), cadmium (1%), and other metals (1%). For the dermal absorption of contaminants in water, USEPA guidance (*Dermal Exposure Assessment: Principles and Applications*, 1992d) suggests a method that uses contaminant-specific permeability constants. Table 11 presents the absorption factors for each pathway and each medium.

The ADD equations used in the risk assessment are presented below.

3.5.1 ADD for soil/sediment ingestion

$$ADD_{ingestion}(mg/kg-day) = \frac{EPC \times IR \times RAF \times EF \times ED}{BW \times AP \times CF}$$

where:

EPC = Exposure point concentration for soil/sediment (mg/kg)
IR = Ingestion rate (mg/day)
RAF = Relative absorption factor (unitless; assumed to be 100%)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Average body weight of receptor (kg)
AP = Averaging period (days)
CF = Units conversion factor (10^6 mg/kg)

3.5.2 ADD for soil/sediment dermal contact

$$ADD_{dermalcontact}(mg/kg-day) = \frac{EPC \times TSA \times FSA \times AF \times RAF \times EF \times ED}{BW \times AP \times CF}$$

where:

EPC = Exposure point concentration for soil/sediment (mg/kg)
TSA = Total skin area (cm^2)
FSA = Fraction of skin area exposed (1/day)
AF = Adherence factor (mg/cm^2)
RAF = Relative absorption factor (unitless; contaminant specific)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Average body weight of receptor (kg)
AP = Averaging period (days)

CF = Units conversion factor (10^6 mg/kg)

For each body part exposed, the FSA exposed is multiplied by the AF from Kissel *et al.* (1996).

3.5.3 Average Daily Exposure for inhalation of fugitive dust

$$ADE_{dust}(mg/m^3) = \frac{EPC_{dust} \times EF \times ED}{AP \times CF}$$

where:

EF = Exposure frequency (days/year)
ED = Exposure duration (years)
AP = Averaging period (days)
CF = Units conversion factor (1 for non-cancer, 1000 µg/mg for lifetime ADE)

3.5.4 Average Daily Exposure for inhalation of vapors

$$ADE_{vapor\,inhalation}(mg/m^3) = \frac{EPC_{vapor} \times EF \times ED}{AP \times CF}$$

where:

EPC_{vapor} = Exposure point concentration for air (mg/m^3)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
AP = Averaging period (days)
CF = Units conversion factor (1)

3.5.5 ADD for groundwater (drinking water) ingestion

$$\mathrm{ADD}_{gw\,ingestion}(\mathrm{mg/kg-day}) = \frac{\mathrm{EPC_{gw}} \times \mathrm{IR} \times \mathrm{RAF} \times \mathrm{EF} \times \mathrm{ED}}{\mathrm{BW} \times \mathrm{AP} \times \mathrm{CF}}$$

where:

EPC_{gw} = Exposure point concentration for groundwater (mg/L)
IR = Groundwater ingestion rate (L/day)
RAF = Relative absorption factor (unitless; assumed to be 100%)
EF = Exposure frequency (days/year)

ED = Exposure duration (years)
BW = Average body weight of receptor (kg)
AP = Averaging period (days)
CF = Units conversion factor (1)

4.0 TOXICITY ASSESSMENT

The potential toxic effects of a chemical are evaluated through a review of available data that relate its observed toxic effects to doses at which these effects occur. The toxicity assessment consists of two components; the determination as to whether a chemical results in observed toxic effects in animals or humans and, the dose-response assessment, which relates the chemical's observed toxic effects to doses at which those effects occur. The toxicity assessment considers the following information:

- potential non-cancer health effects of compounds; and,
- potential for compounds to cause cancer.

Quantitative estimates of a chemical's toxicity are referred to as toxicity factors. The following toxicity factors are used to evaluate the toxic effects of compounds:

- reference doses or reference concentrations at which effects have not been observed for non-carcinogenic compounds; or
- carcinogenic slope factors for carcinogenic compounds.

Toxicity factors were identified from the following USEPA databases:

- USEPA (1996c) Integrated Risk Information System (IRIS); and
- USEPA (1995b) Health Effects Assessment Summary Tables (HEAST).

These databases provide toxicity factors for the inhalation and oral routes of exposure. Toxicity factors for oral routes of exposure are adjusted to evaluate the dermal exposure pathway, as discussed in Section 4.4.

Table 11 presents the reference doses and cancer slope factors for each of the COPCs and identifies the sources of these toxicity factors.

4.1 Non-carcinogenic Health Effects

Contaminants detected at the site may have the potential for effects that are non-carcinogenic in nature. The toxicity factors which express non-carcinogenic effects are reference doses (RfDs) or reference concentrations (RfCs). The non-cancer hazard indexes for oral and dermal routes of exposure are calculated using RfDs. The non-cancer hazard indexes for inhalation routes of exposure are calculated using RfCs.

The assessment of non-carcinogenic effects is complex. There is a broad interaction of time scales with types of effects (subchronic and chronic). Subchronic and chronic health effects are those that might occur as a result of longer-term exposure. USEPA defines subchronic exposure as up to seven years. Chronic exposure is defined as greater than seven years. In various risk assessment guidance documents, most of the attention focuses on evaluating the consequences of chronic exposure to various compounds. USEPA has focused its efforts at establishing reference doses and reference concentrations for chronic exposures.

The RfD values for chemicals serve as benchmarks for assessing the potential subchronic and chronic non-carcinogenic health effects. They represent "threshold" levels below which no adverse health effects are anticipated to occur due to exposure over a lifetime. The smaller the RfD, the lower the threshold is, or the more potent the compound. Safety factors (*e.g.*, uncertainty factors and modifying factors) are applied to the supporting data base to ensure that these benchmarks are sufficiently protective.

A reference dose is defined in the USEPA Integrated Risk Information System as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that may be without an appreciable risk of deleterious effects during a lifetime. The critical effect refers to the health endpoint upon which the reference dose is based. Uncertainty factors are incorporated in the reference doses as divisors to the dose associated with the critical effect, which is usually a No Observed Adverse Effect Level (NOAEL) or a Lowest Observed Adverse Effect Level (LOAEL). NOAELS or LOAELS may be divided by the following standardized uncertainty factors:

- 10-fold factor for extrapolation from animals to humans;
- 10-fold factor for variability in the human population;
- 10-fold factor for use of a less-than-chronic study; and
- 1 to 10-fold factor for extrapolation from a LOAEL.

The use of ten-fold uncertainty factors is traditional. However, there may be situations where data support the application of smaller uncertainty factors.

Modifying factors also contribute as divisors to the NOAEL or LOAEL. The IRIS review group uses a modifying factor based on collective professional judgment to further adjust the reference dose. A modifying factor may be applied when the study design was less than adequate.

The result of applying various multiples of 10 is that for many compounds, the RfD is calculated to be a factor of 100 less than the NOAEL. For other compounds, the resulting RfD is as much as a factor of 1000 less than the NOAEL.

The USEPA has published ingestion RfD values for some but not all chemicals. Substitutions are identified in the notes in Table 11. For this assessment, the oral RfD values for naphthalene

were selected to represent toxicity values for 1-methylnaphthalene and 2-methylnaphthalene, pyrene was used to represent phenanthrene toxicity per EPA Region III recommendation (EPA, 3/11/98). Also, based on NCEA recommendation, benzo(g,h,i)perlyene and acenaphthylene toxicity is evaluated qualitatively due to lack of experimental data to base the derivation of a reference dose (NCEA; 2/23/95, cited by EPA Region III, March 11, 1998). Reference doses for trichloroethene, aluminum, and iron were adopted from EPA Region III Risk Based Screening Table (EPA, 1997b). RfDs are not available for all time-frames of exposure, *i.e.*, subchronic and chronic. Chronic RfDs were used to evaluate subchronic exposures where a subchronic value was not available. Assigning RfD values to other compounds for which no values exist introduces uncertainty into the analysis but is judged to be more appropriate than ignoring these compounds altogether.

EPA also publishes adjustment factors for the conversion of oral RfDs to Dermal RfDs. These values are shown in Table 11.

Reference concentrations represent a contaminant concentration in air that does not pose a significant adverse health effect to humans RfCs are developed as inhalation RfDs in units of mg/kg-day. In Tables 11A and 11B, we convert the inhalation RfDs to RfCs by the following equation:

$$RfC_{inhalation}(mg/m^3) = \frac{RfD_{inh}(mg/kg-day) \times BW(70kg)}{IR(20m3/day)}$$

where:

BW = body weight
IR = inhalation rate

4.2 Carcinogenic Health Effects

Carcinogenicity is described in two ways: (1) through the USEPA Weight-of-Evidence classification scheme (A through E), which provides information on the type and quantity of data available; and (2) the Cancer Slope Factor (CSF) in $(mg/kg\text{-}day)^{-1}$ which provides a quantitative estimate of the carcinogenic potency of the contaminant to humans. This assessment estimates cancer risk for chemicals of potential concern classified as Group A, B1, or B2.

The weight of evidence regarding the potential carcinogenicity of a compound varies as a result of variations in the available test data, adequacy of studies, types of studies, and observed responses. These factors are taken into account by USEPA in assigning weight-of-evidence categories for characterizing carcinogenic compounds. As described in the documentation supporting the IRIS database, chemicals that give rise to cancer and/or gene mutations are generally classified by USEPA as follows:

- Group A: Human Carcinogen, sufficient human data;
- Group B1: Probable Human Carcinogen, limited human data;
- Group B2: Probable Human Carcinogen, sufficient evidence in animals and limited evidence or no evidence in humans;
- Group C: Possible Human Carcinogen, limited evidence in animals and limited or no evidence in humans;
- Group D: Not Classifiable as to Human Carcinogenicity, insufficient tests for carcinogenesis or mutagenesis are available; and,
- Group E: Evidence of Non-Carcinogenicity in Humans.

The CSF, as calculated by the USEPA, is usually the 95% statistical upper bound on the slope of the dose-response curve in the low-dose linear portion as estimated by the multistage linearized model. The larger the CSF, the more potent the compound. The USEPA and others estimate CSFs based on the assumption that there are no threshold levels for carcinogenic effects and that the response is linear with doses at low levels (including those dose levels encountered in the environment). Thus, there is always some level of cancer risk at every exposure concentration.

Cancer slope factors for some of the chemicals of potential concern have been estimated by USEPA and are available in IRIS (1996) and HEAST (1995). For other compounds, *e.g.*, carcinogenic PAHs, a toxicity equivalency approach was used. The current oral CSF of 7.3 $(mg/kg\text{-}day)^{-1}$ was used to calculate CSFs for the other carcinogenic PAH compounds using "estimated orders of potential potency", in accordance with USEPA guidance (USEPA, 1993b).

CSFs are not available for all of the carcinogenic chemicals of potential concern. Unlike the approach taken with the RfDs, toxicity values were not cross-assigned to structurally similar compounds as it has been shown that different isomers or varying valences of the same compound are not equally carcinogenic. Benzo(g,h,i)perylene and acenaphthylene are currently unclassifiable as to their carcinogenicity due to a lack of data (NCEA; 2/23/95, as cited by EPA Region III, March 10, 1998). In the case of lead, the Weight of Evidence Classification is B2, a Probable Human Carcinogen. However, no Slope Factor has been derived for lead. Lead in soil is present at exposure point concentrations from 75 mg/kg to 634 mg/kg. The maximum detected levels exceed risk based target level of 400 mg/kg, but are not expected to be significant to a degree where carcinogenic effects are noted. Non-cancer endpoints are likely to be more sensitive endpoints that cancer for lead.

In addition, EPA has withdrawn the inhalation RfD for benzo(a)pyrene (EPA, 1997a), however EPA requested we evaluate carcinogenic risk from inhalation of benzo(a)pyrene using an inhalation slope factor of 3.1 per mg/kg-day due to the presence of PAHs at the site. Risks associated with inhalation of other carcinogenic PAHs are not evaluated quantitatively, nor is the risk associated with dermal exposure to carcinogenic PAHs (EPA, March 10, 1998).

4.3 Toxicity Assessment for Lead

There are currently no USEPA-verified RfDs or CSFs for lead. Toxicity values were withdrawn from IRIS due to recent scientific information on the toxicity of lead, which invalidates the use of previously accepted values. To evaluate the toxicity of lead to children (up to age 6), we used the USEPA uptake/biokinetic (IEU/BK) model software (version 0.99, USEPA, 1994). Lead toxicity to persons older than 6 years was not evaluated quantitatively since lead is significantly less toxic to older children and adults.

4.4 Internal (Absorbed) Versus Intake (Administered) Doses

Toxicity values may need to be adjusted depending on the basis of the derived value in IRIS or HEAST. Adjustments may be necessary to match the intake dose estimate with the toxicity value if one is based on an internal (absorbed) dose and the other is based on an intake or administered dose. This is the case for the dermal pathway, in which the potential dose is expressed as an internal or absorbed dose rather than an intake dose. Adjustments may also be necessary for different routes of exposure. If USEPA's published toxicity value is based on an administered dose, it is necessary to convert the toxicity value to an internal dose to be in accordance with the estimated ADD. Based on chemical-specific absorption information in IRIS, the following adjustments may be necessary:

- If the published CSF is based on an administered dose, divide the CSF by the absorption efficiency to derive an adjusted internal (absorbed) dose CSF.

- If the published RfD is based on an administered dose, multiply the RfD by the absorption efficiency to derive an adjusted internal (absorbed) dose RfD.

Ingestion and inhalation exposures were calculated as intake doses. If the toxicity value for a contaminant was expressed as an intake dose, then no adjustment was made. Table 11 presents the factors used to reflect absorption efficiencies for the potential exposure media/routes of exposure.

4.5 Toxicity Profiles for COPCs

For each COPC, toxicity profiles presented in Appendix D describe the potential exposures to the chemical, physical and chemical properties, toxicity of the chemical, toxicokinetics, ecological effects, federal regulations, standards, guidelines and criteria, USEPA-derived toxicity factors, and references.

5.0 RISK CHARACTERIZATION

Risk characterization involves the integration of estimates developed in the exposure assessment and health effects information developed in the toxicity assessment. The exposure and toxicity information is used to estimate non-carcinogenic and carcinogenic risk estimates.

5.1 Estimating Non-carcinogenic Risk Estimates

The potential for non-carcinogenic health effects is evaluated by comparing an exposure dose to a reference dose. This ratio of exposure to toxicity is termed a hazard index. The equation for estimating a hazard index is:

$$Hazard\ Index = \frac{ADD}{RfD}$$

where:

ADD	=	Average daily dose (mg/kg-day)
RfD	=	Reference dose (mg/kg-day)

For the inhalation route, risks are evaluated by comparing an exposure concentration to a Reference concentration. The equation for estimating a hazard index for inhaled COCs is:

$$Hazard\ Index = \frac{ADE}{RfC}$$

where:

ADE	=	Average Daily Exposure (mg/m^3)
RfC	=	Reference Concentration (mg/m^3)

A hazard index is estimated for each COPC and each exposure pathway. To assess the potential for non-carcinogenic effects posed by multiple COPCs and multiple exposure pathways, a pathway risk is calculated by adding the hazard indices for each exposure route; the pathway risks are then summed for all routes of exposure to provide a total hazard index for the exposure scenario.

Individual or total hazard indices greater than 1 indicate that there is the potential for non-cancer health effects. A total hazard index less than 1 indicates that it is unlikely that even sensitive subpopulations would experience adverse health effects.

The significance of an HI exceeding 1 requires additional evaluation. Although hazard quotients are typically summed regardless of target organ effects, hazard quotients for individual compounds should be summed only if their target organs or mechanisms of action are similar.

5.1.1 Target Organ/Mechanism Specific Hazard Indices

In those situations where the Hazard Index exceeds 1, critical effects and target organs associated with each COC were identified by examination of the IRIS database. Generally, the critical effect and target organ is identified in the study from which the RfD was derived. For several COCs, EPA requested we evaluate risks using RfDs not published in IRIS, for which background documentation of the derived reference dose was unavailable in time to meet the agreed upon schedule. These RfDs are used as stated in EPA's March 1998 comment letter without evaluation. Other sources were used to determine the critical effects. These are presented in Table 11B.

The COCs in this assessment have associated with them critical effects that target the kidney, the liver, the blood (hematological), the Central Nervous System (CNS), Cardiovascular systems, cause systemic toxicity, or were not associated with any organ toxicity. For many of the compounds, particularly the PAHs, effects were associated with more than one target organ. Therefore, we have grouped the following: target organ effects together: kidney, liver, hematologic, systemic, no observed effects. Table 17 shows the target organs associated with site COC toxicity.

The kidney, liver, hematologic, systemic, and no target organ Hazard Index includes the following COCs: benzene, ethylbenzene, tetrachloroethene, toluene, 1,2,4-trimethylbenzene, xylenes, anthracene, fluoranthene, acenaphthene, 1-methylnaphthalene, 2-methylnaphthanene, naphthalene, phenanthrene, pyrene, bis(2-ethylhexyl)phthalate, dibenzofuran, arsenic, beryllium, cadmium, chromium, cyanide, nickel, and thallium.

The Central Nervous System (CNS) Specific Hazard Index includes manganese. Several VOCs also are associated with central nervous system toxicity, however this is not the critical effect observed in the derivation of the reference dose.

The Cardiovascular Hazard Index includes Barium.

The Reproductive Organ-Specific Hazard Index includes the following COCs: xylenes and bis(2-ethylhexyl)phthalate.

5.2 Estimating Carcinogenic Risk Estimates

The potential for carcinogenic effects is estimated by calculating the incremental probability of an individual developing cancer during a lifetime. The equation for estimating incremental lifetime cancer risk is as follows:

$$Incremental\ Lifetime\ Cancer\ Risk = ADD_{lifetime} \times CPF$$

where:

$ADD_{lifetime}$ = Lifetime average daily dose (mg/kg-day)
CPF = Cancer slope factor ([mg/kg-day]$^{-1}$)

Where risks approach 1E-2, a one-hit equation is applied:

$$Incremental\ Lifetime Cancer\ Risk = 1 - E\text{^}(ADD_{lifetime} \times CPF)$$

where:

$ADD_{lifetime}$ = Lifetime average daily dose (mg/kg-day)
CPF = Cancer slope factor ([mg/kg-day]$^{-1}$)

For estimating carcinogenic effects for inhalation exposures, the equation becomes:

$$Incremental\ Lifetime Cancer\ Risk = ADE_{lifetime} \times URF$$

where:

$ADE_{lifetime}$ = Lifetime average daily exposure ($\mu g/m^3$)
URF = Unit risk factor $(\mu g/m^3)^{-1}$

For each exposure scenario, incremental lifetime cancer risks are estimated for carcinogenic COPCs. To assess the potential for carcinogenic effects posed by multiple COPCs and multiple exposure pathways, a pathway risk is calculated by adding the cancer risks for each exposure route; the pathway risks are then summed for all routes of exposure to provide a total cancer risk for the exposure scenario.

Total cancer risk estimates greater than 1×10^{-4} are generally considered unacceptable, and risks less than 1×10^{-6} are generally considered *de minimis*. Estimates that are between these levels require regulatory action based on a combination of factors, such as the size of the exposed population or cost-effectiveness considerations (EPA, 1990).

5.3 Summary of Risk Estimates

This section discusses the risk estimates for each scenario. The total non-carcinogenic and carcinogenic risk estimates for all exposure scenarios are summarized in Table 18. Appendix E presents the risk calculation spreadsheets.

5.3.1 Current trespasser

The total Hazard Indices for the CTE exposure case and RME cases are less than 1. The total cancer risks are less than 1 x 10^{-6} for the CTE exposure case (9.6 x 10^{-7}) and greater than 1 x 10^{-6} the RME case (1.5 x 10^{-6}). The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in on-site surface soils. The evaluation conservatively assumes that trespassers contact on-site soils for 40 days per year for 7 years, even though the site is surrounded by a chain link fence. If exposure to on-site surface soils was eliminated (and exposure to off-site areas persisted), the cancer risk estimates for this scenario would be less than 1 x 10^{-6}, as shown in Appendix E.

5.3.2 Future on-site industrial worker

The total Hazard Indices for the CTE exposure case and RME case are less than 1. The total cancer risk for the CTE exposure case (8 x 10^{-6}) and for the RME case (2 x 10^{-5}) is greater than 1 x 10^{-6}. The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in on-site soil.

5.3.4 Future on-site construction worker

The total Hazard Indices for the CTE exposure case are less than 1 and exceed 1 for the RME case (1.2). The total cancer risks are greater than 1 x 10^{-6} for both the CTE exposure case (5 x 10^{-6}) and the RME case (2 x 10^{-5}). The cancer risk estimate for this scenario is driven by potential exposure to carcinogenic PAHs in on-site soils.

5.3.5 Future off-site construction worker

Two distinct locations were evaluated for this receptor, south of Front Street near the railroad tracks, and near the Susquehanna River. The total Hazard Indices for the CTE exposure case and RME cases are less than 1 for both off-site exposure points. Near the Susquehanna River, the total cancer risks are greater than 1 x 10^{-6} for the CTE exposure case (5 x 10^{-6}) and for the RME case (8 x 10^{-6}). Near Front Street and the railroad tracks, the total cancer risks are greater than 1 x 10^{-6} for the CTE exposure case (7 x 10^{-6}) and exceed 1 x 10^{-5} for the RME case (2 x 10^{-5}). The cancer risk estimates for this scenario are driven by potential exposure to carcinogenic PAHs in off-site subsurface soils.

5.3.6 Future hypothetical resident

Two scenarios were evaluated for a hypothetical resident, one living on-site and one off-site. Both scenarios are judged to be highly unlikely to occur, and these scenarios were evaluated per EPA's request (EPA, 1996a, 1997a).

5.3.6.1 Future hypothetical off-site resident

Assuming exposure to contaminants in soil and drinking water, the total Hazard Indices and total cancer risks for this scenario are greater than 1 and 1 x 10^{-4}, respectively, for both the CTE exposure and RME cases. The results are:

	Total Hazard Index		Cancer Risk	
	CTE	RME	CTE	RME
Child (0-6 yrs)	78	920	6×10^{-4}	7×10^{-3}
Adult (7-30 yrs)	31	370	9×10^{-4}	1×10^{-2}

The risk estimates for this scenario are driven by potential exposure to groundwater as drinking water. Risks from inhalation and dermal contact with groundwater were not calculated, although these pathways are likely to significantly contribute to the risks associated with residential exposure to groundwater. Clearly, the risks associated with residential exposure to groundwater already exceed acceptable risk levels by ingestion only, adding the dermal and inhalation exposure pathways would increase the risk. Because the Total Hazard Index represents risks to different target organs, we separated Hazard Indices specific to different target organs below.

Since many COCs have more than one target organ/critical effect, specifically liver, kidney, and hematopoietic system, a HI specific for those compounds is segregated from the HI specific to the cardiovascular system, the reproductive system, and the CNS. Therefore, four Hazard Indices for the hypothetical resident have been estimated and are presented in Table 19. The results are:

	Systemic HI		Cardiovascular HI		Reproductive HI		CNS HI	
	CTE	RME	CTE	RME	CTE	RME	CTE	RME
Child (0-6 yrs)	76	910	0.09	0.12	0.01	0.02	2.1	9.5
Adult (7-30 yrs)	30	360	0.04	0.05	0.004	0.008	0.9	3.7

In summary, organs affected by chemicals causing Systemic effects, benzene, in particular, present the significant risk to a hypothetical off-site resident. The only other target organ to exceed 1 is the RME case for manganese (CNS), and the exceedence is not significantly relative to the systemic HI.

It is our opinion that a future residence in the area (near the Susquehanna River, wastewater treatment plant, and water intake facility) is highly unlikely based on zoning regulations and surrounding land use. It is also our opinion that risks for this scenario are likely overstated, considering the nature of the exposures evaluated, even with the exclusion of inhalation and dermal exposure pathways to groundwater. Exposure to contaminants in groundwater is assumed to occur regularly via use of a private well. These exposures are evaluated assuming an exposure duration of 30 years.

5.3.6.2 Future Hypothetical On-site Resident

Assuming exposure to contaminants in soil and drinking water, the total Hazard Indices and total cancer risks for this scenario are greater than 1 and 1 x 10^{-4}, respectively, for both the CTE exposure and RME cases. The results are:

	Total Hazard Index		Cancer Risk	
	CTE	RME	CTE	RME
Child (0-6 yrs)	230	580	4 x 10^{-3}	1 x 10^{-2}
Adult (7-30 yrs)	93	230	7 x 10^{-3}	2 x 10^{-2}

The cancer risk estimates for this scenario are driven by potential exposure to groundwater as drinking water. Risks from inhalation and dermal contact with groundwater were not calculated, although these pathways are likely to significantly contribute to the risks associated with residential exposure to groundwater. Clearly, the risks associated with residential exposure to groundwater already exceed acceptable risk levels by ingestion only, adding the dermal and inhalation exposure pathways would increase the risk.

Since many COCs have more than one target organ/critical effect, specifically liver, kidney, and hematopoietic system, a HI specific for those compounds is segregated from the HI specific to the cardiovascular system, the reproductive system, and the CNS. Therefore, four Hazard Indices for the hypothetical resident have been estimated and are presented in Table 20. The results for the on-site resident are:

	Systemic HI		Cardiovascular HI		Reproductive HI		CNS HI	
	CTE	RME	CTE	RME	CTE	RME	CTE	RME
Child (0-6 yrs)	230	570	0.2	0.2	0.3	0.4	1.5	4.7
Adult (7-30 yrs)	92	230	0.07	0.07	0.1	0.2	0.5	1.8

In summary, organs affected by chemicals causing Systemic effects, benzene, in particular, present the significant risk to a hypothetical off-site resident. The only other target organ to exceed 1 is the CNS due to for manganese, and the exceedence is not significant relative to the systemic HI.

5.3.6.4 Risk To Hypothetical Child Resident from Lead Exposure

Risks due to lead exposure for the child resident were evaluated using EPA's IEU/BK computer model. On-site hypothetical child residents were exposed to lead in surface soil and drinking water. The 1994 Lead99d program was run with default parameters set for dust, food, and maternal exposure. The off-site resident was not evaluated because there is no surface soil exposure due to the presence of fill, and the average off-site groundwater concentration (8.2 µg/l) did not exceed the EPA action level (15 µg/l).

For hypothetical on-site residents, the inputs were a groundwater (drinking water) lead concentration of 4.3 µg/l, and a soil concentration of 141 mg/kg. Results of the model indicate predicted geometric mean blood lead levels of 4.6 µg/dl, below the threshold of 10 µg/dl. The model predicted 4.7 % above the 10 µg/dl cutoff, below the 5% limit of the acceptable range. Therefore, lead does not appear to pose a significant risk to a hypothetical child resident. Appendix F contains the output of the model.

5.4 Uncertainty

There are numerous sources of uncertainty associated with the data and modeling used in the baseline human health risk assessment. Due to the uncertainty that may be associated with each risk estimate and each component of the risk process, the risk estimates were conservatively calculated. Although the degree to which uncertainty may influence the risk estimates cannot be quantified, it is our opinion that the risk estimates presented in this assessment do not underestimate potential risk for the exposure pathways and scenarios evaluated, for the following reasons.

For both the CTE and RME risk estimates, upper bound exposure assumptions were used. The RME exposure point concentration represents an upper bound concentration. The toxicity factors, both non-cancer and cancer, are based on upper bound exposures, and the non-cancer RfDs apply uncertainty factors in their derivation.

5.4.1 Hazard identification

As with any risk assessment, there are uncertainties associated with the analytical data. For example, there is uncertainty introduced into the analysis with regard to estimated values below detection limits and the treatment of non-detects. A conservative approach is taken in assigning non-detects a value of one-half the sample detection limit. In assigning a value of one half of the detection limit, the analysis captures those compounds that may have been reported as non-detect due to either matrix interferences or the presence of the compound below the practical quantitation limit.

Much of the Atlantic Environmental Services, Inc. analytical soil and groundwater data for VOCs and SVOCs were measured using microscale solvent extraction (MSE) in lieu of USEPA Method 8240 and 8270. Several soil samples were split by Atlantic Environmental Services, Inc. and analyzed using both MSE and 8240/8270 for organic compounds. Two of the three confirmatory samples agreed reasonably well with the MSE data (samples SS4 and SB1). A third confirmatory sample yielded 107 ppm total PAHs versus 45 and 148 ppm total PAHs for the MSE sample and its duplicate. Duplicate confirmatory samples were not collected. This report uses the validated analytical data presented in *Microscale Solvent Extraction Method Data Validation Reports and Analytical Data* (Atlantic Environmental Services, 1997).

5.4.2 Exposure assessment

Uncertainty associated with the exposure assessment is related to characterizing potential exposures. Actual measurements of exposure to contaminants at hazardous waste sites have not been made. We rely upon estimates of exposure based on assumptions about the population exposed including characteristics of the receptor group, the frequency and intensity of exposure, and the concentrations to which they may be exposed. Exposures are uncertain and variable, as they are estimates of human activities that we cannot measure and which may vary. The exposure assumptions used in this analysis are health protective in that they are far more likely to overestimate than to underestimate potential exposures.

Modeling of exposure point concentrations introduces uncertainty into the analysis as models are based on assumptions about a chemical's behavior in the environment, site conditions that influence the fate and transport of contaminants, and the representativeness of the analytical data used in the models. The uncertainty is addressed in part by using upper bound contaminant concentrations in the models.

There is uncertainty associated with the use of absorption factors used in estimating ADDs. There are limited data on the amount of a chemical that is absorbed into the body following environmental exposures to contaminated media. Often, 100% absorption is assumed as a default. Because risk estimates are significantly influenced by absorption factors, the result is overstated potential risks. For example, the cancer risk estimate associated with ingestion of benzo(a)pyrene in soil (based on 100% absorption) is 1×10^{-5} for the industrial worker scenario (RME case). If a bioavailability factor of 0.3 for soil were adopted, evaluating the pathway based on 30% absorption would yield a cancer risk estimate of 3×10^{-6}.

For dermal contact with soil, a factor of 10% is assumed for SVOCs based on USEPA Region III guidance (1995). There is evidence that indicates when PAHs are present as a mixture, the contaminants are not easily desorbed from soil and therefore not bioavailable, *i.e.*, cannot be absorbed through the skin (Alexander, 1995).

5.4.3 Toxicity assessment

The most difficult uncertainty to reduce is that inherent in the toxicity assessment. All human health risk assessments rely on toxicity factors developed by USEPA using numerous assumptions to estimate reference doses and cancer slope factors for chemicals. A significant uncertainty in this area is the extrapolation of effects data in animal studies to humans.

Ingestion and inhalation RfDs are not available for all COPCs. To avoid excluding COPCs from the analysis, published toxicity values were cross-assigned to compounds for which none were available. Similarly, chronic RfDs were used to evaluate subchronic exposures when subchronic

RfDs were not available. The surrogate values allow for a more complete evaluation of additive effects, but may result in overestimates of risk.

Due to uncertainty regarding the mechanism of action, the toxicity of acenaphthylene and benzo(g,h,i) perylene is not quantified. This may result in an underestimation of the non-cancer and carcinogenic toxicity associated with exposure to PAHs in soil.

5.4.4 Risk characterization

The risk characterization is also subject to uncertainty. The risk estimates provided in this report are point estimates, whereas the actual risks are a range of values based on the varying levels of exposure and sensitivity in the population. Given the conservative nature of the analysis performed, it is our opinion that the risk estimates in the assessment do not underestimate potential risk for the exposure pathways and scenarios evaluated.

Uncertainty is introduced because not all exposures were quantified. For several COCs, toxicity factors were not available for the pathway of concern, for example, risks associated with inhalation of PAHs in fugitive dust were not calculated because EPA has withdrawn the Inhalation Reference Dose for Benzo(a)pyrene. However, EPA Region III requested we evaluate inhalation risk associated with Benzo(a)pyrene in soil by vapor inhalation (EPA, 3/11/98). Other exposures that were not quantified include risks associated with dermal exposure to PAHs, and ingestion exposure to benzo(g,h,i) perylene and acenaphthylene, two PAHs for which toxic potency has not been determined. This contributes to uncertainty in the risk characterization, and perhaps underestimate risks associated with exposure to these compounds. Additionally, risks associated with hypothetical residential exposure to groundwater contaminants by inhalation and dermal pathways were not calculated.

Additional uncertainty is introduced into the assessment due to the lack of surface soil data for the off-site construction and hypothetical resident scenarios. We assumed that surface soils are not contaminated because of the presence of fill and gravel in off-site soils. If these media were contaminated, it could increase an off-site receptor's risk.

6.0 CONCLUSIONS

The conclusions of this baseline risk assessment are intended to provide risk managers with insight on how to conceptualize the potential risks associated with the Former UGI Columbia MGP site. The results are discussed as they relate to media, locations, chemicals, and pathways with the intention of promoting an appropriate remedial strategy.

On-site soil
Contamination of on-site soil has been a focus of the remedial investigation. Under existing conditions, wherein the site is vacant and inaccessible, there is the potential for surface soil to pose a moderately low cancer risk to trespassers (between 1×10^{-6} and 2×10^{-5}). The cancer risk is due to incidental ingestion and dermal contact with carcinogenic PAHs in on-site soil; the risks associated with exposure to off-site surface soils is unknown, but assumed to be low due to the presence of fill material or gravel.

Under future conditions, assuming the site is being redeveloped, there is the potential for soil to pose a moderately low cancer risk to construction workers (between 5×10^{-6} and 5×10^{-5}). The cancer risk is associated with incidental ingestion of carcinogenic PAHs in surface and subsurface soil while excavating at the site.

Under future conditions, assuming the site has been developed for industrial purposes, there is the potential for soil to pose a moderately low cancer risk to industrial workers. The cancer risk estimate for the CTE exposure case (8×10^{-6}) is between 1×10^{-6} and 1×10^{-5}. The cancer risk estimate for the RME case (2×10^{-5}) is between 1×10^{-5} and 1×10^{-4}. The cancer risks are associated with incidental ingestion of carcinogenic PAHs in soil.

Though unlikely, the site could be developed in the future for residential land use. There is potential for soil to pose a moderate cancer risk (between 1×10^{-5} and 1×10^{-4}) to future residents at this area. The cancer risk is associated with incidental ingestion of carcinogenic PAHs in soil. There is potential for surface soils to pose a high non-cancer risk to systemic target organs, due primarily to exposure to benzene vapors from soil.

Off-site soils between Front Street and the railroad tracks
Under future conditions, assuming the area is being redeveloped, there is the potential for soil to pose a moderately low cancer risk to construction workers (between 5×10^{-6} and 5×10^{-5}). The cancer risk is associated with incidental ingestion of carcinogenic PAHs in subsurface soil while excavating at the site.

Off-site soils near the Susquehanna River and the wastewater treatment plant
Under future conditions, assuming the area is being redeveloped, there is the potential for soil to pose a relatively low cancer risk to construction workers (between 1×10^{-6} and 1×10^{-5}). The

cancer risk is associated with incidental ingestion of carcinogenic PAHs in subsurface soil while excavating at the site.

Though unlikely, the area between the site and river could be developed in the future for residential land use. There is potential for soil to pose a moderate cancer risk (between 1×10^{-5} and 1×10^{-4}) to future residents at this area. There is potential for soils to pose a moderate non-cancer risk to systemic target organs.

Sediment at near-shore reach of the Susquehanna River
Excavation of these sediments during a removal action occurred in January, 1998 which eliminated any human health risk associated with exposure to site- related compounds in these sediments.

Sediment in Shawnee Creek
Under current conditions, the potential risks associated with exposure (incidental ingestion and dermal contact) to Shawnee Creek sediment are *de minimis, i.e.,* less than 1×10^{-6}.

Surface Water in the Susquehanna River

Due to the planned remediation of the contaminated river sediment, it was assumed that the source of COPCs in surface water will be removed, so risks associated with exposure to surface water were not calculated. In addition, no COPCs were detected in river surface water.

Surface Water in Shawnee Creek
There were no COPCs detected in Shawnee Creek surface water.

Groundwater
Under current conditions, residents located approximately one quarter mile to the northwest of the site use groundwater from private wells. Groundwater samples collected from these wells were analyzed for site-related contaminants; there were no COPCs detected. Transport of COPCs toward these residences appears unlikely based on the location of the wells, the low mobility of the MGP-related chemicals, and the nature and direction of the fractures in bedrock (Atlantic Environmental Services, Inc., 1996).

Though unlikely, the area between the site and river could be developed in the future for residential land use. If a well were placed in this area, the use of groundwater as drinking water could pose non-carcinogenic risks greater than 1 and potential cancer risks greater than 1×10^{-4}. Currently, there are no residents relying on groundwater for drinking water between the site and the river.

Though unlikely, the site could be developed in the future for residential land use. If a well were placed in this area, the use of groundwater as drinking water could pose non-carcinogenic risks

greater than 1 and potential cancer risks greater than 1×10^{-4}. Currently, the site is not zoned for residential use.

7.0 REFERENCES

Alexander, Martin. 1995. How toxic are toxic chemicals in soil? *Environ. Sci. Technol.* 29:2713-2717.

Atlantic Environmental Services, Inc. 1995. *River Sediment Investigation - Former UGI Manufactured Gas Plant.* Columbia, Pennsylvania. Colchester, CT.

Atlantic Environmental Services, Inc. 1996. *Draft Remedial Investigation - Former UGI Manufactured Gas Plant.* Columbia, Pennsylvania. Colchester, CT.

Atlantic Environmental Services, a division of GEI Consulting, Inc. 1997. *Microscale Solvent Extraction Method Data Validation Reports and Analytical Data.* Columbia, Pennsylvania. Colchester, CT.

Gilbert, R.O. 1987. *Statistical Methods for Environmental Pollution Monitoring.* Van Nostrand Rheinhold Co., New York, pp. 152-185.

Gas Research Institute. 1988. *Management of manufactured gas plant sites.* Vol. III, Risk assessment. Chicago, Illinois: Gas Research Institute.

Howard, P.H. 1989. *Handbook of fate and exposure data for organic chemicals.* Chelsea, Michigan: Lewis Publishers.

Johnson, P.C. 1989. *Estimates for hydrocarbon vapor emissions resulting from service station remediations and buried gasoline-contaminated soils.* Presented at the Fourth National Conference on Petroleum Contaminated Soils, University of Massachusetts at Amherst, September 25-28, 1989.

Kissel, J., Richter, K., Duff, R,. and Fenske, R. (1996) Field measurements of dermal soil loading attributable to various activities: Implications for exposure assessment. *Risk Analysis* **16**(1): 116-125.

Lyman, W.J. et al. 1982. *Handbook of chemical property estimation methods.* New York, New York. McGraw-Hill Book Company.

Menzie-Cura & Associates, Inc. *Preliminary Human Health Risk Assessment for the UGI Manufactured Gas Plant.* May 1995. Chelmsford, Massachusetts.

META Environmental, Inc. December 1, 1997 *Review of Remedial Investigation Data for the Former UGI Manufactured Gas Plant,* Columbia, Pennsylvania.

NUS Corporation. 1991. *Expanded Site Inspection of UGI Columbia Gas Plant*. Columbia, Pennsylvania. TDD No. F3-9011-56;

Shacklette, H.T. and J.C. Boerngen. 1984. *Element concentrations in soils and other surficial materials of the conterminous United States*. U.S. Geological Survey Professional Paper 1270. Washington, DC.

TRC Environmental Consultants, Inc. 1986. *Final Report of Investigations - Columbia Gas Plant Site*. Columbia, Pennsylvania. Volumes I & II;

U.S. Environmental Protection Agency (USEPA). 1989a. *Risk Assessment Guidance for Superfund*. Volume I - Human Health Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response. EPA 540/1-89/002. December 1989. Washington, D.C.: Government Printing Office.

U.S. Environmental Protection Agency (USEPA). 1989b. *Exposure Factors Handbook*. Office and Health and Environmental Assessment. EPA 600/8-89/043. July 1989. Washington, D.C.: Government Printing Office.

U.S. Environmental Protection Agency (USEPA). 1990. *Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions*. OSWER Directive 9355-0-30. . Office of Emergency and Remedial Response.

U.S. Environmental Protection Agency (USEPA). 1991. *Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors."* Office of Solid Waste and Remedial Response. OSWER Directive 9285.6-03. March 25, 1991. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1992a. *Guidance for Data Useability in Risk Assessment (Part A)*. Office of Emergency and Remedial Response. Publication Number 9285.7-09A.

U.S. Environmental Protection Agency (USEPA). 1992b. *Supplemental Guidance to RAGS: Calculating the Concentration Term*. Office of Solid Waste and Emergency Response. Washington, D.C. Publication 9285.7-081.

U.S. Environmental Protection Agency (USEPA). 1992c. *National Air Quality and Emissions Trends Report, 1991*. Office of Air Quality. Report 450-R-92-001.

U.S. Environmental Protection Agency (USEPA). 1992d. *Dermal Exposure Assessment: Principles and Applications (Interim Report)*. Office of Research and Development, EPA/600/8-91/011B.

U.S. Environmental Protection Agency (USEPA). 1993a. *Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening.* EPA/903/R-93-001.

U.S. Environmental Protection Agency (USEPA). 1993b. *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons.* EPA/600/R-93/089.

U.S. Environmental Protection Agency (USEPA). 1994. *PC Software Application of the Uptake/Biokinetic Model.* Version 0.99. Office of Research and Development. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1995a. *Assessing Dermal Exposure from Soil. Region III Technical Guidance Manual.* Philadelphia, Pennsylvania. EPA/903-K-95-003.

U.S. Environmental Protection Agency (USEPA). 1995b. *Health Effects Assessment Summary Tables (HEAST).* Annual FY-1995. Office of Research and Development/ Office of Emergency and Remedial Response. OERR 9200.6-303. Washington D.C.: Government Printing Office.

U.S. Environmental Protection Agency (USEPA). March 22, 1996a. *UGI Columbia Gas MGP Site. EPA Comments on Preliminary Human Health Risk Assessment.* USEPA Region III, Philadelphia, Pennsylvania.

U.S. Environmental Protection Agency (USEPA). 1996b. *Soil Screening Guidance*: Technical Background Document, 2nd Edition. EPA/540/R-95/128. Office of Solid Waste and Emergency Response, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1996c. *Integrated Risk Information System* (IRIS). On-line database search.

U.S. Environmental Protection Agency (USEPA). August, 22, 1997a. *UGI Columbia Gas MGP Site. EPA Comments on Draft Baseline Human Health Risk Assessment Report.* USEPA Region III, Philadelphia, Pennsylvania.

U.S. Environmental Protection Agency (USEPA). 1997b. *Risk-Based Concentration Table.* January-June, 1997b. USEPA Region III, Philadelphia, Pennsylvania.

U.S. Environmental Protection Agency (USEPA). January 6, 1998. *UGI Columbia Gas MGP Site. EPA Comments on Draft Baseline Human Health Risk Assessment Report.* USEPA Region III, Philadelphia, Pennsylvania.

U.S. Environmental Protection Agency (USEPA). March 11, 1998. Letter regarding *UGI Columbia Gas MGP Site. Baseline Human Health Risk Assessment Report.* USEPA Region III, Philadelphia, Pennsylvania.

TABLES

TABLES

ORIGINAL (Red)

TABLE 1
COMPOUNDS DETECTED BY MEDIA
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	MEDIA				
	Surface Soil	Subsurface Soil	Groundwater	Sediments	Surface Water
Volatile Organic Compounds					
Acetone	✓				
Benzene	✓	✓	✓		
Ethylbenzene	✓	✓	✓		
Tetrachloroethene			✓		
Styrene		✓	✓		
Trichloroethene			✓		
Toluene	✓	✓	✓		
1,2,4-Trimethylbenzene	✓	✓	✓		
Xylenes (total)	✓	✓	✓		
Semi-Volatile Organic					
Polycyclic Aromatic Hydrocarbons					
Acenaphthene	✓	✓	✓		
Acenaphthylene	✓	✓	✓		
Anthracene	✓	✓	✓		
Benzo(a)anthracene	✓	✓	✓	✓	
Benzo(b)fluoranthene	✓	✓	✓	✓	
Benzo(k)fluoranthene	✓	✓		✓	
Benzo(g,h,i)perylene	✓	✓		✓	
Benzo(a)pyrene	✓	✓	✓	✓	
Chrysene	✓	✓	✓	✓	
Dibenzo(a,h)anthracene	✓	✓			
Fluoranthene	✓	✓	✓	✓	
Fluorene	✓	✓	✓		
Indeno(1,2,3-cd)pyrene	✓	✓		✓	
1-Methylnaphthalene	✓	✓	✓		
2-Methylnaphthalene	✓	✓	✓		
Naphthalene	✓	✓	✓		
Phenanthrene	✓	✓	✓	✓	
Pyrene	✓	✓	✓	✓	
Other SVOCs					
Bis(2-ethylhexyl)phthalate	✓		✓	✓	
Dibenzofuran	✓	✓	✓		
Di-n-octylphthalate	✓				
2-Methylphenol			✓		
4-Methylphenol			✓		
Phenol (total)			✓		

TABLE 1
COMPOUNDS DETECTED BY MEDIA
UGI Columbia Former MGP Site
Columbia, Pennsylvania

ORIGINAL (Red)

COMPOUNDS	MEDIA				
	Surface Soil	Subsurface Soil	Groundwater	Sediments	Surface Water
Inorganic Compounds					
Aluminum	✓	✓	✓	✓	
Arsenic	✓	✓		✓	
Barium	✓	✓	✓	✓	✓
Beryllium	✓	✓	✓	✓	
Cadmium	✓			✓	
Calcium	✓	✓	✓	✓	✓
Chromium	✓	✓	✓	✓	
Cobalt	✓	✓	✓	✓	
Copper	✓	✓	✓	✓	
Cyanide	✓	✓	✓		
Iron	✓	✓	✓	✓	
Lead	✓	✓	✓	✓	
Magnesium	✓	✓	✓	✓	✓
Manganese	✓	✓	✓	✓	
Mercury	✓	✓		✓	
Nickel	✓	✓	✓	✓	
Potassium	✓	✓	✓		
Selenium	✓	✓	✓	✓	✓
Sodium	✓	✓	✓		✓
Thallium	✓	✓	✓		
Vanadium	✓	✓	✓	✓	
Zinc	✓	✓	✓	✓	✓

TABLE 2
SCREENING ORGANIC COMPOUNDS DETECTED IN ONSITE AND OFFSITE SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Soil mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Soil
Volatile Organic Compounds			
Acetone	0.056	780[N]	No[1]
Benzene	0.79	22[C]	No[1]
Ethylbenzene	0.25	780[N]	No[1]
Toluene	1.30	1,600[N]	No[1]
1,2,4-Trimethylbenzene	1.4	390[N]	No[1]
Xylenes (total)	3.3	16,000[N]	No[1]
Semivolatile Organic Compounds			
Polycyclic Aromatic Hydrocarbons			
Acenaphthene	0.26	470[N]	No[1]
Acenaphthylene	3.8	NA	Yes[4]
Anthracene	1.3	2300[N]	No[1]
Benzo(ghi)perylene	9.0	NA	Yes[4]
Fluoranthene	6.6	310[N]	No[1]
Fluorene	0.5	310[N]	No[1]
1-Methylnaphthalene	0.8	310[2]	No[1]
2-Methylnaphthalene	1.0	310[N]	No[1]
Naphthalene	1.7	310[N]	No[1]
Phenanthrene	3.4	230[3]	No[1]
Pyrene	12	230[N]	No[1]
Benzo(a)anthracene	4.6	0.88[C]	Yes
Benzo(a)pyrene	6.2	0.088[C]	Yes
Benzo(b)fluoranthene	6.7	0.88[C]	Yes
Benzo(k)fluoranthene	6.1	8.8[C]	No[1]
Chrysene	5.5	88[C]	No[1]

TABLE 2
SCREENING ORGANIC COMPOUNDS DETECTED IN ONSITE AND OFFSITE SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Soil mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Soil
Dibenzo(ah)anthracene	0.11	0.088[C]	Yes
Indeno(123-cd)pyrene	5.9	0.88[C]	Yes
Other SVOCs			
Bis(2-ethylhexyl)phthalate	0.25	46[C]	No[1]
Di-n-octyl phthalate	0.76	160[N]	No[1]
Dibenzofuran	0.38	31[N]	No[1]

NA = Not Available

[1] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[2] The Risk-Based Concentration for naphthalene is used as the RBC for these compounds

[3] The Risk-Based Concentration for pyrene is used as the RBC for this compound.

[4] Evaluated quantitatively due to a lack of toxicity data.

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

Surface soil is defined as 0 to 6 inches deep.

TABLE 3
SCREENING INORGANIC COMPOUNDS DETECTED IN ONSITE AND OFFSITE SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected In Soil mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Soil
Inorganic Compounds			
Aluminum	11,200	7,800[N]	**Yes**
Arsenic	14	0.43[C]	**Yes**
Barium	236	550[N]	**No[2]**
Beryllium	0.57	0.15[C]	**Yes**
Cadmium	5.8	3.9[N]	**Yes**
Calcium	257,000	NA	**No[1]**
Chromium	32.4	39[N 5]	**No[2]**
Cobalt	21.6	470[N]	**No[2]**
Copper	275	310[N]	**No[2]**
Cyanide	48.2	160[N]	**No[2]**
Iron	41,800	2,300[N]	**Yes**
Lead	634	400[3]	**Yes**
Magnesium	43,500	NA	**No[1]**
Manganese	1,700	180[N]	**Yes**
Mercury	0.44	2.3[N]	**No[2]**
Nickel	23.2	160[N]	**No[2]**
Potassium	4,370	NA	**No[1]**
Selenium	0.74	39[N]	**No[2]**
Sodium	143	NA	**No[1]**
Thallium	0.26	0.63[N 4]	**No[2]**
Vanadium	24.2	55[N]	**No[2]**
Zinc	783	2,300[N]	**No[2]**

NA = Not available

[1] Compound removed from list of COPCs because it is an essential nutrient (EPA, 1989)

[2] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[3] EPA "action level" for lead in residential soil

[4] The Risk-Based Concentration for thallium corresponds to thallium chloride because there is no RBC for elemental thallium.

[5] The Risk-Based Concentration corresponds to the RBC for chromium VI

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

* The RBC given for cyanide is for free cyanide

TABLE 4
SCREENING ORGANIC COMPOUNDS DETECTED IN SUBSURFACE SOIL ONSITE AND OFFSITE
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Soil mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Soil
Volatile Organic Compounds			
Benzene	3.9	22[C]	No[1]
Ethylbenzene	29	780[N]	No[1]
Styrene	4.4	1,600[N]	No[1]
Toluene	22	1,600[N]	No[1]
1,2,4-Trimethylbenzene	46	390[N]	No[1]
Xylenes (total)	30	16,000[N]	No[1]
Semivolatile Organic Compounds			
Polycyclic Aromatic Hydrocarbons			
Acenaphthene	140	470[N]	No[1]
Acenaphthylene	160	NA	Yes[4]
Anthracene	99	2300[N]	No[1]
Benzo(a)anthracene	47	0.88[C]	Yes
Benzo(a)pyrene	35	0.088[C]	Yes
Benzo(b)fluoranthene	39	0.88[C]	Yes
Benzo(g,h,i)perylene	11	NA	Yes[4]
Benzo(k)fluoranthene	18	8.8	Yes
Chrysene	52	88[C]	No[1]
Dibenz(a,h)anthracene	8.4	.088[C]	Yes
Fluoranthene	92	310[N]	No[1]
Fluorene	59	310[N]	No[1]
Indeno(1,2,3-cd)pyrene	15	0.88[C]	Yes
1-Methylnaphthalene	240	310[2]	No[1]
2-Methylnaphthalene	130	310[N]	No[1]
Naphthalene	190	310[N]	No[1]
Phenanthrene	170	230[3]	No[1]
Pyrene	150	230[N]	No[1]
Other SVOCs			
Dibenzofuran	18	31[N]	No[1]

NA = Not Available

[1] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[2] The Risk-Based Concentration for naphthalene is used as the RBC for these compounds

[3] The Risk-Based Concentration for pyrene is used as the RBC for this compound.

[4] Evaluated quantitatively due to a lack of toxicity data.

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

Subsurface soil is defined as 6 inches to 15 feet deep.

TABLE 5
SCREENING INORGANIC COMPOUNDS DETECTED IN SUBSURFACE SOIL ONSITE AND OFFSITE
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Soil mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Soil
Inorganic Compounds			
Aluminum	21,400	7,800[N]	Yes
Arsenic	19	0.43[C]	Yes
Barium	191	550[N]	No[2]
Beryllium	3.9	0.15[C]	Yes
Calcium	132,000	NA	No[1]
Chromium	21	39[N]	No[2]
Cobalt	23	470[N]	No[2]
Copper	68	310[N]	No[2]
Cyanide	45	160[N]	No[2]
Iron	37,400	2,300[N]	Yes
Lead	140	400[3]	No[2]
Magnesium	34,900	NA	No[1]
Manganese	2,490	180[N]	Yes
Mercury	0.21	2.3[N]	No[2]
Nickel	29	160[N]	No[2]
Potassium	5,750	NA	No[1]
Selenium	2.2	39[N]	No[2]
Sodium	115	NA	No[1]
Thallium	3.0	0.63[N 4]	Yes
Vanadium	35	55[N]	No[2]
Zinc	194	2,300[N]	No[2]

NA = Not available

[1] Compound removed from list of COPCs because it is an essential nutrient and/or it is not likely to be toxic at the detected

[2] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[3] EPA "action level" for lead in residential soil

[4] The Risk-Based Concentration for thallium corresponds to thallium chloride because there is no RBC for elemental thalliu

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

* The RBC given for cyanide is for free cyanide

TABLE 6
SCREENING ORGANIC COMPOUNDS DETECTED IN SHAWNEE CREEK SEDIMENT
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Sediment mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Sediment
Semivolatile Organic Compounds			
Polycyclic Aromatic Hydrocarbons			
Benzo(a)anthracene	1.0	0.88[C]	Yes
Benzo(a)pyrene	0.74	0.088[C]	Yes
Benzo(b)fluoranthene	1.3	0.88[C]	Yes
Benzo(g,h,i)perylene	0.61	NA	Yes [3]
Benzo(k)fluoranthene	0.71	8.8[C]	No [1]
Chrysene	1.0	88[C]	No [1]
Fluoranthene	2.9	310[N]	No [1]
Indeno(1,2,3-cd)pyrene	0.78	0.88[C]	No [1]
Phenanthrene	0.94	230[2]	No [1]
Pyrene	2.7	230[N]	No [1]
Other SVOCs			
Bis(2-ethylhexyl)phthalate	1.4	46[C]	No[1]

NA = Not Available

[1] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[2] The Risk-Based Concentration for pyrene is used as the RBC for this compound.

[3] Evaluated quantitatively due to a lack of toxicity data.

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

TABLE 7
SCREENING INORGANIC COMPOUNDS DETECTED IN SHAWNEE CREEK SEDIMENT
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Sediment mg/kg	Risk-Based Concentrations for Residential Soil mg/kg	Retained as Chemical of Potential Concern for Sediment
Inorganic Compounds			
Aluminum	9,200	7,800[N]	Yes
Arsenic	16.1	0.43[C]	Yes
Barium	133	550[N]	No[2]
Cadmium	1.5	3.9[N]	No[2]
Calcium	16,600	NA	No[1]
Chromium	21.3	39[N]	No[2]
Copper	391	310[N]	Yes
Iron	27,200	2,300[N]	Yes
Lead	307	400[3]	No[2]
Magnesium	4,540	NA	No[1]
Manganese	1,300	180[N]	Yes
Mercury	0.19	2.3[N]	No[2]
Nickel	27.1	160[N]	No[2]
Selenium	2.6	39[N]	No[2]
Vanadium	19.6	55[N]	No[2]
Zinc	1,510	2,300[N]	No[2]

NA = Not available

[1] Compound removed from list of COPCs because it is an essential nutrient (EPA, 1989)

[2] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration (Residential)

[3] EPA "action level" for lead in residential soil

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

* The RBC given for cyanide is for free cyanide

TABLE 8
SCREENING INORGANIC COMPOUNDS DETECTED IN SHAWNEE CREEK SURFACE WATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Water mg/L	Risk-Based Concentrations for Tap Water mg/L	Retained as Chemical of Potential Concern for Water
Inorganic Compounds			
Barium	0.028	0.26[N]	No[2]
Calcium	56	NA	No[1]
Magnesium	16	NA	No[1]
Selenium	0.009	.018[N]	No[2]
Sodium	28	NA	No[1]
Zinc	0.034	1.1[N]	No[2]

NA = Not available

[1] Compound removed from list of COPCs because it is an essential nutrient (EPA, 1989)

[2] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

TABLE 9
SCREENING ORGANIC COMPOUNDS DETECTED IN GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Water mg/L	Risk-Based Concentrations for Tap Water mg/L	Retained as Chemical of Potential Concern for Water
Volatile Organic Compounds			
Benzene	39	0.00036[C]	Yes
Ethylbenzene	4.7	0.13[N]	Yes
Styrene	0.076	0.16[N]	No[1]
Tetrachloroethene	0.005	0.0011[C]	Yes
Toluene	9.5	0.075[N]	Yes
Trichloroethene	0.003	0.0016[C]	Yes
1,2,4-Trimethylbenzene	0.47	0.0012[N]	Yes
Xylenes (total)	3.7	1.2[N]	Yes
Semi-Volatile Organic Compounds			
Polycyclic Aromatic Hydrocarbons			
Acenaphthene	0.75	0.22[N]	Yes
Acenaphthylene	0.49	NA	Yes[4]
Anthracene	0.32	1.1[N]	No[1]
Benzo(a)anthracene	0.19	0.000092[C]	Yes
Benzo(a)pyrene	0.15	0.0000092[C]	Yes
Benzo(b)fluoranthene	0.13	0.000092[C]	Yes
Chrysene	0.14	0.0092[C]	Yes
Fluoranthene	0.28	0.15[N]	Yes
Fluorene	0.12	0.15[N]	No[1]
1-Methylnaphthalene	0.75	0.15[2]	Yes
2-Methylnaphthalene	2.6	0.15[2]	Yes
Naphthalene	8.2	0.15[N]	Yes
Phenanthrene	1.2	0.11[3]	Yes
Pyrene	0.72	0.11[N]	Yes
Other SVOCs			
Bis(2-ethylhexyl)phthalate	0.069	0.0048[C]	Yes
Dibenzofuran	0.081	0.015[N]	Yes
2-Methylphenol	0.009	0.18[N]	No[1]
4-Methylphenol	0.006	.018[N]	No[1]
Phenol	0.036	2.2[N]	No[1]

NA = Not available
[1] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration
[2] The Risk-Based Concentration for naphthalene is used as the RBC for these compounds
[3] The Risk-Based Concentration for pyrene is used as the RBC for this compound
[4] Evaluated quantitatively due to a lack of toxicity data.
[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)
[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-6})

TABLE 10
SCREENING INORGANIC COMPOUNDS DETECTED IN GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	Maximum Concentration Detected in Water mg/L	Risk-Based Concentrations for Tap Water mg/L	Retained as Chemical of Potential Concern for Water
Inorganic Compounds			
Aluminum	9.6	3.7[N]	Yes
Barium	0.541	.26[N]	Yes
Calcium	204	NA	No[1]
Chromium	0.0082	0.018[*N]	No[2]
Cobalt	0.0078	.22[N]	No[2]
Copper	0.031	.15[N]	No[2]
Cyanide	0.22	.073[N]	Yes
Iron	42	1.1[N]	Yes
Lead	0.019	.015[3]	Yes
Magnesium	34	NA	No[1]
Manganese	3.0	.084[N]	Yes
Nickel	0.023	.073[N]	No[2]
Potassium	13	NA	No[1]
Selenium	0.0062	.018[N]	No[2]
Sodium	39	NA	No[1]
Vanadium	0.019	.026[N]	No[2]
Zinc	0.143	1.1[N]	No[2]

NA = Not available

[1] Compound removed from list of COPCs because it is an essential nutrient (EPA, 1989).

[2] Compound removed from list of COPCs based on comparison to EPA Region III Risk-Based Concentration

[3] EPA "action level" for lead in groundwater

[N] Indicates that the RBC is based on noncarcinogenic effects (using a target hazard quotient of 0.1)

[C] Indicates that the RBC is based on carcinogenic effects (using a target cancer risk of 10^{-5})

* The RBC given for cyanide is for free cyanide

TABLE 11A
SUBCHRONIC DOSE-RESPONSE INFORMATION FOR COCs
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site																		
Subchronic Toxicity Database																		
(MM/DYR)																		
(TYPE)																		
UGI Columbia (Former MGP Site)																		
Columbia, Pennsylvania																		
CAS #	COMPOUND			Toxic Equivalent Factor (TEF)	Source	Oral to Dermal Absorption Extrapolation Factor	Source	Subchronic Dermal RfD (mg/kg/day)	Subchronic Oral RfD (mg/kg-day)	RfD Source	Test Species	Study Type & Length (Route of Exposure)	Target Organ/ Critical Effect	Confidence Level	Uncertainty and Modifying Factors UF	MF
	Volatile Organic Compounds															
71-43-2	Benzene					1		3 00E-03	0 003	NCEA; substitute chronic for subchronic						
100-41-4	Ethylbenzene					0 92	NCEA 11-1-93	8 20E-02	1 00E-01	IRIS		Chronic adopted as subchronic				
127-18-4	Tetrachloroethene					1	EPA Region III	1 00E-01	1 00E-01	HEAST	mouse	8 week, oral	hepatotoxicity		100	
108-88-3	Toluene					1	NCEA 10-8-92	2 00E+00	2	HEAST	rat	13 weeks, gavage	liver and kidney, altered weight			
79-01-6	Trichloroethene					1	EPA Region III	6 00E-03	0 006	EPA Provisional, Substitute Chronic for Subchronic (NCEA)						
	1,2,4-Trimethylbenzene					1		5 00E-02	5 00E-02	EPA Provisional, Substitute Chronic for Subchronic						
1330-20-7	Xylenes (Mixed)					0 9	NCEA 10-8-92	1 80E+00	2	IRIS; Substitute chronic for subchronic						
	Semivolatile Organic Compounds															
	Non-Carcinogenic PAHs															
83-32-9	Acenaphthene					0 7	ATSDR 1993	4 20E-01	6 00E-01	HEAST	mouse	90 day gavage	hepatotoxicity		3 00E+02	1 00E+00
208-96-8	Acenaphthylene					0 7	ATSDR 1993	NA	NA, Data Inadequate (NCEA,1993)							
120-12-7	Anthracene					0 9	ATSDR 1993	1 80E+00	3 00E+00	HEAST	mouse	90 day gavage	no effects observed		3 00E+02	1 00E+00
191-24-2	Benzo(ghi)perylene					1		NA	NA, Data Inadequate (NCEA,1995)							
206-44-0	Fluoranthene					0 7	ATSDR 1993	2 80E-01	4 00E-01	HEAST	mouse	90-day gavage	nephropathy, weight changes, hematological changes		3 00E+02	1 00E+00
86-73-7	Fluorene					0 7	ATSDR 1993	2 80E-01	4 00E-01	HEAST	mouse	13 week gavage	decreased erythrocyte counts		3 00E+02	1 00E+00
	1-Methylnaphthalene					1		4 00E-01	4 00E-01	Substitute value for naphthalene						
91-57-6	2-Methylnaphthalene					0 8	ATSDR 1993	3 20E-01	4 00E-01	Substitute value for naphthalene						
91-20-3	Naphthalene					0 8	ATSDR 1993	3 20E-01	4 00E-01	Substitute value for fluoranthene						
85-01-8	Phenanthrene					1		NA	NA, Data Inadequate (HEAST)							
129-00-0	Pyrene					0 6	ATSDR 1993	3 40E-01	3 00E-01	HEAST	mouse	13 week gavage	kidney effects		3 00E+02	1 00E+00
	Carcinogenic PAHs															
56-55-3	Benzo(a)anthracene					NA	NCEA 2-9-93	NA	NA	HEAST						
50-32-8	Benzo(a)pyrene					NA	NCEA 7-21-91	NA								
205-99-2	Benzo(b)fluoranthene					NA	NCEA 2-11-92	NA	NA	HEAST						
207-08-9	Benzo(k)fluoranthene					NA		NA								
218-01-9	Chrysene					NA	NCEA 2-11-92	NA	NA	HEAST						
53-70-3	Dibenz(ah)anthracene					NA	NCEA 12-29-92	NA								
193-39-5	Indeno(123-cd)pyrene					NA	NCEA 2-11-92	NA								
	Phthalates															
117-81-7	bis(2-Ethylhexyl)phthalate					0 55	ATSDR 1991	1 10E-02	2 00E-02	IRIS						
	Other SVOCs															
132-64-9	Dibenzofuran					0 7	ATSDR 1993	2 80E-03	4 00E-03	EPA Provisional						
	Inorganic Compounds															
7429-90-5	Aluminum					0 27	ATSDR 1992	2 70E-01	1	NCEA						
7440-38-2	Arsenic					0 85	NCEA 10-8-92	2 85E-04	3 00E-04	HEAST	human	oral	keratosis	chronic RfD adopted as subchronic		
7440-39-3	Barium					1	NCEA 8-8-93	7 00E-02	7 00E-02	HEAST	human	10 weeks, drinking water	increased blood pressure	chronic RfD adopted as subchronic	3 00E+00	1 00E+00
7440-41-7	Beryllium					0 01	NCEA 5-12-94	5 00E-05	5 00E-03	HEAST	rat	lifetime	no critical effect observed	chronic RfD adopted as subchronic		
7440-43-9	Cadmium (Food)					0 025	IRIS	2 50E-05	1 00E-03	IRIS						
7440-43-9	Cadmium (Water)					0 05	IRIS	2 50E-05	5 00E-04	IRIS						
16065-83-1	Chromium III					0 01	ATSDR 1993	1 00E-02	1 00E+00	HEAST	rat	840 day, diet	none observed		1 00E+03	1 00E+00
18540-29-9	Chromium (VI)					0 01	ATSDR 1993	2 00E-04	2 00E-02	HEAST	rat	1 year, drinking water	none observed		1 00E+02	1 00E+00
7440-50-8	Copper					0 8	NCEA 8-24-92	7 80E-01	1 3	HEAST*	human	oral	gastrointestinal irritation			
57-12-5	Cyanide					NA	NCEA 6-3-91	NA	2 00E-02	HEAST	rat	104 weeks, diet	decreased body weight, thyroid effects, myelin degeneration		5 00E+02	1 00E+00
7439-89-6	Iron					1		3 00E-01	0 3	NCEA						
7439-92-1	Lead					1		NA	NA	HEAST						
7439-96-5	Manganese					0 05	NCEA 1/22/96	1 15E-03	2 30E-02	IRIS**	human	diet	CNS effects	chronic RfD adopted as subchronic	1 00E+00	1 00E+00
7440-02-0	Nickel					0 01	NCEA 8-5-92	2 00E-04	2 00E-02	HEAST	rat	2 year, diet	decrease body/organ weight	chronic RfD adopted as subchronic	3 00E+02	1 00E+00
7440-28-0	Thallium					1	ATSDR 1992	8 00E-05	8 00E-05	Substitute Chronic for Subchronic						
7440-62-2	Vanadium					0 02	ATSDR 1992	1 40E-04	7 00E-03	HEAST	rat	lifetime, drinking water				
7440-66-6	Zinc					0 25	EPA Region III	7 50E-02	3 00E-01	HEAST	human	10 weeks, diet	decreased blood enzyme	chronic RfD adopted as subchronic	3 00E+00	1 00E+00

TABLE 11A
SUBCHRONIC DOSE-RESPONSE INFORMATION FOR COCs
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site																									
Subchronic Toxicity Database																									
(MM/DD/YR)																									
(TYPE)																									
UGI Columbia (Former MGP Site)																									
Columbia, Pennsylvania																									
																			Absorption Adjustment Factors						
		Subchronic Inhalation	RID	Inhalation	RfC	Test	Study Type	Target Organ/	Confidence	Uncertainty and Modifying		Dermal Absorption Factor		Dermal Absorption Factor (K_p)		Soil/Sediment Ingestion		Water Ingestion		Food Ingestion		Inhalation	
		RID	Source	RfC	Source	Species	& Length	Critical Effect	Level	Factors		(Soil)		(Water)		AF		AF		AF		AF	
CAS #	COMPOUND	(mg/kg-day)		(mg/m³)			(Route of Exposure)			UF	MF	(unitless)	Source	(cm/hr)	Source	(fraction)	Source	(fraction)	Source	(fraction)	Source	(fraction)	Source
	Volatile Organic Compou																						
71-43-2	Benzene	0 00171	NCEA	5 88E-03	NCEA							5 00E-04	Region III	2.10E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
100-41-4	Ethylbenzene			1 00E+00	IRIS							3 00E-02	Region III	7.40E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
127-18-4	Tetrachloroethene			4 8	Superfund Technical Information Center							3 00E-02	Region III	4 80E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
108-88-3	Toluene	0 114	IRIS	4 00E-01	IRIS							3 00E-02	Region III	4 50E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
79-01-6	Trichloroethene			NA								3 00E-02	Region III	1 80E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	1,2,4-Trimethylbenzene			6 00E-03	EPA1993	(EPA 63 42/08-02-93)						3 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
1330-20-7	Xylenes (Mixed)											3 00E-02	Region III	8 00E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Semivolatile Organic Co																						
	Non-Carcinogenic PAHs																						
83-32-9	Acenaphthene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
208-96-8	Acenaphthylene											1 00E-01	Region III	9 25E-03	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
120-12-7	Anthracene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
191-24-2	Benzo(ghi)perylene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
206-44-0	Fluoranthene											1 00E-01	Region III	3 80E-01	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
86-73-7	Fluorene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
	1-Methylnaphthalene											1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
91-57-6	2-Methylnaphthalene											1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
91-20-3	Naphthalene			Contact the Superfund Health Risk Technical Support Center (HEAST)								1 00E-01	Region III	6 80E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
85-01-8	Phenanthrene			NA	HEAST							1 00E-01	Region III	2 30E-01	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
129-00-0	Pyrene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
	Carcinogenic PAHs																						
56-55-3	Benzo(a)anthracene			NA	HEAST							1 00E-01	Region III	8 10E-01	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
50-32-8	Benzo(a)pyrene											1 00E-01	Region III	1 20E+00	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
205-99-2	Benzo(b)fluoranthene			Contact Superfund Health Risk Technical Support Center (HEAST)								1 00E-01	Region III	1 20E+00	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
207-08-9	Benzo(k)fluoranthene											1 00E-01	Region III	3 16E-02	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
218-01-9	Chrysene											1 00E-01	Region III	8 10E-01	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
53-70-3	Dibenzo(ah)anthracene											1 00E-01	Region III	2 70E+00	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
193-39-5	Indeno(123-cd)pyrene											1 00E-01	Region III	1 80E+00	EPA Dermal	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
	Phthalates																						
117-81-7	bis(2-Ethylhexyl)phthalate			CALL (513) 569-7300								1 00E-01	Region III	3 30E-02		1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Other SVOCs																						
132-64-9	Dibenzofuran											1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Inorganic Compounds																						
7429-90-5	Aluminum	0 001	NCEA	3 50E-03	NCEA							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00		1 00E+00	Assumed	1 00E+00	Assumed
7440-38-2	Arsenic											3 20E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
7440-39-3	Barium			5 00E-03	HEAST	rat	4 month, inh	fetotoxicity		1 00E+02	1 00E+00	1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-41-7	Beryllium											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-43-9	Cadmium (Food)	0 0000575	NCEA	2 00E-04	EPA Region III							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	5 00E-02	Personal Ca	1 00E+00	Assumed
7440-43-9	Cadmium (Water)	0 0000575	NCEA	2 00E-04	EPA Region III							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	5 00E-02	Personal Ca	1 00E+00	Assumed
18065-03-1	Chromium III			NA	HEAST							1 00E-02	Region III			1 00E+00	Personal	1 00E+00	Personal	1 00E+00	Personal Ca	1 00E+00	
18540-29-9	Chromium (VI)			Contact the Superfund Health Risk Technical Support Center								1 00E-02	Region III			1 00E+00	Personal	1 00E+00	Personal	1 00E+00	Personal Ca	1 00E+00	
7440-50-8	Copper											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
57-12-5	Cyanide											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
7439-89-6	Iron			NA	HEAST							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
7439-92-1	Lead			NA	IRIS							1 00E-02	Region III			1 00E+00	Personal	1 00E+00	Personal	1 00E+00	Personal Ca	1 00E+00	
7439-96-5	Manganese	0 0000143	IRIS	5 01E-05	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-02-0	Nickel											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Personal Ca	1 00E+00	Assumed
7440-28-0	Thallium											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-62-2	Vanadium											1 00E-02	Region III	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assumed
7440-66-6	Zinc											1 00E-02	Region III			1 00E+00	Personal	1 00E+00	Personal	1 00E+00	Personal Ca	1 00E+00	

TABLE 11B
CHRONIC DOSE-RESPONSE INFORMATION FOR COCs
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site Chronic Toxicity Database (MM/D/YR) (TYPE) UGI Columbia (Former MGP Site) Columbia, Pennsylvania																		
CAS #	COMPOUND	Toxic Equivalent Factor (TEF)	Source	Oral to Dermal Absorption Extrapolation Factor	Source	Chronic Dermal RfD (mg/kg/day)	Chronic Oral RfD (mg/kg/day)	RfD Source	Test Species	Study Type & Length (Route of Exposure)	Target Organ/ Critical Effect	Confidence Level	Uncertainty and Modifying Factors UF	MF
	Volatile Organic Compounds													
71-43-2	Benzene			1		3 00E-03	3 00E-03	NCEA						
100-41-4	Ethylbenzene			0 82	NCEA 11-1-93	8 20E-02	1 00E-01	IRIS	rat	oral	liver, kidney	low	1000	1
127-18-4	Tetrachloroethene			1	NCEA 11-1-93	1 00E-02	1 00E-02	IRIS	mouse	6 week, gavage	hepatotoxicity	med	1000	1
108-88-3	Toluene			1	NCEA 10-9-92	2 00E-01	2 00E-01	IRIS	rat	13 week gavage	liver and kidney effects, change in weight	high	1000	1
79-01-6	Trichloroethene			1		6 00E-03	0 006	EPA Provisional, NCEA						
	1,2,4 Trimethylbenzene			1		5 00E-02	5 00E-02	EPA Provisional, NCEA						
1330-20-7	Xylenes (Mixed)			0 9	NCEA 10-9-92	1 80E+00	2	IRIS	rat	gavage	hypoactivity, decreased body weight, increased mortality	medium	100	1
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene			0 7	ATSDR 1993	4 20E-02	6 00E-02	IRIS	mouse	90 day gavage	hepatotoxicity	low	3 00E+03	1 00E+00
208-96-8	Acenaphthylene			0 7	ATSDR 1993	NA	NA	Data inadequate (NCEA,1995)						
120-12-7	Anthracene			0 9	ATSDR 1993	1 80E-01	3 00E-01	IRIS	mouse	90 day gavage	none observed	low	3 00E+03	1 00E+00
191-24-2	Benzo(ghi)perylene			1		NA	NA	Data inadequate (NCEA,1988)						
206-44-0	Fluoranthene			0 7	ATSDR 1993	2 80E-02	4 00E-02	IRIS	mouse	13 week gavage	nephropathy, increased liver weight, hematological alterations	medium	3 00E+03	1 00E+00
86-73-7	Fluorene			0 7	ATSDR 1993	2 80E-02	4 00E-02	IRIS	mouse	13 week gavage	decreased RBCs, packed cell volume, and hemoglobin	medium	3 00E+03	1 00E+00
	1-Methylnaphthalene			1		4 00E-02	4 00E-02	Substitute value for naphthalene						
91-57-6	2-Methylnaphthalene			0 8	ATSDR 1993	3 20E-02	4 00E-02	Substitute value for naphthalene						
91-20-3	Naphthalene			0 8	ATSDR 1993	3 20E-02	4 00E-02	Substitute value for fluoranthene						
85-01-8	Phenanthrene			1		3 00E-03	3 00E-02	Substitute value for pyrene						
129-00-0	Pyrene			0 8	ATSDR 1993	2 40E-02	3 00E-02	IRIS	mouse	13 week, gavage	kidney effects	medium	3 00E+03	1 00E+00
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene			NA	NCEA 2 8 93	NA	NA	IRIS						
50-32-8	Benzo(a)pyrene			NA	NCEA 7-21 93	NA	NA	IRIS						
205-99-2	Benzo(b)fluoranthene			NA	NCEA 2-11-92	NA	NA	IRIS						
207-08-9	Benzo(k)fluoranthene			1		NA	NA	IRIS						
218-01-9	Chrysene			NA	NCEA 2-11-92	NA	NA	IRIS						
53-70-3	Dibenzo(ah)anthracene			NA	NCEA 12-29-92	NA	NA	IRIS						
193-39-5	Indeno(123-cd)pyrene			NA	NCEA 2-11-92	NA	NA	IRIS						
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate			0 06	ATSDR 1991	1 10E-02	2 00E-02	IRIS						
	Other SVOCs													
132-64-9	Dibenzofuran			0 7	ATSDR 1993	2 80E-03	4 00E-03	EPA Provisional Value						
	Inorganic Compounds													
7429-90-5	Aluminum			0 27	ATSDR 1992	2 70E-01	1	NCEA						
7440-38-2	Arsenic			0 95	NCEA 10-9-92	2 85E-04	3 00E-04	IRIS	human	drinking water, food	hyperpigmentation, keratosis	medium	3 00E+00	1 00E+00
7440-39-3	Barium			1	NCEA 8-6-93	7 00E-02	7 00E-02	IRIS	human	drinking water	increased blood pressure	medium	3 00E+00	1 00E+00
7440-41-7	Beryllium			0 01	NCEA 8-12-94	5 00E-05	5 00E-03	IRIS	rat	drinking water	no adverse effects	low	1 00E+02	1 00E+00
7440-43-9	Cadmium (Food)			0 025	IRIS	2 50E-05	1 00E-03	IRIS		diet	proteinuria	high	1 00E+01	1 00E+00
7440-43-9	Cadmium (Water)			0 05	IRIS	2 50E-05	5 00E-04	IRIS	human	drinking water	proteinuria	high	1 00E+01	1 00E+00
16065-83-1	Chromium III			0 01	ATSDR 1993	1 00E-02	1 00E+00	IRIS	rat	feeding study	none observed	low	1 00E+02	1 00E+01
18540-29-9	Chromium (VI)			0 01	ATSDR 1993	5 00E-05	5 00E-03	IRIS	rat	1 year, drinking	none reported	low	5 00E+02	1 00E+00
7440-50-8	Copper			0 8	NCEA 8-24-92	0 76	1 3	HEAST	human	oral	gastrointestinal irritation			
57-12-5	Cyanide			NA	NCEA 6-3-91	NA	2 00E-02	IRIS	rat	7 year, dietary	weight loss, thyroid effects, myelin degeneration	medium	1 00E-02	5 00E-00
7439-89-6	Iron			1		0 3	0 3	NCEA						
7439-92-1	Lead			1		NA	NA	IRIS						
7439-96-5	Manganese			0 05	NCEA 1/22/96	0 00118	2 30E-02	IRIS	human	food ingestion	CNS effects	medium	1 00E+00	1 00E+00
7440-02-0	Nickel			0 01	NCEA 6-8-92	0 0002	2 00E-02	IRIS	rat	2 year, feeding study	decreased body and organ weight	low	3 00E-02	1 00E+00
7440-28-0	Thallium			1	ATSDR 1992	0 00008	8 00E-05	IRIS (Thallium Sulfate)						
7440-62-2	Vanadium			0 02	ATSDR 1992	0 00014	7 00E-03	HEAST	rat	lifetime, drinking water study				
7440-66-6	Zinc			0 25	EPA Region III	0 075	3 00E-01	IRIS	human	10 week diet supplement study	47% decrease in erythrocyte superoxide dismutase concentration	medium	3 00E+00	1 00E+00

TABLE 11B
CHRONIC DOSE-RESPONSE INFORMATION FOR COCs
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site																												
Chronic Toxicity Database																												
(MM/DD/YR)																												
(TYPE)																												
UGI Columbia (Former MGP Site)																												
Columbia Pennsylvania																												

		Chronic														Absorption Adjustment Factors							
		Inhalation	RfD	Inhalation	RfC	Test	Study Type		Confidence	Uncertainty and Modifying Factors		Dermal Absorption Factor	Dermal Absorption Factor (K_p)			Soil/Sediment Ingestion		Water Ingestion		Food Ingestion		Inhalation	
CAS #	COMPOUND	RfD (mg/kg/day)	Source	RfC (mg/m³)	Source	Species	& Length (Route of Exposure)	Target Organ/ Critical Effect	Level	UF	MF	(Soil) (unitless)	Source	(Water) (cm/hr)	Source	AF (fraction)	Source	AF (fraction)	Source	AF (fraction)	Source	AF (fraction)	Source
	Volatile Organic Compoun																						
71-43-2	Benzene	0 00171	NCEA	9 99E-03	NCEA							8 00E-04	Region III	2 10E-02	TABLE 5-7 Dermal Guidance	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
100-41-4	Ethylbenzene			1 00E+00	IRIS	rat, rabbit	inhalation	developmental toxicity	low	3 00E-02	1 00E+00	3 00E-02	Region III	7 40E-02	TABLE 5-7 Dermal Guidance	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
127-18-4	Tetrachloroethene			4 6	Superfund Technical Information Center							3 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed
108-88-3	Toluene	1 14E-01	IRIS	4 00E-01	IRIS	rat	2 year occupational	neurological effect	medium	300	1	3 00E-02	Region III	4 50E-02	TABLE 5-7 Dermal Guidance	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
79-01-6	Trichloroethene			NA								3 00E-02	Region III	1 80E-02	TABLE 5-7 Dermal Guidance	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed
	1,2,4-Trimethylbenzene			6 00E-03	EPA1993 (EPA 93-42/08-02-93)							3 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
1330-20-7	Xylenes (Mixed)			NA								3 00E-02	Region III	8 00E-02	TABLE 5-7 Dermal Guidance for m-Xylene	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Semivolatile Organic Comp																						
	Non-Carcinogenic PAHs																						
83-32-9	Acenaphthene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
208-96-8	Acenaphthylene			NA	IRIS							1 00E-01	Region III	6 25E-03	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
120-12-7	Anthracene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
191-24-2	Benzo(ghi)perylene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
206-44-0	Fluoranthene			NA	IRIS							1 00E-01	Region III	3 80E-01	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
86-73-7	Fluorene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
	1-Methylnaphthalene											1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
91-57-6	2-Methylnaphthalene											1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
91-20-3	Naphthalene			NA	IRIS							1 00E-01	Region III	6 80E-03	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
85-01-8	Phenanthrene			NA	IRIS							1 00E-01	Region III	2 30E-01	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
129-00-0	Pyrene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
	Carcinogenic PAHs																						
56-55-3	Benzo(a)anthracene			NA	IRIS							1 00E-01	Region III	8 10E-01	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
50-32-8	Benzo(a)pyrene			NA	IRIS							1 00E-01	Region III	1 20E+00	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
205-99-2	Benzo(b)fluoranthene			NA	IRIS							1 00E-01	Region III	1 20E+00	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
207-08-9	Benzo(k)fluoranthene			NA	IRIS							1 00E-01	Region III	3 16E-02	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
218-01-9	Chrysene			NA	IRIS							1 00E-01	Region III	6 10E-01	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
53-70-3	Dibenz(ah)anthracene			NA	IRIS							1 00E-01	Region III	2 70E+00	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
193-39-5	Indeno(123-cd)pyrene			NA	IRIS							1 00E-01	Region III	1 80E+00	EPA Dermal Exposure Assessment (1992)	1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
	Phthalates																						
117-81-7	bis(2-Ethylhexyl)phthalate			NA								1 00E-01	Region III	3 30E-02	TABLE 5-7	1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Other SVOCs																						
132-64-9	Dibenzofuran			NA								1 00E-01	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
	Inorganic Compounds																						
7429-90-5	Aluminum	0 001	NCEA	3 50E-03	NCEA							1 00E-02	Region III										
7440-38-2	Arsenic			NA	IRIS							3 20E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-39-3	Barium			5 00E-04	HEAST	rat	4 month, inhalation	fetotoxicity		1 00E+03	1 00E+00	1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-41-7	Beryllium			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	5 00E-02	Persona	1 00E+00	Assumed
7440-43-9	Cadmium (Food)	0 0000875	A Regio	3 00E-04	EPA Region III							1 00E-03	Region III			1 00E+00	Assumed	1 00E+00	Assumed	5 00E-02	Persona	1 00E+00	Assumed
7440-43-9	Cadmium (Water)	0 0000875	A Regio	2 00E-04	EPA Region III							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
16065-83-1	Chromium III			NA	IRIS							1 00E-02	Region III			1 00E+00	Personal Com	1 00E+00	Personal C	1 00E+00	Personal Communication (see below)		
18540-29-9	Chromium (VI)	0 0000001	NCEA	3 50E-07	NCEA							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed
7440-50-8	Copper			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
57-12-5	Cyanide			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed
7439-89-6	Iron											1 00E-02	Region III			1 00E+00	Personal Com	1 00E+00	Personal C	1 00E+00	Personal Communication (see below)		
7439-92-1	Lead			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed
7439-96-5	Manganese	0 0000143	IRIS	5 01E-05	IRIS	human	occupational	impairment of neuro-behavioral function				1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Persona	1 00E+00	Assumed
7440-02-0	Nickel			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-28-0	Thallium											1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed			1 00E+00	Assumed
7440-62-2	Vanadium											1 00E-02	Region III			1 00E+00	Personal Com	1 00E+00	Personal C	1 00E+00	Personal Communication (see below)		
7440-66-6	Zinc			NA	IRIS							1 00E-02	Region III			1 00E+00	Assumed	1 00E+00	Assumed	1 00E+00	Assume	1 00E+00	Assumed

TABLE 11C
CARCINOGENIC DOSE-RESPONSE INFORMATION FOR COCS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site																									
Cancer Toxicity Database																									
(MM/DYR)																									
(TYPE)																									
UGI Columbia (Former MGP Site)																									
Columbia, Pennsylvania																									
CAS #	COMPOUND	Oral to Dermal Absorption Extrapolation Factor	Source	Dermal Slope Factor (mg/kg/day)	Oral Slope Factor (mg/kg/day)-1	Source	Weight o Evidenc	Test Species	Study Type & Length (Route of Exposure)	Tumor Type (Carc. Class)	Uncertainty and Modifying Factors UF	MF	Inhalation Unit Risk (µg/m³)-1	Source	Weight of Evidence	Test Species	Study Type & Length (Route of Exposure)	Tumor Type (Carc. Type)	Dermal Absorption Factor (Soil) (unitless)	Source	Dermal Absorption Facto (Water) (cm/hr)
	Volatile Organic Compounds																				
71-43-2	Benzene	1		0 029	2 90E-02	IRIS	A	animals -	occupational, inhalation	leukemia			8 30E-06	IRIS	A	human	inhalation occupational	leukemia	5 00E-04	Region III	2 10E-02
100-41-4	Ethylbenzene	0 92	NCEA 11-1-93	0			D												3 00E-02	Region III	7 40E-02
127-18-4	Tetrachloroethene	1	NCEA 11-1-93	0 052	5 20E-02	NCEA							5 80E-07	Superfund Technical Information Center					3 00E-02	Region III	4 80E-02
108-88-3	Toluene	1	NCEA 10-8-92	0						D									3 00E-02	Region III	4 50E-02
79-01-6	Trichloroethene	1		0 011	1 10E-02	NCEA							1 70E-06	Superfund Technical Information Center					3 00E-02	Region III	1 80E-02
	1,2,4-Trimethylbenzene	1		NA	NA								NA						3 00E-02	Region III	
1330-20-7	Xylenes (Mixed)	0 9	NCEA 10-8-92	0			D			D								D	3 00E-02	Region III	8 00E-02
	Semivolatile Organic Compounds																				
	Non-Carcinogenic PAHs																				
83-32-9	Acenaphthene	0 7	ATSDR 1993	NA	NA	IRIS							NA	IRIS					1 00E-01	Region III	3 16E-02
208-96-8	Acenaphthylene	0 7	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	5 25E-03
120-12-7	Anthracene	0 8	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	3 16E-02
191-24-2	Benzo(ghi)perylene	1		NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	3 16E-02
206-44-0	Fluoranthene	0 7	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	3 80E-01
86-73-7	Fluorene	0 7	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	3 16E-02
	1-Methylnaphthalene	1		0															1 00E-01	Region III	
91-57-6	2-Methylnaphthalene	0 8	ATSDR 1993	0															1 00E-01	Region III	
91-20-3	Naphthalene	0 8	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	6 80E-02
85-01-8	Phenanthrene	1		NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	2 30E-01
118-00-0	Pyrene	0 8	ATSDR 1993	NA	NA	IRIS	D						NA	IRIS					1 00E-01	Region III	3 16E-02
	Carcinogenic PAHs																				
56-55-3	Benzo(a)anthracene	NA	NCEA 2-9-93	NA	7 30E-01	EPA Provisional							NA	IRIS					1 00E-01	Region III	9 10E-01
50-32-8	Benzo(a)pyrene	NA	NCEA 7-21-91	NA	7 30E+00	EPA Provisional				stomach tumors			8 86E-04	EPA Region III					1 00E-01	Region III	1 20E+00
205-99-2	Benzo(b)fluoranthene	NA	NCEA 2-11-92	NA	7 30E-01	EPA Provisional							NA	IRIS					1 00E-01	Region III	1 20E+00
207-08-9	Benzo(k)fluoranthene	NA		NA	7 30E-02	EPA Provisional				lung adenoma			NA						1 00E-01	Region III	3 16E-02
218-01-9	Chrysene	NA	NCEA 2-11-92	NA	7 30E-03	EPA Provisional				carcinomas and malignant lymphoma, skin carc			NA	HEAST					1 00E-01	Region III	9 10E-01
53-70-3	Dibenz(ah)anthracene	NA	NCEA 12-29-92	NA	7 3	EPA Provisional				pulmonary, mammary carcinomas			NA						1 00E-01	Region III	2 70E+00
193-39-5	Indeno(123-cd)pyrene	NA	NCEA 2-11-92	NA	7 30E-01	EPA Provisional				epidermoid carcinomas			NA	HEAST					1 00E-01	Region III	1 90E+00
	Phthalates																				
117-81-7	bis(2-Ethylhexyl)phthalate	0 55	ATSDR 1991	0 025454545	1 40E-02	IRIS	C	rat	dietary	leukemia			4 00E-08	EPA Region III					1 00E-01	Region III	3 30E-02
	Other SVOCs																				
132-64-9	Dibenzofuran	0.7	ATSDR 1993	0					no human or animal data	D								D	1 00E-01	Region III	
	Inorganic Compounds																				
7429-90-5	Aluminum	0 27	ATSDR 1993	0															1 00E-02	Region III	
7440-38-2	Arsenic	0 95	NCEA 10-8-92	1 878847368	1 50E+00	IRIS	A	human	inhalation, drinking water	lung cancer, multiple organ cancers, skin cance			4 30E-03	IRIS		human	occupational study	respiratory tumors	3 20E-02	Region III	
7440-39-3	Barium	1	NCEA 8-6-93	NA	NA	IRIS							NA	IRIS					1 00E-02	Region III	
7440-41-7	Beryllium	0 01	NCEA 5-12-94	430	4 30E+00	IRIS	B2	rats, mon	inhalation, injection	lung cancer, osteosarcomas			2 40E-03	IRIS		human	occupational study	lung, tumors	1 00E-02	Region III	
7440-43-9	Cadmium (Food)	0 025	IRIS	NA	NA	IRIS	B1	human	occupational	lung cancer			1 80E-03	IRIS					1 00E-02	Region III	
7440-43-9	Cadmium (Water)	0 05	IRIS	NA	NA	IRIS	B1	human	occupational	lung cancer			1 80E-03	IRIS					1 00E-02	Region III	
16065-83-1	Chromium III	0 01	ATSDR 1993	NA	NA	IRIS													1 00E-02	Region III	
18540-29-9	Chromium (VI)	0 01	ATSDR 1993	NA	NA	IRIS	A	human	occupational	lung cancer			1 20E-02	IRIS		human	occupational	lung tumors	1 00E-02	Region III	
7440-50-8	Copper	0 6	NCEA 6-24-92	NA	NA	IRIS	D						NA	IRIS					1 00E-02	Region III	
57-12-5	Cyanide	NA	NCEA 8-3-91	NA	NA	IRIS	D						NA	IRIS					1 00E-02	Region III	
7439-89-6	Iron	1		NA	NA														1 00E-02	Region III	
7439-92-1	Lead	1		NA	NA	IRIS	B2	rat	dietary, subcutaneous injection	renal tumors			NA	HEAST					1 00E-02	Region III	
7439-96-5	Manganese	0 05	NCEA 1/22/96	NA	NA	IRIS	D						NA	IRIS					1 00E-02	Region III	
7440-02-0	Nickel	0 01	NCEA 8-6-92	NA	NA	IRIS													1 00E-02	Region III	
7440-28-0	Thallium	1	ATSDR 1992	NA	NA								NA						1 00E-02	Region III	
7440-62-2	Vanadium	0 02	ATSDR 1992	NA	NA														1 00E-02	Region III	
7440-66-6	Zinc	0 25	NCEA 10-8-92	NA	NA	IRIS	D						NA	IRIS					1 00E-02	Region III	

TABLE 11C
CARCINOGENIC DOSE-RESPONSE INFORMATION FOR COCS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

UGI Columbia Former MGP Site											
Cancer Toxicity Database											
(MM/D/YR)											
(TYPE)											
UGI Columbia (Former MGP Site)											
Columbia, Pennsylvania											
		[Kp]		Absorption Adjustment Factor							
				Soil/Sediment Ingestion		Water Ingestion		Food Ingestion		Inhalation	
CAS #	COMPOUND	Source		AF (fraction)	Source	AF (fraction)	Source	AF (fraction)	Source	AF (fraction)	Source
	Volatile Organic Co										
71-43-2	Benzene	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
100-41-4	Ethylbenzene	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
127-18-4	Tetrachloroethene	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
108-88-3	Toluene	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
79-01-6	Trichloroethene	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
	1,2,4-Trimethylbenz			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
1330-20-7	Xylenes (Mixed)	TABLE 5-7 for m-Xylene		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
	Semivolatile Organ										
	Non-Carcinogenic P										
83-32-9	Acenaphthene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
208-96-8	Acenaphthylene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
120-12-7	Anthracene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
191-24-2	Benzo(ghi)perylene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
206-44-0	Fluoranthene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
86-73-7	Fluorene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
	1-Methylnaphthalene			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
91-57-6	2-Methylnaphthalene			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
91-20-3	Naphthalene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
85-01-8	Phenanthrene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
129-00-0	Pyrene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
50-32-8	Benzo(a)pyrene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
205-99-2	Benzo(b)fluoranthen	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
207-08-9	Benzo(k)fluoranthen	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
218-01-9	Chrysene	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
53-70-3	Dibenz(a,h)anthrace	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
193-39-5	Indeno(1,2,3-cd)pyren	EPA Dermal Exposure Assessment (1		1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
	Phthalates										
117-81-7	bis(2-Ethylhexyl)phl	TABLE 5-7		1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
	Other SVOCs										
132-64-9	Dibenzofuran			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
	Inorganic Compou										
7429-90-5	Aluminum			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
7440-38-2	Arsenic			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
7440-39-3	Barium			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
7440-41-7	Beryllium			1.00E+00	Assumed	1.00E+00	Assumed	5.00E-02	Personal Com	1.00E+00	Assumed
7440-43-9	Cadmium (Food)			1.00E+00	Assumed	1.00E+00	Assumed	5.00E-02	Personal Com	1.00E+00	Assumed
7440-43-9	Cadmium (Water)			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
16065-83-1	Chromium III			1.00E+00	Personal Com	1.00E+00	Personal	1.00E+00	Personal Com	1.00E+00	
18540-29-9	Chromium (VI)			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
7440-50-8	Copper			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
57-12-5	Cyanide			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
7439-89-6	Iron			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
7439-92-1	Lead			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed
7439-96-5	Manganese			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Personal Com	1.00E+00	Assumed
7440-02-0	Nickel			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
7440-28-0	Thallium			1.00E+00	Assumed	1.00E+00	Assumed			1.00E+00	Assumed
7440-62-2	Vanadium			1.00E+00	Personal Com	1.00E+00	Personal	1.00E+00	Personal Com	1.00E+00	
7440-66-6	Zinc			1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed	1.00E+00	Assumed

TABLE 12
CHEMICALS OF POTENTIAL CONCERN (COPCs) BY MEDIA
UGI Columbia Former MGP Site
Columbia, Pennsylvania

ORIGINAL

COMPOUNDS	MEDIA				
	Surface Soil	Subsurface Soil	Groundwater	Sediments	Surface Water
Volatile Organic Compounds					
Benzene			✓		
Ethylbenzene			✓		
Tetrachloroethene			✓		
Toluene			✓		
Trichloroethene			✓		
1,2,4-Trimethylbenzene			✓		
Xylenes (total)			✓		
Semi-Volatile Organic Compounds					
Polycyclic Aromatic Hydrocarbons					
Acenaphthene			✓		
Acenaphthylene	✓ [1]	✓ [1]	✓ [1]		
Benzo(a)anthracene	✓	✓	✓	✓	
Benzo(b)fluoranthene	✓	✓	✓	✓	
Benzo(k)fluoranthene		✓			
Benzo(g,h,i)perylene	✓ [1]	✓ [1]		✓ [1]	
Benzo(a)pyrene	✓	✓	✓	✓	
Chrysene			✓		
Dibenzo(a,h)anthracene	✓	✓			
Fluoranthene			✓		
Indeno(1,2,3-cd)pyrene	✓	✓			
1-Methylnaphthalene			✓		
2-Methylnaphthalene			✓		
Naphthalene			✓		
Phenanthrene			✓		
Pyrene			✓		
Other SVOCs					
Bis(2-ethylhexyl)phthalate			✓		
Dibenzofuran			✓		
Inorganic Compounds					
Aluminum	✓	✓	✓	✓	
Arsenic	✓	✓		✓	
Barium			✓		
Beryllium	✓	✓			
Cadmium	✓				
Copper				✓	
Cyanide			✓		
Iron	✓	✓	✓	✓	
Lead	✓		✓		
Manganese	✓	✓	✓	✓	
Thallium		✓			

[1] Evaluated quantitatively

TABLE 13
SUMMARY OF POTENTIAL PATHWAYS FOR HUMAN EXPOSURE
UGI Columbia Former MGP Site
Columbia, Pennsylvania

EXPOSURE SCENARIO	LOCATION	EXPOSURE MEDIA	EXPOSURE PATHWAY	SAMPLE LOCATIONS
CURRENT LAND USE				
Trespasser	Exposure at the site property, south and west of the property	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4 [6]
				Atlantic (1996): SS-1,SS-2,SS-3,SS-4,SS-5,SS-6,SS-7, SS-8
			Inhalation of fugitive dust[3]	see footnote
		Sediment [4]	Incidental ingestion and dermal contact	Atlantic (1996): SD-3, SD-4 [7,8]
FUTURE LAND USE (in absence of remediation)				
Industrial Worker	Exposure at the property under a future industrial facility	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			Inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface soil [2]	inhalation of volatiles [4]	NUS (1991): SUB-4
				Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
On-site Construction Worker	Excavation project assumed to occur at the site property	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			Inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface soil [2]	Incidental ingestion and dermal contact	
			inhalation of volatiles [4]	NUS (1991): SUB-4
				Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
Off-site Construction Worker	Excavation project assumed to occur EITHER	Surface soil [1]	Incidental ingestion and dermal contact	Not evaluated because it consists of fill and gravel
			inhalation of fugitive dust[3]	
	between the Susquehanna River and the waste water treatment plant	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-11A,TP-F,TP-G
	OR		inhalation of volatiles [4]	Atlantic (1994): SB-10A
	between Front St. and the railroad tracks	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-8A, SB-8B, TP-A
			inhalation of volatiles [4]	Atlantic (1994): SB-7A
Hypothetical Off-Site Resident	Future residence located near the	Surface soil [1]	Incidental ingestion and dermal contact	Not evaluated because it consists of fill and gravel
			inhalation of fugitive dust[3]	
	Susquehanna River - it is assumed that soils have been reworked	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-11A,TP-F,TP-G
			inhalation of volatiles[4]	Atlantic (1994): SB-10A
		Groundwater[5]	Drinking water	Atlantic (1995): MW-6S,MW-6S2, MW-6D,MW-6D2, MW-7S,MW-7S2, MW-7D, MW-7D2 MW-2R, MW-5, MW-8S,MW-8S2, MW-8D, MW-8D2, CWW-1, CWW-1-2, CWW-2, CWW-2-2, MW-2R2, MW-52
Hypothetical On-Site Resident	Future residence located on-site	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface Soil	Inhalation of volatiles[4]	NUS (1991): SUB-4
				Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
		Groundwater[5]	Drinking water	NUS (1991): MW-3S
				Atlantic (1995):MW-3D,MW-3D2, MW-4,MW-9S2

[1]Surface Soil data includes soils from 0 to 6 inches deep
[2]Subsurface Soil data includes soils from 6 inches to 15 feet deep
[3] Fugitive dust calculated from surface soil (EPA, 1996b)
[4] Volatiles calculated from subsurface soil concentration
[6] See Table 13 for rationale
[6] Sample S-9 was excluded because it is anthropogenic background
[7] Sample SD-4 is a duplicate of SD-2
[8] Sample SD-1 was excluded because it is anthropogenic backgroun

TABLE 13
SUMMARY OF POTENTIAL PATHWAYS FOR HUMAN EXPOSURE
UGI Columbia Former MGP Site
Columbia, Pennsylvania

EXPOSURE SCENARIO	LOCATION	EXPOSURE MEDIA	EXPOSURE PATHWAY	SAMPLE LOCATIONS
CURRENT LAND USE				
Trespasser	Exposure at the site property, south and west of the property	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4 [6] Atlantic (1996): SS-1,SS-2,SS-3,SS-4,SS-5,SS-6,SS-7, SS-8
			Inhalation of fugitive dust[3]	see footnote
		Sediment [4]	Incidental ingestion and dermal contact	Atlantic (1996): SD-3, SD-4 [7,8]
FUTURE LAND USE (in absence of remediation)				
Industrial Worker	Exposure at the property under a future industrial facility	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			Inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface soil [2]	Inhalation of volatiles [4]	NUS (1991): SUB-4 Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
On-site Construction Worker	Excavation project assumed to occur at the site property	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			Inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface soil [2]	Incidental ingestion and dermal contact	
			Inhalation of volatiles [4]	NUS (1991): SUB-4 Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
Off-site Construction Worker	Excavation project assumed to occur EITHER	Surface soil [1]	Incidental ingestion and dermal contact	Not evaluated because it consists of fill and gravel
			Inhalation of fugitive dust[3]	
	between the Susquehanna River and the waste water treatment plant	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-11A,TP-F,TP-G
	OR		Inhalation of volatiles [4]	Atlantic (1994): SB-10A
	between Front St. and the railroad tracks	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-8A, SB-8B, TP-A
			Inhalation of volatiles [4]	Atlantic (1994): SB-7A
Hypothetical Off-Site Resident	Future residence located near the	Surface soil [1]	Incidental ingestion and dermal contact	Not evaluated because it consists of fill and gravel
			Inhalation of fugitive dust[3]	
	Susquehanna River - it is assumed that soils have been reworked	Subsurface soil [2]	Incidental ingestion and dermal contact	Atlantic (1995): SB-11A,TP-F,TP-G
			Inhalation of volatiles[4]	Atlantic (1994): SB-10A
		Groundwater[5]	Drinking water	Atlantic (1995): MW-6S,MW-6S2, MW-6D,MW-6D2, MW-7S,MW-7S2, MW-7D, MW-7D2 MW-2R, MW-5, MW-8S,MW-8S2, MW-8D, MW-8D2, CWW-1, CWW-1-2, CWW-2, CWW-2-2, MW-2R2, MW-52
Hypothetical On-Site Resident	Future residence located on-site	Surface soil [1]	Incidental ingestion and dermal contact	NUS (1991): S-1,S-2,S-3,S-4
			Inhalation of fugitive dust[3]	Atlantic (1996): SS-2,SS-3,SS-4
		Subsurface Soil	Inhalation of volatiles[4]	NUS (1991): SUB-4
				Atlantic (1994): SB-14A,SB-2B,SB-3A,SB-4A,SB-5A, SB-5B, SB-6A,SB-6B
		Groundwater[5]	Drinking water	NUS (1991): MW-3S
				Atlantic (1995),MW-3D,MW-3D2, MW-4,MW-9S2

[1]Surface Soil data includes soils from 0 to 6 inches deep
[2]Subsurface Soil data includes soils from 6 inches to 15 feet deep
[3]Fugitive dust calculated from surface soil (EPA, 1996b)
[4]Volatiles calculated from subsurface soil concentration
[5]See Table 13 for rationale
[6]Sample S-9 was excluded because it is anthropogenic background
[7]Sample SD-4 is a duplicate of SD-2
[8]Sample SD-1 was excluded because it is anthropogenic background

TABLE 14A
EXPOSURE ASSUMPTIONS BY SCENARIO
UGI Columbia Former MGP Site
Columbia, Pennsylvania

SCENARIO: **Trespasser**
Case/Timing: Current Conditions/Current Land Use
Primary Activity: Playing at and in the vicinity of the site

PARAMETER	Reasonable Maximum Exposure (RME) Values		Notes/References
Characteristics of Population			
Age	11 to 18	years	
Bodyweight (BW)	50	kg	USEPA, 1989b
Average Lifetime	70	years	USEPA, 1989b
Total Skin Area	15,300	cm^2	USEPA, 1989b (50th Percentile men and women)
Exposure Duration			
Exposure Frequency (EF)	40	days/year	2 days/week during warm weather
Exposure Duration (ED)	7	years	Chronic Exposure
Averaging Time (non-cancer) (AT)	2,555	days	7 years
Averaging Time (cancer) (AT)	25,550	days	70 year lifetime; EPA 1989
Dermal Contact with Sediment/Soil			
Soil to Skin Adherence Factor (AF)			
Hands	0.11	mg/cm^2	Kissel et al. 1996 (Soccer No. 1-3) 95% UCL
Arms	0.011	mg/cm^2	Kissel et al. 1996
Legs	0.054	mg/cm^2	Kissel et al. 1996
Face	0.019	mg/cm^2	Kissel et al. 1996; 95% UCL for head
Feet	0.11	mg/cm^2	Kissel et al. 1996; 95% UCL for hand
Skin Surface Area Available for Contact (SA)			
Hands	815	cm^2	USEPA, 1989b
Forearms	2157	cm^2	arms; USEPA, 1989b
Lower Legs	4855	cm^2	legs; USEPA, 1989b
Head	1310	cm^2	USEPA, 1989b
Feet	1120	cm^2	USEPA, 1989b
Incidental Ingestion of Sediment/Soil			
Ingestion Rate (IR)	50	mg/day	Estimate
Fraction Ingested (FI)	1	unitless	Assumed
Inhalation of Fugitive Dust			
Q/C Factor	71.87	g/m2-s per kg/m3	EPA 1996 (Table 3); Harrisburg - 1 Acre

[1]RME exposure assumptions are used for both central tendency and reasonable maximum exposure calculations in agreement with EPA 1996.

TABLE 14B
EXPOSURE ASSUMPTIONS BY SCENARIO
UGI Columbia Former MGP Site
Columbia, Pennsylvania

SCENARIO: **Industrial Worker**
Case/Timing: Future Conditions/Future Land Use
Primary Activity: Site walkovers/maintenance activity at future industrial facility

PARAMETER	Reasonable Maximum Exposure (RME)		
	Values		Notes/References
Characteristics of Population			
Age	18 to 45	years	
Bodyweight (BW)	70	kg	USEPA, 1989b
Average Lifetime	70	years	USEPA, 1989b
Total Skin Area	18,150	cm^2	USEPA, 1989b (50^{th} percentile men and women)
Exposure Duration			
Exposure Frequency (EF)	250	days/year	EPA 1997
Exposure Duration (ED)	25	years	USEPA, 1991
Averaging Time (non-cancer) (AT)	9,125	days	Exposure Duration x 365 days/year
Averaging Time (cancer) (AT)	25,550	days	365 x Lifetime
Dermal Contact with Soil			
Soil to Skin Adherence Factor (AF)			
Hands	0.11	mg/cm^2	Kissel et al. 1996 (Groundskeeper #2-5) 95% UCL
Arms	0.022	mg/cm^2	Kissel et al. 1996
Faces	0.011	mg/cm^2	Kissel et al. 1996; 95% UCL for head
Skin Surface Area Available for Contact (SA)			
Hands	904	cm^2	USEPA, 1989b
Forearms	2605	cm^2	USEPA, 1989b; arms
Head	1205	cm^2	USEPA, 1989b
Incidental Ingestion of Soil			
Ingestion Rate (IR)	50	mg/day	USEPA, 1991
Fraction Ingested (FI)	1	unitless	Assumed
Inhalation of Fugitive Dust			
Q/C Factor	71.87	g/m2-s per kg/m3	EPA 1996 (Table 3); Harrisburg - 1 Acre

[1]RME exposure assumptions are used for both central tendency and reasonable maximum exposure calculations in agreement with EPA 1996.

ORIGINAL (Red)

TABLE 14C
EXPOSURE ASSUMPTIONS BY SCENARIO
UGI Columbia Former MGP Site
Columbia, Pennsylvania

SCENARIO: **On-site and Off-site Construction Workers**
Case/Timing: Future Conditions/Future Land Use
Primary Activity: Project-specific activities including excavation

PARAMETER	Reasonable Maximum Exposure (RME)		
	Values		Notes/References
Characteristics of Population			
Age	18 to 19	years	
Bodyweight (BW)	70	kg	USEPA, 1989b
Average Lifetime	70	years	USEPA, 1989b
Total Skin Area	18,150	m^2	USEPA, 1989b
Exposure Duration			
Exposure Frequency (EF)	250	days/year	EPA 1997
Exposure Duration (ED)	1	year	Estimate
Averaging Time (non-cancer) (AT)	365	days	365 x Exposure Period
Averaging Time (cancer) (AT)	25,550	days	365 x Lifetime
Dermal Contact with Soil			
Soil to Skin Adherence Factor (AF)			
Hands	0.31	mg/cm^2	Kissel et al. 1996 (Irrigation Worker) 95% UCL
Arms	0.062	mg/cm^2	Kissel et al. 1996 (Irrigation Worker) 95% UCL
Faces (=Head)	0.0086	mg/cm^2	Kissel et al. 1996 (Irrigation Worker) 95% UCL
Skin Surface Area Available for Contact (SA)			
Hands	904	cm^2	USEPA, 1989b
Forearms (=Arms)	2605	cm^2	USEPA, 1989b
Head	1205	cm^2	USEPA, 1989b
Incidental Ingestion of Soil			
Ingestion Rate (IR)	480	mg/day	USEPA, 1989b
Fraction Ingested (FI)	1	unitless	Assumed
Inhalation of Fugitive Dust			
Q/C Factor	71.87	g/m2-s per kg/m3	EPA 1996 (Table 3); Harrisburg - 1 Acre

[1]RME exposure assumptions are used for both central tendency and reasonable maximum exposure calculations in agreement with EPA 1996.

TABLE 14D
EXPOSURE ASSUMPTIONS BY SCENARIO
UGI Columbia Former MGP Site
Columbia, Pennsylvania

ORIGINAL (Red)

SCENARIO: **Hypothetical Resident - Child (0 to 6 years old)**
Case/Timing: Future Conditions/Future Land Use
Primary Activity: Direct contact with soil during outdoor activity; drinking water

PARAMETER	Reasonable Maximum Exposure (RME)		
	Values		Notes/References
Characteristics of Population			
Age	0 to 6	years	
Bodyweight (BW)	14	kg	USEPA, 1989b
Average Lifetime	70	years	USEPA, 1989b
Total Skin Area	6,880	cm^2	USEPA, 1989b
Exposure Duration			
Exposure Frequency (EF): total, hands, head	350	days/year	EPA 1997
Exposure Frequency (EF): forearms, lower legs, feet	233	days/year	EPA 1997; 8 month exposure
Exposure Duration (ED)	6	years	6 year portion of residence
Averaging Time (non-cancer) (AT)	2,190	days	Exposure Duration x 365 days/year
Averaging Time (cancer) (AT)	25,550	days	Lifetime x 365 days/year
Dermal Contact with Soil			
Soil to Skin Adherence Factor (AF)			
Hands	1	mg/cm^2	EPA, 1998
Arms	1	mg/cm^2	EPA, 1998
Legs	1	mg/cm^2	EPA, 1998
Faces	1	mg/cm^2	EPA, 1998
Feet	1	mg/cm^2	EPA, 1998
Skin Surface Area Available for Contact (SA)			
Hands	386	cm^2	USEPA, 1989b
Forearms	921	cm^2	USEPA, 1989b; arms
Lower Legs	1672	cm^2	USEPA, 1989b; legs
Head	1050	cm^2	USEPA, 1989b
Feet	473	cm^2	USEPA, 1989b
Incidental Ingestion of Soil			
Ingestion Rate	200	mg/day	USEPA, 1991
Fraction Ingested	1	(unitless)	Assumed
Ingestion of Groundwater			
Volume Ingested	1	liters/day	USEPA, 1989b
Inhalation of Dust/Volatiles			
Q/C Factor	71.87	g/m2-s per kg/m3	EPA 1996 (Table 3); Harrisburg - 1 Acre

[1]RME exposure assumptions are used for both central tendency and reasonable maximum exposure calculations in agreement with EPA 1996.

ORIGINAL (Red)

TABLE 14E
EXPOSURE ASSUMPTIONS BY SCENARIO
UGI Columbia Former MGP Site
Columbia, Pennsylvania

SCENARIO: **Hypothetical Resident - Teen/Adult (6 to 30 years old)**
Case/Timing: Future Conditions/Future Land Use
Primary Activity: Direct contact with soil during outdoor activity; drinking water

PARAMETER	Reasonable Maximum Exposure (RME) Values		Notes/References
Characteristics of Population			
Age	6 to 30	years	
Bodyweight (BW)	70	kg	USEPA, 1989b; EPA 1991
Average Lifetime	70	years	USEPA, 1989b
Total Skin Area	15,634	cm^2	USEPA, 1989b
Exposure Duration			
Exposure Frequency (EF): total, hands, head	350	days/year	EPA 1997
Exposure Frequency (EF): forearms, lower legs, feet	233	days/year	EPA 1997; 8 month exposure
Exposure Duration (ED)	24	years	EPA 1991; Chronic exposure
Averaging Time (non-cancer) (AT)	8,760	days	Exposure duration x 365 days/year
Averaging Time (cancer) (AT)	25,550	days	Lifetime x 365 days/year
Dermal Contact with Soil			
Soil to Skin Adherence Factor (AF)			
Hands	0.11	mg/cm^2	Kissel et al. 1996 (Soccer No. 1-3) 95% UCL
Arms	0.011	mg/cm^2	Kissel et al. 1996
Legs	0.054	mg/cm^2	Kissel et al. 1996
Faces	0.019	mg/cm^2	Kissel et al. 1996;95% UCL for head
Feet	0.11	mg/cm^2	Kissel et al. 1996; 95% UCL for hands
Skin Surface Area Available for Contact (SA)			
Hands	795	cm^2	USEPA, 1989b
Forearms	2197	cm^2	USEPA, 1989b
Lower Legs	3182	cm^2	USEPA, 1989b; legs
Head	1252	cm^2	USEPA, 1989b
Feet	1091	cm^2	USEPA, 1989b
Incidental Ingestion of Soil			
Ingestion Rate (IR)	100	mg/day	USEPA, 1991
Fraction Ingested (FI)	1	unitless	Assumed
Ingestion of Groundwater			
Volume Ingested	2	liters/day	USEPA, 1989b
Inhalation of Dust/Volatiles			
Q/C Factor	71.87	g/m2-s per kg/m3	EPA 1996 (Table 3); Harrisburg - 1 Acre

[1]RME exposure assumptions are used for both central tendency and reasonable maximum exposure calculations in agreement with EPA 1996.

TABLE 15
RATIONALE FOR SELECTION OF GROUNDWATER WELLS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

EXCLUDED WELLS	RATIONALE FOR EXCLUSION
MW-1S, MW-1D	Situated upgradient of site

INCLUDED WELLS	COMMENT
MW-3S, MW-3D, MW-3D2, MW-4, MW-9S2	Used for evaluation of a hypothetical on-site resident situtated on the MGP site
MW-2, MW-2R2, MW-5, MW-52	Used for evaluation of a hypothetical off-site resident situated downgradient of the site, south of former MGP operations and adjacent to the Susquehanna Riverbank.
MW-6S, MW-6S2, MW-6D, MW-6D2	Contain some of the highest groundwater contaminant levels; situated downgradient of the site along a fracture zone, south of the Lancaster Water Authority Pumping Station and adjacent to the Susquehanna Riverbank. MW-6S and MW-6D represent a shallow/deep well pair.
MW-7S, MW-7S2, MW-7D, MW-7D2	Less contaminated than MW-6S and MW-6D; situated downgradient of the site, south of former MGP operations and adjacent to the Susquehanna Riverband. MW-7S and MW-7D represent a shallow/deep well pair.
CWW-1, CWW-2, CWW1-2, CWW2-2, MW-8S, MW-8S2, MW-8D, MW-8D2	We cannot conclusively determine that the contaminants in these wells are not site-related, therefore, we include them in our off-site assessment to be conservative.

TABLE 16A
TRESPASSER SCENARIO
EXPOSURE POINT CONCENTRATIONS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SURFACE SOIL		FUGITIVE DUST		SEDIMENT	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthylene	[b]	[b]	[b]	[b]	[b]	[b]
Benzo(ghi)perylene	[b]	[b]	[b]	[b]	[b]	[b]
Carcinogenic PAHs [c]						
Benzo(a)anthracene	2.0E+00	4.6E+00	NC	NC	0.96	1
Benzo(a)pyrene	2.7E+00	6.2E+00	2.6E-09	6.0E-09	0.71	0.74
Benzo(b)fluoranthene	1.9E+00	6.7E+00	NC	NC	1.2	1.3
Dibenzo(ah)anthracene	1.1E-01	NC	NC	NC	NC	NC
Indeno(123-cd)pyrene	2.0E+00	5.9E+00	NC	NC	NC	NC
Inorganic Compounds						
Aluminum	5.5E+03	8.0E+03	5.3E-06	7.6E-06	8.09E+03	9.20E+03
Arsenic	5.9E+00	8.9E+00	5.7E-09	8.6E-09	1.33E+01	1.61E+01
Beryllium	2.3E-01	3.8E-01	2.2E-10	3.6E-10	NC	NC
Cadmium	1.2E+00	3.4E+00	1.2E-09	3.3E-09	NC	NC
Copper	NC	NC	NC	NC	2.93E+02	3.91E+02
Iron	1.8E+04	3.1E+04	1.7E-05	3.0E-05	2.61E+04	2.72E+04
Lead	1.3E+02	6.3E+02	1.2E-07	6.1E-07	NC	NC
Manganese	4.3E+02	1.0E+03	4.1E-07	9.9E-07	9.93E+02	1.30E+03

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit.

[b] Evaluated qualitatively

[c] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE 16B
INDUSTRIAL WORKER SCENARIO
EXPOSURE POINT CONCENTRATIONS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SURFACE SOIL		FUGITIVE DUST		VAPOR	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthylene	[b]	[b]	[b]	[b]	[b]	[b]
Benzo(ghi)perylene	[b]	[b]	[b]	[b]	[b]	[b]
Carcinogenic PAHs [c]						
Benzo(a)anthracene	2.4E+00	4.6E+00	NC	NC	NC	NC
Benzo(a)pyrene	3.2E+00	6.2E+00	3.1E-09	6.0E-09	4.2E-07	1.4E-06
Benzo(b)fluoranthene	2.5E+00	6.7E+00	NC	NC	NC	NC
Benzo(k)fluoranthene	NC	NC	NC	NC	NC	NC
Dibenzo(ah)anthracene	1.3E-01	NC	NC	NC	NC	NC
Indeno(123-cd)pyrene	2.5E+00	5.9E+00	NC	NC	NC	NC
Inorganic Compounds						
Aluminum	5.2E+03	7.9E+03	5.0E-06	7.6E-06	NC	NC
Arsenic	5.6E+00	1.0E+01	5.3E-09	9.9E-09	NC	NC
Beryllium	2.7E-01	5.4E-01	2.5E-10	5.2E-10	NC	NC
Cadmium	1.8E+00	5.8E+00	1.7E-09	5.6E-09	NC	NC
Iron	1.9E+04	4.2E+04	1.8E-05	4.0E-05	NC	NC
Lead	1.4E+02	6.3E+02	1.4E-07	6.1E-07	NC	NC
Manganese	3.3E+02	8.1E+02	3.2E-07	7.7E-07	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit.

[b] Evaluated qualitatively

[c] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE16C
ON-SITE CONSTRUCTION WORKER SCENARIO
EXPOSURE POINT CONCENTRATIONS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SURFACE AND SUBSURFACE SOIL		FUGITIVE DUST		VAPOR	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthylene	[b]	[b]	[b]	[b]	[b]	[b]
Benzo(ghi)perylene	[b]	[b]	[b]	[b]	[b]	[b]
Carcinogenic PAHs [c]						
Benzo(a)anthracene	9.2E+00	4.7E+01	NC	NC	NC	NC
Benzo(a)pyrene	7.2E+00	3.5E+01	3.1E-09	6.0E-09	4.1E-07	1.4E-06
Benzo(b)fluoranthene	7.0E+00	3.9E+01	NC	NC	NC	NC
Benzo(k)fluoranthene	2.2E+00	1.8E+01	NC	NC	NC	NC
Dibenzo(ah)anthracene	1.1E-01	1.4E-01	NC	NC	NC	NC
Indeno(123-cd)pyrene	3.0E+00	1.5E+01	NC	NC	NC	NC
Inorganic Compounds						
Aluminum	9.4E+03	1.4E+04	5.0E-06	7.6E-06	NC	NC
Arsenic	5.7E+00	1.1E+01	5.3E-09	9.9E-09	NC	NC
Beryllium	5.3E-01	8.1E-01	2.5E-10	5.2E-10	NC	NC
Cadmium	1.8E+00	5.8E+00	1.7E-09	5.6E-09	NC	NC
Iron	2.2E+04	3.3E+04	1.8E-05	4.0E-05	NC	NC
Lead	7.5E+01	6.3E+02	1.4E-07	6.1E-07	NC	NC
Manganese	4.8E+02	9.4E+02	3.2E-07	7.7E-07	NC	NC
Thallium	1.2E+00	3.0E+00	NC	NC	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit.

[b] Evaluated qualitatively

[c] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE16D
OFF-SITE CONSTRUCTION WORKER SCENARIO
EXPOSURE POINT CONCENTRATIONS
NEAR THE SUSQUEHANNA RIVER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUBSURFACE SOIL		VAPOR	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)
Semivolatile Organic Compounds				
Non-Carcinogenic PAHs				
Acenaphthylene	[b]	[b]	[b]	[b]
Benzo(ghi)perylene	[b]	[b]	[b]	[b]
Carcinogenic PAHs [c]				
Benzo(a)anthracene	5.9E+00	1.1E+01	NC	NC
Benzo(a)pyrene	5.6E+00	9.3E+00	2.3E-07	3.7E-07
Benzo(b)fluoranthene	4.1E+00	7.6E+00	NC	NC
Benzo(k)fluoranthene	2.4E+00	6.6E+00	NC	NC
Dibenzo(ah)anthracene	1.9E-01	3.6E-01	NC	NC
Indeno(123-cd)pyrene	3.2E+00	6.0E+00	NC	NC
Inorganic Compounds				
Aluminum	1.4E+04	1.6E+04	NC	NC
Arsenic	6.8E+00	6.9E+00	NC	NC
Beryllium	1.2E+00	1.6E+00	NC	NC
Iron	2.4E+04	2.6E+04	NC	NC
Manganese	6.1E+02	8.5E+02	NC	NC
Thallium	8.8E-01	NC	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit.

[b] Evaluated qualitatively

[c] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE16E
OFF-SITE CONSTRUCTION WORKER SCENARIO
EXPOSURE POINT CONCENTRATIONS BETWEEN FRONT ST. AND THE RAILROAD TRACKS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUBSURFACE SOIL		VAPOR	
COMPOUND	**Average Concentration [a] (mg/kg)**	**Reasonable Maximum Concentration (mg/kg)**	**Average Concentration [a] (mg/m^3)**	**Reasonable Maximum Concentration (mg/m^3)**
Semivolatile Organic Compounds				
Non-Carcinogenic PAHs				
Acenaphthylene	[b]	[b]	[b]	[b]
Benzo(ghi)perylene	[b]	[b]	[b]	[b]
Carcinogenic PAHs [c]				
Benzo(a)anthracene	1.3E+01	4.7E+01	NC	NC
Benzo(a)pyrene	6.9E+00	2.4E+01	2.8E-07	9.7E-07
Benzo(b)fluoranthene	6.0E+00	2.1E+01	NC	NC
Benzo(k)fluoranthene	1.1E+00	3.2E+00	NC	NC
Dibenzo(ah)anthracene	2.2E+00	8.4E+00	NC	NC
Indeno(123-cd)pyrene	1.1E+00	3.1E+00	NC	NC
Inorganic Compounds				
Aluminum	6.6E+03	1.6E+04	NC	NC
Arsenic	6.9E+00	7.0E+00	NC	NC
Beryllium	8.3E-01	1.6E+00	NC	NC
Iron	1.9E+04	2.6E+04	NC	NC
Manganese	5.0E+02	8.5E+02	NC	NC
Thallium	8.0E-01	NC	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit.

[b] Evaluated qualitatively

[c] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE16F
HYPOTHETICAL OFF-SITE RESIDENT SCENARIO
EXPOSURE POINT CONCENTRATIONS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUBSURFACE SOIL		VAPOR		GROUNDWATER	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/L)	Reasonable Maximum Concentration (mg/L)
Volatile Organic Compounds						
Benzene	NC	NC	NC	NC	3.1E+00	3.9E+01
Ethylbenzene	NC	NC	NC	NC	3.7E-01	2.0E+00
Tetrachloroethene	NC	NC	NC	NC	NA	NA
Toluene	NC	NC	NC	NC	9.0E-01	2.6E+00
Trichloroethene	NC	NC	NC	NC	NA	NA
1,2,4-Trimethylbenzene	NC	NC	NC	NC	5.7E-02	1.1E-01
Xylenes (Mixed)	NC	NC	NC	NC	3.1E-01	6.0E-01
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthene	NC	NC	NC	NC	4.8E-02	1.5E-01
Acenaphthylene	[c]	[c]	[c]	[c]	[c]	[c]
Benzo(ghi)perylene	[c]	[c]	[c]	[c]	[c]	[c]
Fluoranthene	NC	NC	NC	NC	NA	NA
1-Methylnaphthalene	NC	NC	NC	NC	1.1E-01	3.5E-01
2-Methylnaphthalene	NC	NC	NC	NC	1.1E-01	3.9E-01
Naphthalene	NC	NC	NC	NC	8.0E-01	6.3E+00
Phenanthrene	NC	NC	NC	NC	3.0E-02	4.2E-02
Pyrene	NC	NC	NC	NC	NA	NA
Carcinogenic PAHs [d]						
Benzo(a)anthracene	5.9E+00	1.1E+01	NC	NC	ND	ND
Benzo(a)pyrene	5.6E+00	9.3E+00	2.2E-07	3.7E-07	ND	ND
Benzo(b)fluoranthene	4.1E+00	7.6E+00	NC	NC	ND	ND
Benzo(k)fluoranthene	2.4E+00	6.6E+00	NC	NC	NA	NA
Chrysene	NC	NC	NC	NC	ND	ND
Dibenzo(ah)anthracene	1.9E-01	3.6E-01	NC	NC	NA	NA
Indeno(123-cd)pyrene	3.2E+00	6.0E+00	NC	NC	NA	NA
Other SVOCs						
Dibenzofuran	NC	NC	NC	NC	1.2E-02	1.4E-02
Inorganic Compounds						
Aluminum	1.4E+04	1.6E+04	NC	NC	4.2E-02	1.0E-01
Arsenic	6.8E+00	6.9E+00	NC	NC	NC	NC
Barium	NC	NC	NC	NC	9.0E-02	1.2E-01
Beryllium	1.2E+00	1.6E+00	NC	NC	NC	NC
Cyanide [b]	NC	NC	NC	NC	6.2E-02	2.2E-01
Iron	2.4E+04	2.6E+04	NC	NC	3.0E+00	2.5E+01
Lead	NC	NC	NC	NC	1.2E-03	1.5E-03
Manganese	6.1E+02	8.5E+02	NC	NC	6.9E-01	3.0E+00
Thallium	8.8E-01	1.1E+00	NC	NC	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit

[b] Free cyanide concentrations are estimated to be 15% of the measured total cyanide concentration

[c] Evaluated qualitatively

[d] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

TABLE 17
COCs SEGREGATED BY TARGET ORGAN EFFECT
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	TARGET ORGAN								
	Systemic					GI	Central Nervous System	Cardiovascular	Reproductive
	Systemic	Kidney	Liver	Hematological	None				
Volatile Organic Compounds									
Benzene	✓			✓					
Ethylbenzene		✓	✓						
Tetrachloroethene			✓						
Trichloroethene[1]									
Toluene		✓	✓						
1,2,4-Trimethylbenzene		✓		✓					
Xylenes (total)	✓								✓
Semi-Volatile Organic									
Polycyclic Aromatic Hydrocarbons									
Acenaphthene			✓						
Acenaphthylene[3]									
Benzo(a)anthracene[1]									
Benzo(b)fluoranthene[1]									
Benzo(k)fluoranthene[1]									
Benzo(g,h,i)perylene[3]									
Benzo(a)pyrene[1]									
Chrysene[1]									
Dibenzo(a,h)anthracene[1]									
Fluoranthene		✓	✓	✓					
Indeno(1,2,3-cd)pyrene[1]									
1-Methylnaphthalene		✓	✓	✓					
2-Methylnaphthalene		✓	✓	✓					
Naphthalene		✓	✓	✓					
Phenanthrene		✓	✓	✓					
Pyrene		✓							
Other SVOCs									
Bis(2-ethylhexyl)phthalate			✓						✓
Dibenzofuran			✓						
Inorganic Compounds									
Aluminum					✓				
Arsenic	✓								
Barium					✓			✓	
Beryllium					✓				
Cadmium		✓							
Cyanide	✓								
Iron					✓				
Lead[2]									
Manganese							✓		
Thallium					✓				

[1] Evaluated for carcinogenic risks only

[2] Risks associated with lead are evaluated separately

[3] Evaluated qualitatively

ORIGINAL (Red)

TABLE 18
SUMMARY OF RISK ESTIMATES
UGI Columbia Former MGP Site
Columbia, Pennsylvania

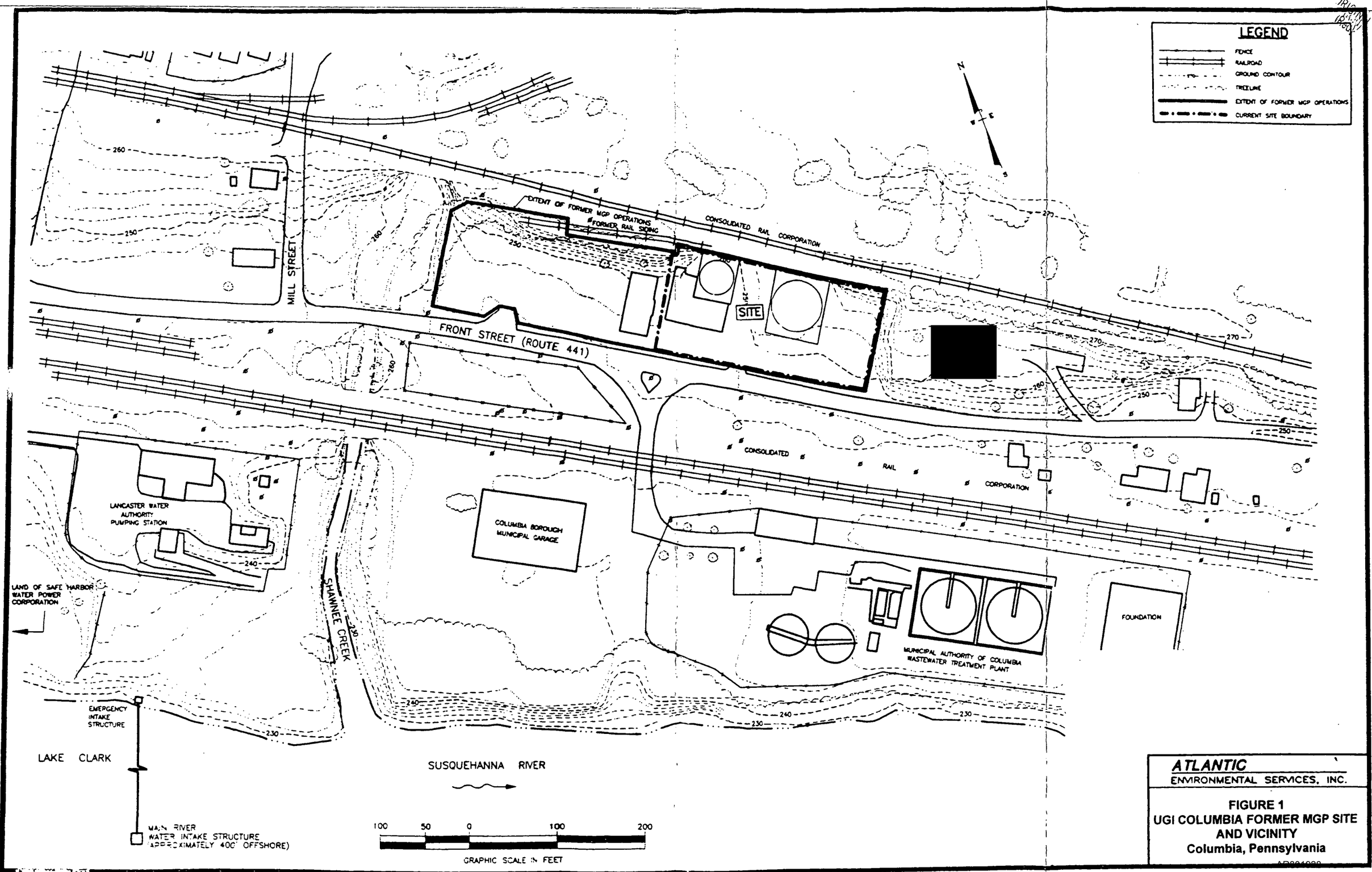
Scenario	Subchronic Total Hazard Index	Chronic Total Hazard Index	Cancer Total Risk Estimates
Current Land Use			
Trespasser			
Central Tendency	NC	5.9E-02	9.6E-07
RME	NC	7.7E-02	1.5E-06
Future Land Use			
Industrial Worker			
Central Tendency	NC	6.4E-02	7.9E-06
RME	NC	1.4E-01	1.5E-05
On-site Construction Worker			
Central Tendency	6.9E-01	NC	5.4E-06
RME	1.2E+00	NC	2.4E-05
Off-site Construction Worker (near river)			
Central Tendency	7.6E-01	NC	4.9E-06
RME	8.1E-01	NC	7.6E-06
Off-site Construction Worker (Front Street)			
Central Tendency	6.1E-01	NC	6.6E-06
RME	8.1E-01	NC	2.1E-05
Hypothetical Off-Site Resident *(soil and drinking water exposure)*			
Child (Central Tendency)	NC	7.8E+01	6.1E-04
Teen/Adult (Central Tendency)	NC	3.1E+01	8.8E-04
Child (RME)	NC	9.2E+02	6.7E-03
Teen/Adult (RME)	NC	3.7E+02	1.1E-02
Risk Criteria	**1**	**1**	**1×10^{-6} to 1×10^{-4}**

NC = Not Calculated

TABLE 19
SUMMARY OF TARGET ORGAN RISK ESTIMATES - HYPOTHETICAL RESIDENT
CHRONIC EXPOSURE
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Scenario	Total Chronic Systemic Effects Hazard Index	Total Chronic Central Nervous System Hazard Index	Total Chronic Cardiovascular System Hazard Index	Total Chronic Reproductive System Hazard Index
Hypothetical On-Site Resident				
Child - CTE	2.3E+02	1.5E+00	1.7E-01	3.0E-01
Child - RME	5.7E+02	4.7E+00	1.8E-01	3.6E-01
Adult - CTE	9.2E+01	5.4E-01	6.7E-02	1.2E-01
Adult - RME	2.3E+02	1.8E+00	7.0E-02	1.5E-01
Hypothetical Off-Site Resident				
Child - CTE	7.6E+01	2.1E+00	8.8E-02	1.1E-02
Child - RME	9.1E+02	9.5E+00	1.2E-01	2.1E-02
Adult - CTE	3.0E+01	8.8E-01	3.5E-02	4.2E-03
Adult - RME	3.6E+02	3.7E+00	4.7E-02	8.2E-03
Risk Criteria	**1**	**1**	**1**	**1**

FIGURES



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ENVIRONMENTAL SERVICES, INC.

FIGURE 1
UGI COLUMBIA FORMER MGP SITE AND VICINITY
Columbia, Pennsylvania

COLUMBIA BOROUGH

OFFICIAL ZONING MAP

THIS IS TO CERTIFY THAT THIS IS THE OFFICIAL ZONING MAP OF COLUMBIA BOROUGH, LANCASTER COUNTY, PENNSYLVANIA ADOPTED THIS 13th DAY, OF FEBRUARY, 1982.

ATTEST: _____ MAYOR

_____ SECRETARY

REVISIONS SUBSEQUENT TO 13th DAY OF FEBRUARY, 1990

DATE	ORDINANCE	ATTEST	
5-21-82	NOS 443 thru 532 incl		MAYOR
			SECRETARY
			MAYOR
			SECRETARY
			MAYOR
			SECRETARY
			MAYOR
			SECRETARY
			MAYOR
			SECRETARY

—LEGEND—

- R-R — RURAL/RESIDENTIAL DISTRICT
- R-1 — RESIDENTIAL DISTRICT
- R-2 — RESIDENTIAL DISTRICT
- R-3 — RESIDENTIAL DISTRICT
- R-4 — RESIDENTIAL DISTRICT
- I — INDUSTRIAL DISTRICT
- C-1 — COMMERCIAL DISTRICT
- C-2 — COMMERCIAL DISTRICT
- F — FLOOD PLAIN DISTRICT (Includes R-R Overlay)

The preparation of this map was financed in part through a urban planning grant from the Housing and Home Finance Agency, under the provisions of Section 701 of the Housing Act of 1954, as amended, administered by the Bureau of Community Development, Pennsylvania Department of Commerce.

FIGURE 2
COLUMBIA ZONING MAP

Prepared by the
LANCASTER COUNTY PLANNING COMMISSION
for the
COLUMBIA BOROUGH PLANNING COMMISSION

SCALE - FEET
400 0 400 800 1200 1600

N

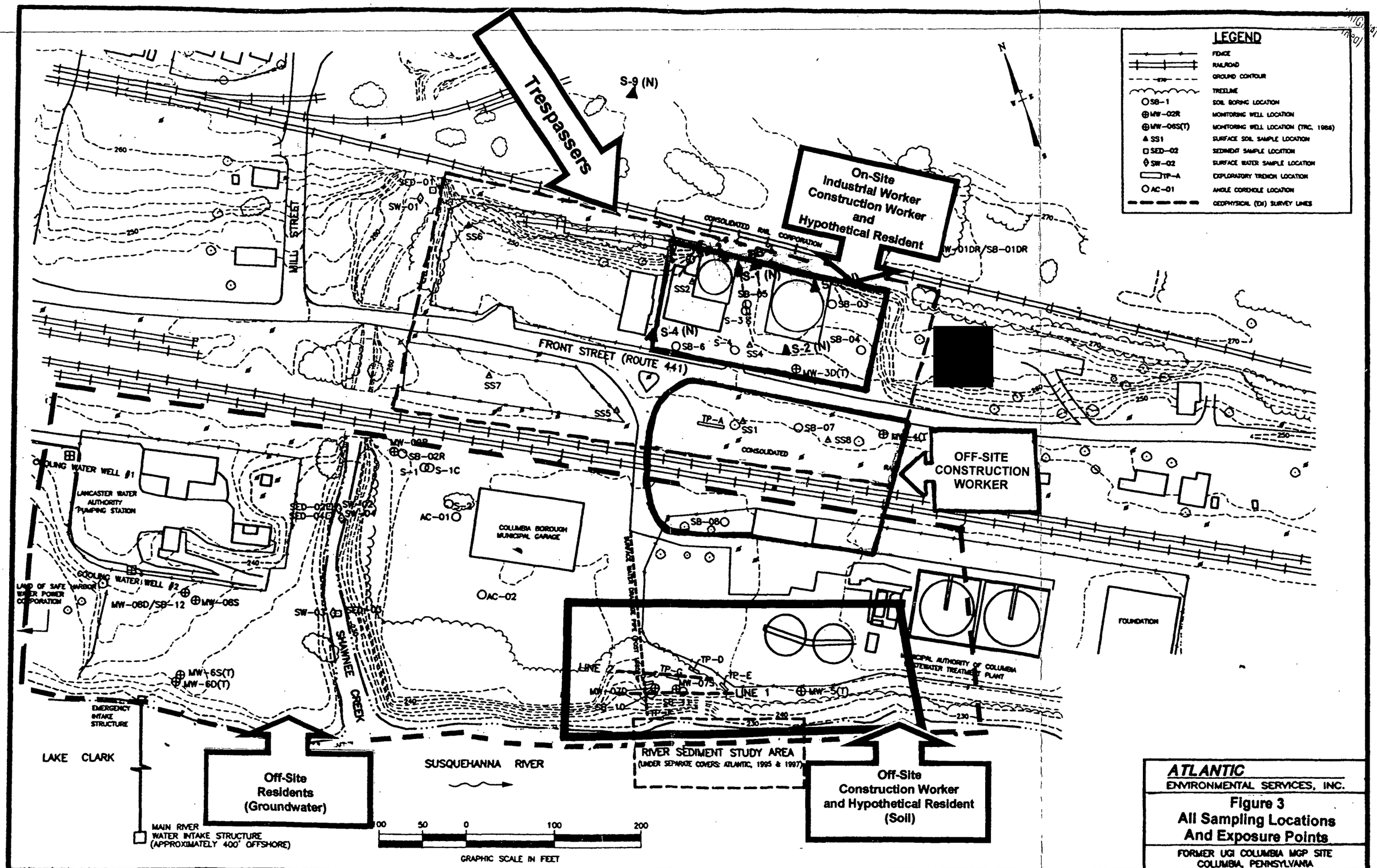

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Figure 3
All Sampling Locations And Exposure Points

FORMER UGI COLUMBIA MGP SITE
COLUMBIA, PENNSYLVANIA

AR301930

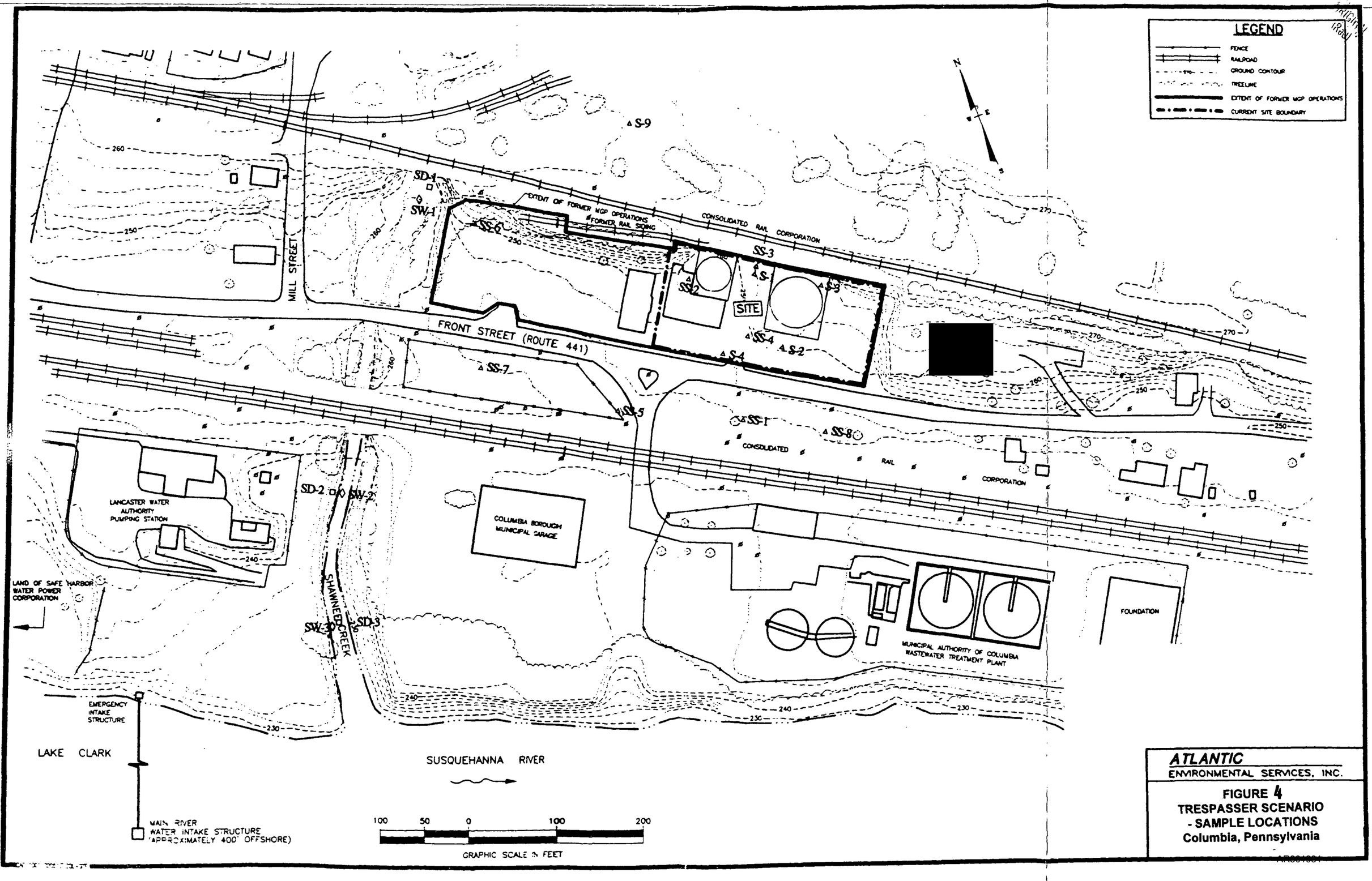

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FIGURE 4
TRESPASSER SCENARIO
- SAMPLE LOCATIONS
Columbia, Pennsylvania

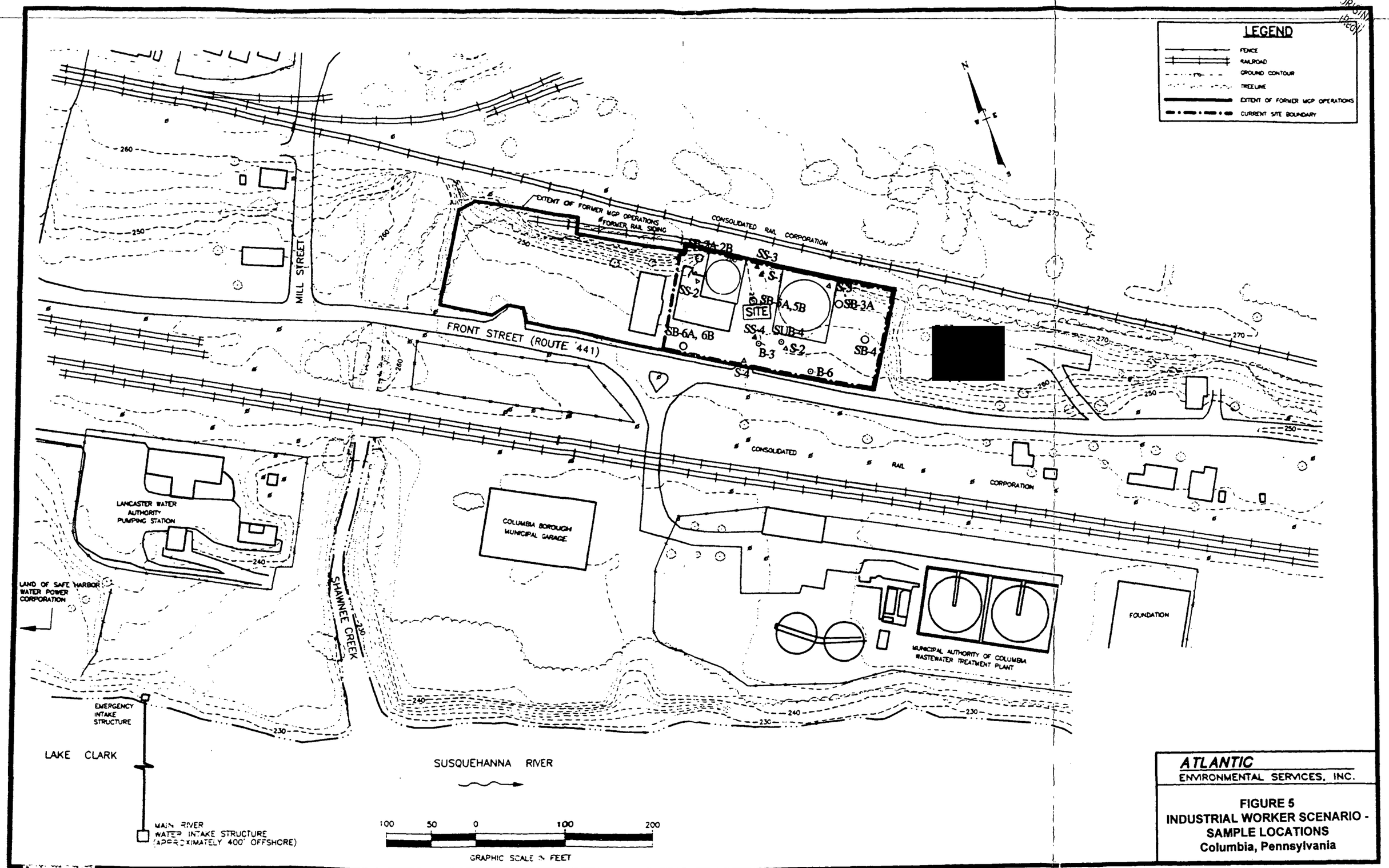

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FIGURE 5
INDUSTRIAL WORKER SCENARIO - SAMPLE LOCATIONS
Columbia, Pennsylvania

AR301932

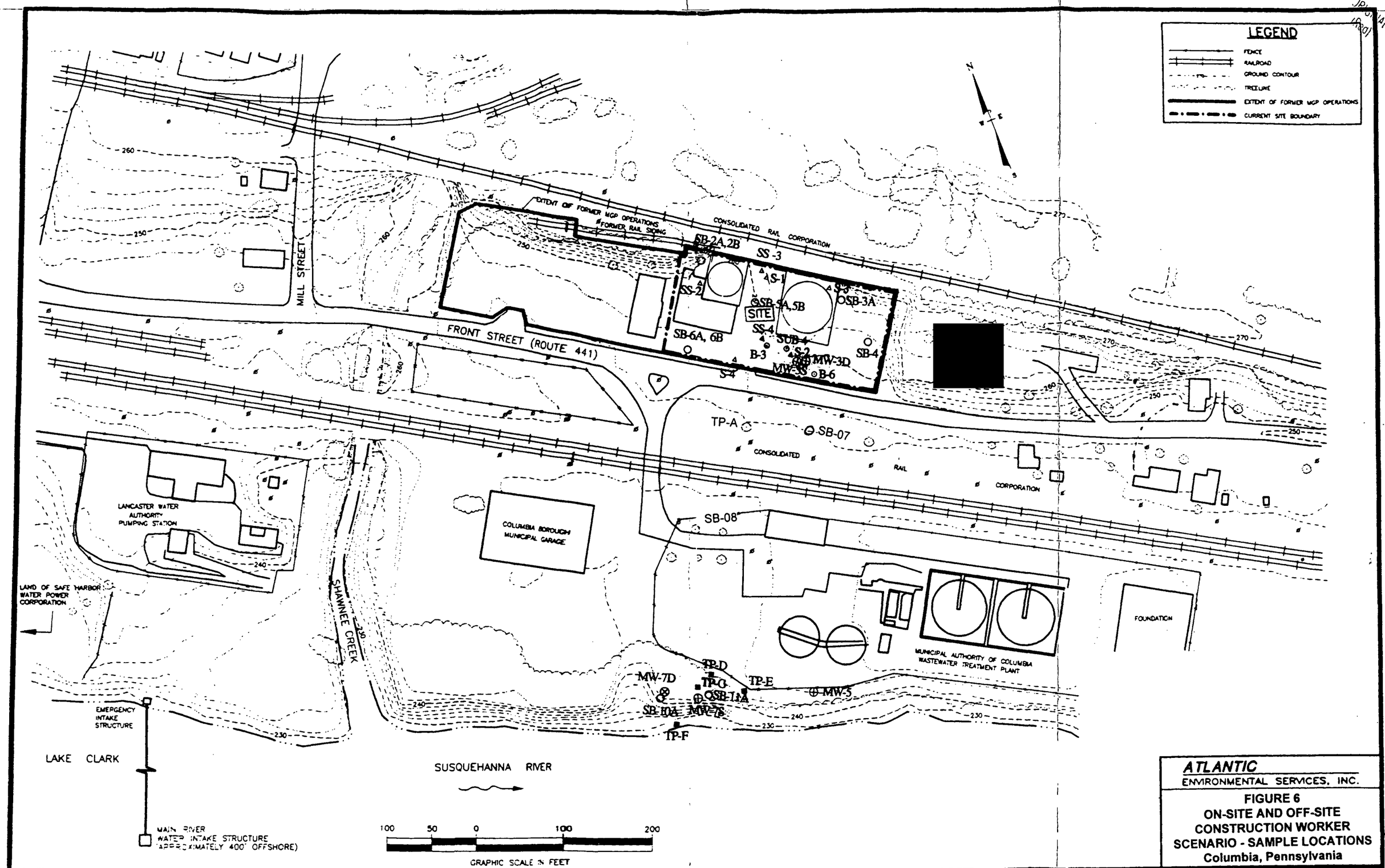

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FIGURE 6
ON-SITE AND OFF-SITE CONSTRUCTION WORKER SCENARIO - SAMPLE LOCATIONS
Columbia, Pennsylvania

AR301955

Legend

S – ▲ Previous Surface Soil Samples (NUS)
SS – ● Surface Soil Locations (Atlantic)
SUB – ⊙ Previous Sub-Soil Samples (NUS)
SB – ⊕ Soil Boring Locations
MW – △ Monitoring Wells

LEGEND

FENCE
RAILROAD
GROUND CONTOUR
TREELINE
EXTENT OF FORMER MGP OPERATIONS
CURRENT SITE BOUNDARY

EXTENT OF FORMER MGP OPERATIONS
FORMER RAIL SIDING
CONSOLIDATED RAIL CORPORATION
MILL STREET
FRONT STREET (ROUTE 441)
SITE
SB-1 (N)
SS-3 (A)
SS-2 (A)
SB-5A,-5B (A)
SB-3A (A)
SB-6A (A)
SB-6B (A)
S-4
SUB-4 (N)
SS-4 (A)
S-2 (N)
SB-4 (A)
MW-3D (A)
MW-3S (A)
SB-14A (A)
MW-4 (A)
CONSOLIDATED RAIL CORPORATION
MW-2R
CWW-1
LANCASTER WATER AUTHORITY PUMPING STATION
COLUMBIA BOROUGH MUNICIPAL GARAGE
FOUNDATION
LAND OF SAFE HARBOR WATER POWER CORPORATION
CWW-2
MW-8D
MW-8S
SHAWNEE CREEK
MW-6S
MW-6D
MW-7D
TP-G
TP-D
SB-11A
TP-E
SB-10AC
MW-7S
MW-5
TP-F
MUNICIPAL AUTHORITY OF COLUMBIA WASTEWATER TREATMENT PLANT
EMERGENCY INTAKE STRUCTURE
LAKE CLARK
SUSQUEHANNA RIVER
MAIN RIVER WATER INTAKE STRUCTURE (APPROXIMATELY 400' OFFSHORE)
260
250
240
230
270
100 50 0 100 200
GRAPHIC SCALE IN FEET

ATLANTIC
ENVIRONMENTAL SERVICES, INC.

FIGURE 7
HYPOTHETICAL RESIDENT SCENARIO - SAMPLE LOCATIONS
Columbia, Pennsylvania

AR301934

APPENDIX A

ANALYTICAL DATA

APPENDIX A-1
ANALYTICAL DATA FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Sample Location:	SS-1		SS-2		SS-3		SS-4		SS-5		SS-6	
Date:	Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996	
Units:	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Volatile Organic Compounds												
Acetone	NA		NA		NA		NA		NA		NA	
Benzene	0.1	J	0.79	J	0.09	J	0.13	J	0.15	J	0.17	U
Ethylbenzene	0.15	U	0.25		0.13	U	0.08	J	0.14	J	0.17	U
Toluene	0.1	J	1.3		0.07	J	0.29		0.21	J	0.13	J
1,2,4-Trimethylbenzene	0.15	U	1.4		0.13	U	0.41		0.5		0.17	U
Xylenes (total)	0.47		3.28		0.16	J	0.79		0.69		0.34	U
Semivolatile Organic Compounds												
Acenaphthene	0.15	U	0.17	J	0.13	U	0.13	UJ	0.26		0.17	U
Acenaphthylene	0.3		1.6		0.19		1.9		1		0.08	J
Anthracene	0.2		0.84		0.13	U	0.81		0.74		0.17	U
Benzo(a)anthracene	0.53		3.4		0.2		2.5		3.7		0.17	U
Benzo(a)pyrene	1.1		5		0.47		3.1		4.9		0.1	J
Benzo(b)fluoranthene	0.47		3		0.13	U	1.6		3.2		0.17	U
Benzo(g,h,i)perylene	2.1	J	4.3	J	0.2	J	2.8	J	3.8	J	0.23	J
Benzo(k)fluoranthene	0.62		6.1		0.36		2		3.9		0.17	U
Chrysene	0.71	J	3.9	J	0.2	J	2.3	J	4.3	J	0.17	U
Dibenz(a,h)anthracene	0.15	UJ	0.17	UJ	0.13	U	0.13	UJ	0.21	U	0.17	UJ
Dibenzofuran	0.15	UJ	0.15	J	0.13	U	0.1	J	0.38	J	0.17	UJ
Fluoranthene	1		3.7		0.19		1.6		6.5		0.17	U
Fluorene	0.15	U	0.17	U	0.13	U	0.06	J	0.21	U	0.17	U
Indeno(1,2,3-cd)pyrene	0.7	J	4.1	J	0.17	J	2.5	J	3.5	J	0.17	UJ
1-Methylnaphthalene	0.12	J	0.76		0.13	U	0.38		0.5		0.17	U
2-Methylnaphthalene	0.09	J	0.93		0.13	U	0.81		0.74		0.17	U
Naphthalene	0.17		1.7		0.06	J	1.2		0.84		0.17	U
Phenanthrene	0.5		1.6		0.07	J	0.86		2.2		0.17	U
Pyrene	1.3		6		0.45		3.1		9.1		0.14	J

APPENDIX A-1
ANALYTICAL DATA FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Sample Location:	SS-1		SS-2		SS-3		SS-4		SS-5		SS-6	
Date:	Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996		Atlantic, 1996	
Units:	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Bis(2-ethylhexyl)phthalate	NA		NA		NA		NA		NA		NA	
Di-n-octyl phthalate	NA		NA		NA		NA		NA		NA	
Inorganic Compounds												
Aluminum	2160		4840		2980		6410		5730		5590	
Arsenic	3.5	J	10.3	J	2	J	4.1	J	13.5	J	3.8	J
Barium	17.3	B	146		12.2	B	63.8		79.5		46.5	
Beryllium	0.06	J	0.31	B	0.15	B	0.35	B	0.4	B	0.19	B
Cadmium	0.34	U	5.8		0.32	U	0.92	B	0.82	B	0.32	U
Calcium	135000		16000		224000		32200		65700		528	B
Chromium	2.2	R	32.4	J	3.7	R	7.4	UJ	14.5	J	11.3	
Cobalt	3	J	8.7	J	2.3	R	12.1	J	7.2	J	4	B
Copper	10.5	J	83.5	J	6.1	J	32.7	J	112	J	9.2	J
Iron	6360	J	25300	J	4710	J	25100	J	16700	J	13900	J
Lead	24.6		634		5		50.7		300		10.6	
Magnesium	7470	J	7730	J	36300	J	14500	J	17800	J	1280	J
Manganese	114	J	367	J	93.3	J	807	J	306	J	104	J
Mercury	0.12	U	0.19	J	0.11	U	0.11	U	0.14	U	0.11	U
Nickel	6.2	B	22		6.3	B	18.7		21.3		7.4	B
Potassium	1140		850	B	2120		2690		1520		863	B
Selenium	0.25	UJ	1	B	0.23	UJ	0.23	U	0.55	B	0.24	U
Sodium	45.8	UJ	72.2	UJ	52.3	R	71.4	UJ	108	J	39.6	U
Thallium	0.13	UJ	0.21	J	0.12	UJ	0.13	UJ	0.16	UJ	0.13	UJ
Vanadium	3.7	B	14.9		8.1	B	9.4	B	18.8		8.4	B
Zinc	34.2	J	349	J	15.4	J	210	J	379	J	31.8	J
Cyanide	2.7		25		1.1	U	1.7		0.7	U	48.2	

APPENDIX A-1
ANALYTICAL DATA FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Sample Location:	SS-7		SS-8		S-1		S-2		S-3		S-4[1]		S-9	
Date:	Atlantic, 1996		Atlantic, 1996		NUS, 1991		NUS, 1991		NUS, 1991		NUS, 1991		NUS, 1991	
Units:	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Volatile Organic Compounds														
Acetone	NA		NA		0.01	U	0.056		0.01	U	0.01	U	0.028	
Benzene	0.14	U	0.13	J	0.005	U	0.005	U	0.005	U	0.005	U	0.005	U
Ethylbenzene	0.14	U	0.15	U	0.005	U	0.005	U	0.005	U	0.005	U	0.005	U
Toluene	0.13	J	0.19		0.005	U	0.005	U	0.005	U	0.005	U	0.005	U
1,2,4-Trimethylbenzene	0.68		0.59		NA		NA		NA		NA		NA	
Xylenes (total)	0.6		1.02		0.005	U	0.005	U	0.005	U	0.005	U	0.005	U
Semivolatile Organic Compounds														
Acenaphthene	0.18		0.17		0.33	U	0.33	U	0.33	U	0.24	J	0.33	U
Acenaphthylene	0.43		0.94		0.860		3.800		0.330	U	2.3		0.330	U
Anthracene	0.35		0.47		0.25	J	1.3		0.33	U	1.2	J	0.33	U
Benzo(a)anthracene	1.1		1.7		1.4		4.6		0.33	U	4.3		0.36	J
Benzo(a)pyrene	1		2		1.6		6.2		0.33	U	6.2		0.37	
Benzo(b)fluoranthene	0.76		1.2		1.1		4.9		0.33	U	6.7		0.39	J
Benzo(g,h,i)perylene	0.73	J	1.7	J	1.2		5.6		0.33	U	9.0		0.29	J
Benzo(k)fluoranthene	0.65		1.7		1.0		3.2		0.33	U	3.9		0.37	J
Chrysene	2.1	J	2.1	J	1.5		5.3		0.33	U	5.5		0.5	J
Dibenz(a,h)anthracene	0.14	UJ	0.15	UJ	0.33	U	0.33	U	0.33	U	0.33	U	0.11	J
Dibenzofuran	0.14	UJ	0.09	J	0.33	U	0.33	U	0.33	U	0.33	U	0.33	U
Fluoranthene	1		2.3		1.5		4.4		0.33	U	6.6		0.64	J
Fluorene	0.49		0.16		0.33	U	0.28	J	0.33	U	0.47	J	0.33	U
Indeno(1,2,3-cd)pyrene	0.65	J	1.7	J	0.84		4.0		0.33	U	5.9		0.26	J
1-Methylnaphthalene	0.66		0.29		NA		NA		NA		NA		NA	
2-Methylnaphthalene	0.54		0.29		0.26	J	0.93	J	0.33	U	1.0	J	0.33	U
Naphthalene	1.2		0.39		0.31	J	1.3	J	0.33	U	1.2	J	0.33	U
Phenanthrene	1.4		0.98		0.67	J	2.7		0.33	U	3.4		0.31	J
Pyrene	1.8		4		3.3		11		0.33	U	12		0.69	J

APPENDIX A-1
ANALYTICAL DATA FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Sample Location:	SS-7		SS-8		S-1		S-2		S-3		S-4[1]		S-9	
Date:	Atlantic, 1996		Atlantic, 1996		NUS, 1991		NUS, 1991		NUS, 1991		NUS, 1991		NUS, 1991	
Units:	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Bis(2-ethylhexyl)phthalate	NA		NA		0.11		0.25		0.33	U	2.3	B	0.19	
Di-n-octyl phthalate	NA		NA		0.33	U	0.33	U	0.76	J	0.33	U	0.33	U
Inorganic Compounds														
Aluminum	5570		10700		2060		7380		4770		7920		11200	
Arsenic	4.5	J	6.4	J	2.4		5.9		4.9		9.4		12.9	
Barium	221		71.2		15.6	J	81.5		47.7		91.8		236	
Beryllium	0.48	B	0.59	B	0.37	J	0.3	J	0.24	J	0.54	J	0.57	J
Cadmium	0.67	B	0.39	U	1.0	U	1.0	U	1.0	U	4.7		5.2	
Calcium	4070		7150		257000		12000	J	5280	J	13100	J	4620	J
Chromium	8.4	UJ	16	J	1.9	J	5.9		2.5		19.2		15.8	
Cobalt	21.6	J	12.5	J	1.5	J	8	J	6.9	J	9.4	J	7.9	J
Copper	73	J	63.9	J	7.2		26.7		7.2		61.6		275	
Iron	19600	J	28600	J	5690		15700		11300		41800		24300	
Lead	104		76.5		23.9		85.9		45.1		143		387	
Magnesium	2950	J	6710	J	43500		4270		3410		7470		1660	
Manganese	1700	J	617	J	95.6		264		137		571		605	
Mercury	0.11	U	0.27	J	0.2	U	0.21		0.2	U	0.35		0.44	
Nickel	15.5		23.2		5.6	J	14		14		22.5		16.8	
Potassium	554	B	1990		1330		2860		4370		3120		1360	
Selenium	0.32	B	0.44	B	1.0	U	1.0	U	1.0	U	1.0	U	0.74	J
Sodium	64	UJ	59.9	UJ	61.8	J	70.3	J	76.7	J	137	J	143	J
Thallium	0.15	J	0.26	J	2.0	U	2.0	U	2.0	U	2.0	U	2.0	U
Vanadium	15.7		22.6		4.5	J	12.6		4.6	J	24.2		23.9	
Zinc	153	J	196	J	24.4		115		18.9		410		783	
Cyanide	0.54	U	1.3		0.69		1.4		2.0	U	1.4		0.47	J

U - not detected at reported detection limit
J - estimated
B - detected value between instrument detection limit and CRDL
R - rejected value
Surface soil is defined as 0 to 6 inches deep.
Table presents compounds detected at least once in surface soil..
[1] - Higher value of duplicates (S-4 and S-8) is reported. Raw data not available.

APPENDIX A-2
ANALYTICAL DATA FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SUB-4		SB-14A[1]		SB-02B		SB-03A		SB-04A		SB-05A		SB-05B	
Date:	NUS, 1991		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994	
Depth (feet):	1 - 4		1.3		13		4.8		4.4		3.9		8.3	
	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Volatile Organic Compounds														
Benzene	0.094		1.4	J	0.16	U	0.21	U	0.15	U	0.7	J	1.49	J
Ethylbenzene	0.012		11	J	0.16	U	0.21	U	0.15	U	9.6	J	29	J
Styrene	0.005	U	0.14	U	0.16	U	0.21	U	0.15	U	0.14	U	0.21	U
Toluene	0.022		2.9	J	0.16	U	0.21	U	0.15	U	0.76	J	0.4	J
1,2,4-Trimethylbenzene	NA		46	J	0.16	U	0.21	U	0.15	U	16	J	22	J
Xylenes, total	0.070		20.6	J	0.32	U	0.42	U	0.2	J	12.1	J	30	J
Semivolatile Organic Compounds														
Acenaphthene	3.1		5.3	J	0.08	J	0.21	U	0.15	U	51	J	51	J
Acenaphthylene	9.1		5.5	J	0.11	J	0.21	U	2.2	J	28	J	10	J
Anthracene	7.2		4.4	J	0.16	U	0.21	UJ	0.72	J	24	J	17	J
Benzo(a)anthracene	22		0.87	J	0.16	U	0.21	U	3	J	45	J	8.9	J
Benzo(a)pyrene	19		0.21	J	0.16	U	0.21	UJ	5.7	J	35	J	4.4	J
Benzo(b)fluoranthene	30		1	J	0.16	U	0.21	UJ	5.3	J	39	J	4.6	J
Benzo(g,h,i)perylene	0.33	U	0.21	J	0.16	U	0.21	UJ	2.8	J	11		1.3	J
Benzo(k)fluoranthene	0.33	U	0.14	U	0.16	U	0.21	U	0.15	C	0.14	C	0.21	C
Chrysene	25		6.4	J	0.16	U	0.21	UJ	3.6	J	52	J	8.5	J
Dibenz(a,h)anthracene	0.33	U	0.14	UJ	0.16	U	0.21	UJ	0.15	C	0.14	UJ	0.21	UJ
Dibenzofuran	1.3		18	J	0.16	U	0.21	UJ	0.15	U	0.14	UJ	13	J
Fluoranthene	34		6.4	J	0.16	U	0.21	U	0.38	J	71	J	15	J
Fluorene	11		4	J	0.16	U	0.21	U	0.15	U	35	J	19	J
Indeno(1,2,3-cd)pyrene	0.33	U	0.14	UJ	0.16	U	0.21	UJ	3.1	J	15	J	1.7	J
1-Methylnaphthalene	NA		13	J	0.15	J	0.21	U	0.15	U	75	J	92	J
2-Methylnaphthalene	30		18	J	0.16	U	0.21	U	0.15	U	19	J	130	J
Naphthalene	13		81	J	0.16	U	0.21	U	0.15	U	36	J	190	J
Phenanthrene	57		8.5	J	0.18	J	0.21	U	0.14	J	51	J	49	J
Pyrene	74		6.6	J	0.09	J	0.21	U	3.8	J	110	J	23	J

APPENDIX A-2
ANALYTICAL DATA FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SUB-4		SB-14A[1]		SB-02B		SB-03A		SB-04A		SB-05A		SB-05B	
Date:	NUS, 1991		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994	
Depth (feet):	1 - 4		1.3		13		4.8		4.4		3.9		8.3	
	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Inorganic Compounds														
Aluminum	8090		15000		19600		6900		13400		16200		21400	
Arsenic	11.8		18		2.6	U	5.6		2.9	U	5.4		1.7	U
Barium	79.7		46	B	33.9	B	45.9	B	56.6		191		53.6	
Beryllium	0.54	J	0.48	B	0.82	B	0.58	B	0.95	B	3.9		0.8	B
Calcium	10700	J	2050		4540		1010	B	2010		56000		6690	
Chromium	8.9		12.7		18.3		9.5		9.8		6.3		13.6	
Cobalt	8	J	2.8	U	18		8.2	B	22.5		11.1	B	7.7	B
Copper	45.4		20.6	J	54.7	J	13.1	J	24.7	J	25.1	J	14	J
Iron	28100		37400		26900		16800		32000		21800		27700	
Lead	140		8.6	J	6	J	15.6	J	10.7	J	15	J	3.5	J
Magnesium	3560		20900		22800		1610		16100		9180		34900	
Manganese	269		56.2		283		395		2490		960		253	
Mercury	0.11		0.06	U	0.06	U	0.06	U	0.06	U	0.06	U	0.07	U
Nickel	15.8		18.5	UJ	28.6	J	11.6	UJ	25	J	9.4	UJ	24.4	J
Potassium	1460		1480		2030		605	B	5750		2870		3620	
Selenium	0.82	J	1	U	1	U	0.92	U	0.96	U	1.2		1.1	U
Sodium	76.7	J	87.7	UJ	117	UJ	39.5	U	42.6	UJ	280	B	94	UJ
Thallium	2	U	2.4	J	3	J	2.1	U	2.7	J	2.1	U	2.4	U
Vanadium	21.6		17.2	J	19.5	J	16		15.1	J	9.3	J	21.9	J
Zinc	115		54.9		77.9		39.3		76		110		77.7	
Cyanide	3.8		45	J	1.2	UJ	0.86	B	0.94	U	1.2	U	0.33	U

APPENDIX A-2
ANALYTICAL DATA FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SB-06A		SB-06B		SB-10A		SB-11A		TP-F		TP-G		SB-7A		SB-8A		SB-8B		TP-A	
Date:	Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1995		Atlantic, 1995		Atlantic, 1995		Atlantic, 1994		Atlantic, 1995		Atlantic, 1995		Atlantic, 1995	
Depth (feet):	4.3		8.8		4.8		2.5		4		14.2		4.8		4.3		18.5		14.2	
	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Volatile Organic Compounds																				
Benzene	0.15	UJ	0.13	UJ	0.15	U	0.16	U	1.2		3.9	J	0.09	J	0.18	UJB	0.14	U	0.22	J
Ethylbenzene	0.42	J	1	J	0.15	U	0.12	J	0.21	J	1.1	J	0.15	U	0.15	U	0.14	U	11	J
Styrene	0.15	U	0.13	U	0.15	U	0.16	C	0.23	C	0.24	C	0.15	U	0.15	U	0.14	U	4.4	J
Toluene	0.19		0.09	J	0.16	J	0.16	U	0.94		22	J	0.07	J	0.15	U	0.14	U	1.4	J
1,2,4-Trimethylbenzene	2.9	J	7.5	J	0.33	J	0.16	U	0.76	J	4.4	J	0.15	U	0.15	U	0.14	U	30	J
Xylenes, total	1.19	J	1.565	J	0.66	J	0.36	J	0.8		4.7	J	0.08	J	0.13	J	0.28	U	7.5	J
Semivolatile Organic Compounds																				
Acenaphthene	85	J	20	J	0.32	J	0.3	J	18	J	76	J	0.15	U	0.15	U	0.14	U	140	J
Acenaphthylene	30	J	4.7	J	2.2	J	1.3	J	12	J	87	J	0.15	U	0.39		0.14	U	160	J
Anthracene	53	J	6.5	J	2	J	1.1	J	6.3	J	15	J	0.15	U	0.3	J	0.14	UJ	99	J
Benzo(a)anthracene	47	J	4	J	5.7	J	2.3	J	4.7	J	11	J	0.15	U	3.6	J	0.14	UJ	47	J
Benzo(a)pyrene	25	J	2.4	J	7	J	3	J	3	J	9.3	J	0.15	UJ	3.6	J	0.14	UJ	24	J
Benzo(b)fluoranthene	12	J	2.6	J	4.6	J	1.8	J	2.4	J	7.6	J	0.15	UJ	3		0.14	U	21	J
Benzo(g,h,i)perylene	6.3	J	0.86	J	4.4	J	2.3	J	1	J	4.3	J	0.15	UJ	2.6	J	0.14	UJ	7	J
Benzo(k)fluoranthene	18	J	0.13	C	6.6	J	2.4	J	0.23	C	0.24	C	0.15	U	3.2		0.14	U		C
Chrysene	48	J	3.6	J	6.8	J	2.7	J	5	J	10	J	0.15	UJ	3.2	J	0.14	UJ	44	J
Dibenz(a,h)anthracene	0.15	UJ	0.13	UJ	0.15	UJ	0.16	U	0.36	J	0.24	C	0.15	UJ	0.15	UJ	0.14	UJ	8.4	J
Dibenzofuran	0.15	UJ	0.13	UJ	0.59	J	0.16	U	3.6	J	6.5	J	0.15	UJ	0.15	UJ	0.14	UJ	15	J
Fluoranthene	92	J	11	J	9.1	J	3.6	J	6.3	J	19	J	0.15	U	4.1		0.14	U	49	J
Fluorene	59	J	12	J	1.3	J	0.79	J	10	J	49	J	0.15	U	0.15	UJ	0.14	UJ	57	J
Indeno(1,2,3-cd)pyrene	8.5	J	1.1	J	6	J	2.1	J	0.5	J	4.1	J	0.15	UJ	3.1	J	0.14	UJ		C
1-Methylnaphthalene	73	J	33	J	0.68	J	0.52	J	18	J	67	J	0.15	U	0.13	J	0.14	U	240	J
2-Methylnaphthalene	0.15	U	20	J	0.6	J	0.45	J	19	J	22	J	0.15	U	0.18		0.14	U	24	J
Naphthalene	19	J	28	J	1.1	J	0.95	J	13	J	59	J	0.15	U	0.38		0.14	U	41	J
Phenanthrene	160	J	21	J	6.4	J	3.9	J	21	J	62	J	0.15	U	0.59	J	0.14	UJ	170	J
Pyrene	150	J	15	J	9.2	J	5.5	J	11	J	29	J	0.15	U	5.2		0.14	U	83	J

APPENDIX A-2
ANALYTICAL DATA FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SB-06A		SB-06B		SB-10A		SB-11A		TP-F	TP-G	SB-7A		SB-8A		SB-8B		TP-A
Date:	Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1995		Atlantic, 1995	Atlantic, 1995	Atlantic, 1994		Atlantic, 1995		Atlantic, 1995		Atlantic, 1995
Depth (feet):	4.3		8.8		4.8		2.5		4	14.2	4.8		4.3		18.5		14.2
	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	mg/kg	mg/kg		mg/kg		mg/kg		mg/kg
Inorganic Compounds																	
Aluminum	4760		8950		12100		16300		NA	NA	4560		12300		3010		NA
Arsenic	3.6		4.3		6.7		6.9		NA	NA	2.3	U	18.9		1.2	B	NA
Barium	35.9	B	26	B	119		66.1		NA	NA	73.4		154		23.3	B	NA
Beryllium	0.47	B	0.34	B	1.6		0.81	J	NA	NA	0.51	B	2.1	J	0.25	UJ	NA
Calcium	659	B	132000		27900		10100	J	NA	NA	935	B	34200	J	6700	J	NA
Chromium	8.6		8		21		19.6	J	NA	NA	6.1		9.8	J	5	J	NA
Cobalt	6.7	B	4.3	B	8.2	B	10.9	B	NA	NA	5.9	B	17.9		4.2	B	NA
Copper	9.9	J	12.7	J	24.7	J	13.5	J	NA	NA	5.8	J	67.6		5.7	U	NA
Iron	15600		13600		22000		25600		NA	NA	9840		37200		9650		NA
Lead	8.4	J	0.44	U	38.8	J	19.4		NA	NA	6.5	J	46.6		5		NA
Magnesium	1300		16900		5570		3250	J	NA	NA	1100	B	6210		1360		NA
Manganese	407		272		847		375		NA	NA	342		1040	J	114	J	NA
Mercury	0.06	U	0.06	U	0.11	B	0.07	B	NA	NA	0.07	B	0.21		0.06	U	NA
Nickel	12.4	UJ	11.3	UJ	15.2	UJ	13.5		NA	NA	9.3	UJ	16.9		6.6	B	NA
Potassium	980	B	1350		1710		1160	B	NA	NA	445	B	1870		303	B	NA
Selenium	0.99	B	0.89	U	0.92	U	0.87	UJ	NA	NA	1	U	2.2	J	0.89	UJ	NA
Sodium	60.4	U	114	J	115	J	97.5	U	NA	NA	54.7	U	214	U	43.4	U	NA
Thallium	2.1	U	2	U	2.1	U	1.4	UJ	NA	NA	2.3	U	1.1	UJ	1.4	UJ	NA
Vanadium	14.5		6	J	21.6	J	34.8	J	NA	NA	7.2	B	15.3	J	4.9	B	NA
Zinc	31.5		36		75.2		43.6	J	NA	NA	25.3		194	J	18.3		NA
Cyanide	0.29	U	0.28	U	5.8		0.6	U	NA	NA	0.32	U	0.98		0.62	U	NA

U - not detected at reported detection limit
J - estimate
NA - not analyzed
C - compound co-eluted with preceding constituent
R - rejected value
B - detected value between instrument limit and CRDL
Subsurface soil is defined as 6 inches to 15 feet deep.
Table presents compounds detected at least once in subsurface soil.
[1] - Duplicate sample of SB-2A

APPENDIX A-3
ANALYTICAL DATA FOR SEDIMENT - SHAWNEE CREEK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SED-1		SED-2		SED-4[1]		SED-3	
Date:	Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994	
Semivolatile Organic Compounds - ug/kg								
Anthracene	390	J	1100	U	2200	UJ	2500	U
Benzo(a)anthracene	1900		450	J	1000	J	920	J
Benzo(a)pyrene	1500	J	480	J	670	J	740	J
Benzo(b)fluoranthene	2400		670	J	1000	J	1300	J
Benzo(g,h,i)perylene	920	J	290	J	510	J	610	J
Benzo(k)fluoranthene	1100	J	310	J	710	J	500	J
Bis(2-ethylhexyl)phthalate	1200	J	410	J	680	J	1400	J
Chrysene	1900		600	J	880	J	1000	J
Fluoranthene	4400		1200		1500	J	2900	
Indeno(1,2,3-cd)pyrene	1100	J	290	J	780	J	750	J
Phenanthrene	2400		560	J	850	J	940	J
Pyrene	4700		1000	J	1700	J	2700	
Inorganic Compounds - mg/kg								
Aluminum	4980		7230		9200		6980	
Arsenic	6		8.5		16.1		10.4	
Barium	65.6		99.4		133		97.4	
Beryllium	1.6		1.2	B	1.3	B	1.6	B
Cadmium	0.96	J	1	J	1.5	J	1.3	J
Calcium	13800		7970		11100		16600	
Chromium	15.8		13.8		19.1		21.3	
Cobalt	9.2	B	10.1	B	11.1	B	9.9	B
Copper	477		251		195		391	
Cyanide	3.5	U	0.44	B	0.42	U	0.77	B
Iron	18500		19400		25000		27200	
Lead	188		139		189		307	
Magnesium	3570		2350		3050		4540	
Manganese	460		506		686		1300	
Mercury	0.07	U	0.12	B	0.08	U	0.19	
Nickel	21.4		18.5		22.4		27.1	
Potassium	1000	B	1560	B	1540	B	1300	B
Selenium	1.1	U	2.1	J	2.6	J	1.5	U
Sodium	184	B	133	B	189	B	175	B
Vanadium	10.6	J	14.1	J	19.6	J	16.9	J
Zinc	1700		1080		1190		1510	

ND - not detected
U - not detected at reported detection limit
J - estimate
B - detected value between instrument detection limit and CRDL
R - rejected
Table presents compounds detected at least once in Shawnee Creek sediment.
[1] - Duplicate of sample SED-2

APPENDIX A-4
ANALYTICAL DATA FOR SURFACE WATER - SHAWNEE CREEK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	SW-1		SW-2		SW-4[1]		SW-3	
Date	Atlantic, 1994		Atlantic, 1994		Atlantic, 1994		Atlantic, 1994	
Units:	ug/l		ug/l		ug/l		ug/l	
Inorganic Compounds								
Barium	26.6	B	27.5	B	27.8		29.4	B
Calcium	52800		54800		54800		55800	
Copper	5.7	B	4	U	4	U	4	U
Magnesium	15300		15700		15700		15900	
Manganese	12.2	B	11.7	B	11.2	B	14.8	B
Potassium	3140	B	3240	B	3160	B	3360	B
Selenium	9		4.8	J	8	J	4.1	J
Sodium	26400		27200		27600		27800	
Zinc	22.9		26.1		34.4		22.9	

ND - not detected
U - not detected at reported detection limit
J - estimate
B - detected value between instrument detection limit and CRDL
R - rejected
Table presents compounds detected at least once in Shawnee Creek surface water.
[1] - Duplicate of sample SW-2

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-1S	MW-1D	MW-3S	MW-1SR	MW-1SR2	MW-1DR	MW-1DR2	MW-2R	MW-2R2
Date:	NUS, 1991	NUS, 1991	NUS, 1991	Atlantic - 3/95	Atlantic - 7/95	Atlantic - 3/95	Atlantic - 7/95	Atlantic - 3/95	Atlantic - 7/95
Volatile Organic Compounds - mg/l									
Benzene	0.005 U	0.005 U	9.6	0.013 U	0.017 U	0.013 U	0.015 U	0.013 U	0.016 UB
Ethylbenzene	0.005 U	0.005 U	2.4	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Styrene	0.005 U	0.005 U	NA	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Tetrachloroethene	0.005 U	0.005 U	0.005	NA	NA	NA	NA	NA	NA
Toluene	0.005 U	0.006	1.5	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Trichloroethene	0.005 U	0.005 U	0.003 J	NA	NA	NA	NA	NA	NA
1,2,4-Trimethylbenzene	NA	NA	NA	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Total Xylenes	0.005 U	0.005 U	1.9	0.026 U	0.028 U	0.026 U	0.028 U	0.026 U	0.028 U
Semivolatile Organic Compounds - mg/l									
Acenaphthene	0.01 U	0.01 U	0.75	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Acenaphthylene	0.01 U	0.01 U	0.49	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Anthracene	0.01 U	0.01 U	0.32 J	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Benzo(a)anthracene	0.01 U	0.01 U	0.19	0.013 U	0.014 UJ	0.013 U	0.014 UJ	0.013 U	0.014 UJ
Benzo(a)pyrene	0.01 U	0.01 U	0.15	0.013 U	0.014 UJ	0.013 U	0.014 UJ	0.013 U	0.014 UJ
Benzo(b)fluoranthene	0.01 U	0.01 U	0.13	0.013 U	0.014 UJ	0.013 U	0.014 UJ	0.013 U	0.014 UJ
Bis(2-ethylhexyl)phthalate	0.01 U	0.01 U	0.069	NA	NA	NA	NA	NA	NA
Chrysene	0.01 U	0.01 U	0.14	0.013 U	0.014 U	0.013 U	0.014 UJ	0.013 U	0.014 U
Dibenzofuran	0.01 U	0.01 U	0.081	0.013 UJ	0.014 UJ	0.013 UJ	0.014 UJ	0.013 UJ	0.014 UJ
Fluoranthene	0.01 U	0.01 U	0.28	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Fluorene	0.01 U	0.01 U	0.12	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
1-Methylnaphthalene	NA	NA	NA	0.013 U	0.014 UJ	0.013 U	0.014 UJ	0.013 U	0.014 UJ
2-Methylnaphthalene	0.01 U	0.01 U	2.6	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
2-Methylphenol	0.01 U	0.01 U	0.009 J	NA	NA	NA	NA	NA	NA
4-Methylphenol	0.01 U	0.01 U	0.006 J	NA	NA	NA	NA	NA	NA
Naphthalene	0.01 U	0.01 U	7.6	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Phenanthrene	0.01 U	0.01 U	1.2	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U
Phenol	0.01 U	0.01 U	0.036	NA	NA	NA	NA	NA	NA
Pyrene	0.01 U	0.01 U	0.72	0.013 U	0.014 U	0.013 U	0.014 U	0.013 U	0.014 U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-1S		MW-1D		MW-3S		MW-1SR		MW-1SR2		MW-1DR		MW-1DR2		MW-2R		MW-2R2	
Date:	NUS, 1991		NUS, 1991		NUS, 1991		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95	
Inorganics - ug/l																		
Aluminum	44900		24000		9570		12	U	14.4	U	182	B	14.4	U	48.3	U	14.4	U
Arsenic	10	U	10	U	10	U	1.4	U	1.3	U	2.6	U	1.3	U	1.4	U	1.3	U
Barium	273		290		177	J	29	B	29	B	116	B	109	B	69.8	B	65.3	B
Beryllium	1.9	J	2.4	J	5	U	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U
Calcium	507000		373000		204000		110000		116000		50800		94600		109000		110000	
Chromium	20.2		10	U	10	U	1.8	UJ	1.6	U	8.2	J	1.6	U	1.8	UJ	1.6	U
Cobalt	18.4	J	22	J	7.8	J	1.4	U	2	U	1.4	U	2	U	1.4	U	2	U
Copper	52.8		56		31		1.1	UJ	4.7	U	1.1	UJ	4.7	U	1.1	UJ	4.7	U
Cyanide	4	J	5.8	J	136		5	U	5	U	5	U	5	U	90.7		125	
Iron	81600		404000		41700		7.3	UJ	3.9	U	7.3	UJ	7.5	U	81	B	75	B
Lead	49.1		60		18.9		0.9	U	1.6	UJ	1.4	U	1.6	UJ	0.9	U	1.6	UJ
Magnesium	83700		49200		31900		16100		16800		13100		25300		22900		20400	
Manganese	2750	J	2410	J	1430	J	3.4	B	5	B	0.5	U	8.6	B	72.9		171	
Nickel	47.8		50.9		23.2	J	3.7	U	5.4	U	3.7	U	8.6	B	3.7	U	5.4	U
Potassium	12600		7780		9270		5350		5040		15500		8690		4900	B	4940	B
Selenium	2.2	J	5	U	5	U	2.6	U	2.7	U	2.6	U	2.7	B	2.6	U	3.7	B
Sodium	45500		71500		19600		28100		26700		30800		29600		23800		27900	
Thallium	1.1	J	10	U	10	U	4.2	U	4.1	U	4.2	U	4.1	U	4.2	U	4.1	U
Vanadium	58.3		53.6		18.8	J	1.6	B	2.7	U	5.2	B	2.6	U	1.8	B	2.6	U
Zinc	141		225		77.2		1.1	UJ	33.2	U	1.1	UJ	6.8	U	1.1	UJ	6.7	U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-3D		MW-3D2		MW-4		MW-9S2		MW-5		MW-52		MW-6S		MW-6S2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95	
Volatile Organic Compounds - mg/l																
Benzene	23	J	14	J	0.013	U	0.018	UB	0.013	UB	0.014	UB	1.7		0.24	
Ethylbenzene	3.9		4.7		0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.5		0.075	J
Styrene	0.072		0.076		0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.013	U	0.014	U
Tetrachloroethene	NA		NA		NA		NA		NA		NA		NA		NA	
Toluene	6.1	J	7		0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.056		0.014	U
Trichloroethene	NA		NA		NA		NA		NA		NA		NA		NA	
1,2,4-Trimethylbenzene	0.35		0.44		0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.072		0.016	J
Total Xylenes	3.1		3.7		0.026	U	0.028	UJ	0.026	UJ	0.028	UJ	0.3		0.028	U
Semivolatile Organic Compounds - mg/l																
Acenaphthene	0.092	J	0.14		0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.038		0.016	J
Acenaphthylene	0.051	J	0.055		0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
Anthracene	0.013	U	0.014	U	0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
Benzo(a)anthracene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.013	U	0.014	UJ
Benzo(a)pyrene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.013	U	0.014	UJ
Benzo(b)fluoranthene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.013	U	0.014	UJ
Bis(2-ethylhexyl)phthalate	NA		NA		NA		NA		NA		NA		NA		NA	
Chrysene	0.013	U	0.014	U	0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
Dibenzofuran	0.013	UJ	0.03	J	0.013	UJ	0.014	UJ	0.013	UJ	0.014	UJ	0.013	UJ	0.014	UJ
Fluoranthene	0.013	U	0.014	U	0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
Fluorene	0.013	U	0.023		0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
1-Methylnaphthalene	0.34	J	0.47	J	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ	0.14		0.038	J
2-Methylnaphthalene	0.49	J	0.67		0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.099		0.014	UJ
2-Methylphenol	NA		NA		NA		NA		NA		NA		NA		NA	
4-Methylphenol	NA		NA		NA		NA		NA		NA		NA		NA	
Naphthalene	6.4	J	8.1		0.013	U	0.014	UJ	0.013	UJ	0.014	U	1.2		0.11	J
Phenanthrene	0.016	J	0.032		0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ
Phenol	NA		NA		NA		NA		NA		NA		NA		NA	
Pyrene	0.013	U	0.014	U	0.013	U	0.014	UJ	0.013	UJ	0.014	U	0.013	U	0.014	UJ

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-3D		MW-3D2		MW-4		MW-9S2		MW-5		MW-52		MW-6S		MW-6S2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95	
Inorganics - ug/l																
Aluminum	15.9	U	14.4	U	63.2	U	83	U	117	B	14.4	U	20.7	U	14.4	U
Arsenic	3.3	U	2.4	B	1.4	U	1.3	U	6.3	U	1.3	U	1.7	U	2.7	B
Barium	118	B	132	B	40.2	B	41.9	B	137	B	79.1	B	163	B	151	B
Beryllium	0.2	U	0.2	U	0.2	U	0.2	U	0.23	U	0.2	U	0.2	U	0.2	U
Calcium	107000		115000		125000		126000		136000		125000		144000		139000	
Chromium	1.8	UJ	1.6	U	1.8	UJ	1.6	U	1.8	UJ	1.6	U	1.8	UJ	1.6	U
Cobalt	3	U	2	U	1.4	U	2	U	3.8	U	2	U	7.9	U	6.2	U
Copper	1.1	UJ	4.7	U	1.1	UJ	4.7	U	3.5	J	4.7	U	1.1	UJ	4.7	U
Cyanide	17.2		14.9		7.6	B	15.7		5	U	5	U	116		116	
Iron	14700		14100		215		130		24500		1880		6250		6110	
Lead	0.9	U	1.6	UJ	0.9	U	1.6	UJ	0.9	U	1.6	UJ	0.9	U	1.6	UJ
Magnesium	19800		22100		17300		17000		22800		21600		24800		24700	
Manganese	350		298		2.2	B	1	B	487		450		2540		2640	
Nickel	3.7	U	5.4	U	3.7	U	5.4	U	3.8	B	5.4	U	5.2	B	5.4	U
Potassium	4830	B	6000		5340		5910		13000		11700		7480		8270	
Selenium	2.6	U	2.7	U	4.2	B	3.5	B	2.6	U	3.3	B	2.6	U	2.7	U
Sodium	22100		25900		21500		23900		28100		27500		31200		38900	
Thallium	4.2	U	4.1	U	4.2	U	4.1	U	4.2	U	4.1	U	4.2	U	4.1	U
Vanadium	1.8	B	1.7	U	1.5	U	1.1	U	3	B	2.9	U	1.6	B	2	U
Zinc	1.1	UJ	6.7	U	1.1	UJ	14.8	U	2.6	J	11.1	U	1.1	UJ	6.7	U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-6D		MW-6D2		MW-7S		MW-7S2		MW-7D		MW-7D2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95	
Volatile Organic Compounds - mg/l												
Benzene	39	J	20		0.013	U	0.014	UB	0.013	UJ	0.022	UB
Ethylbenzene	3.5	J	3.2	J	0.013	U	0.014	U	0.013	UJ	0.014	U
Styrene	0.014	UJ	0.014	UJ	0.013	U	0.014	U	0.013	UJ	0.014	U
Tetrachloroethene	NA		NA		NA		NA		NA		NA	
Toluene	9.5	J	7.6	J	0.013	U	0.014	U	0.013	UJ	0.014	U
Trichloroethene	NA		NA		NA		NA		NA		NA	
1,2,4-Trimethylbenzene	0.47	J	0.37	J	0.013	U	0.014	U	0.013	UJ	0.014	U
Total Xylenes	2.9	J	2.78	J	0.026	U	0.028	U	0.026	UJ	0.028	U
Semivolatile Organic Compounds - mg/l												
Acenaphthene	0.15	J	0.13	J	0.053		0.075		0.013	UJ	0.014	U
Acenaphthylene	0.18	J	0.052	J	0.013	U	0.014	U	0.013	UJ	0.014	U
Anthracene	0.01	J	0.014	UJ	0.013	U	0.014	U	0.013	UJ	0.014	U
Benzo(a)anthracene	0.014	UJ	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ
Benzo(a)pyrene	0.014	UJ	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ
Benzo(b)fluoranthene	0.014	UJ	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.014	UJ
Bis(2-ethylhexyl)phthalate	NA		NA		NA		NA		NA		NA	
Chrysene	0.014	UJ	0.014	UJ	0.013	U	0.014	U	0.013	UJ	0.014	U
Dibenzofuran	0.065	J	0.048	J	0.013	UJ	0.014	UJ	0.013	UJ	0.014	UJ
Fluoranthene	0.014	UJ	0.014	UJ	0.013	U	0.014	U	0.013	UJ	0.014	U
Fluorene	0.043	J	0.023	J	0.013	U	0.014	U	0.013	UJ	0.014	U
1-Methylnaphthalene	0.75	J	0.43	J	0.037		0.037	J	0.013	UJ	0.014	UJ
2-Methylnaphthalene	0.94	J	0.54	J	0.013	U	0.014	U	0.013	UJ	0.014	U
2-Methylphenol	NA		NA		NA		NA		NA		NA	
4-Methylphenol	NA		NA		NA		NA		NA		NA	
Naphthalene	8.2	J	5.2	J	0.013	U	0.03		0.013	UJ	0.019	
Phenanthrene	0.036	J	0.017	J	0.013	U	0.03		0.013	UJ	0.014	U
Phenol	NA		NA		NA		NA		NA		NA	
Pyrene	0.014	UJ	0.014	UJ	0.013	U	0.014	U	0.013	UJ	0.014	U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-6D		MW-6D2		MW-7S		MW-7S2		MW-7D		MW-7D2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95	
Inorganics - ug/l												
Aluminum	12	U	14.4	U	364		253		16.5	U	14.4	U
Arsenic	1.4	U	1.3	U	2.8	U	1.3	U	1.4	U	1.3	U
Barium	541		427		120	B	96.8	B	48.2	B	47.3	B
Beryllium	0.2	U	0.2	U	0.26	U	0.2	U	0.2	U	0.2	U
Calcium	110000		102000		156000		144000		132000		130000	
Chromium	1.8	UJ	1.6	U	1.8	UJ	1.6	U	1.8	UJ	1.6	U
Cobalt	1.4	U	2	U	10.6	B	8.1	U	1.4	U	2	U
Copper	1.1	UJ	4.7	U	4.2	J	7.2	B	1.1	UJ	4.7	U
Cyanide	223		195		23.5		11.9		7.4	B	8.2	
Iron	4370		9620		1120		1530		7.3	UJ	9.3	U
Lead	0.9	U	1.6	UJ	8.2		6.1	UJ	1.1	U	1.6	UJ
Magnesium	31600		32600		33500		26000		19800		20500	
Manganese	107		80.7		2970		2230		3.1	B	6.7	B
Nickel	3.7	U	5.4	U	8.2	B	5.4	U	3.7	U	5.4	U
Potassium	4330	B	6230		5380		5700		5900		5910	
Selenium	2.6	U	2.7	U	3.6	B	2.7	U	4.6	B	6.2	
Sodium	28300		29700		35800		27100		22700		23100	
Thallium	4.2	U	4.1	U	4.2	U	4.1	U	4.2	U	4.1	U
Vanadium	2.2	B	2.3	U	3	B	2.9	U	1.6	B	3.2	U
Zinc	1.1	UJ	6.7	U	10.4	J	22.1	U	8.5	J	6.7	U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-8S		MW-8S2		MW-8D		MW-8D2		CWW-1		CWW-2		CWW-1-2		CWW-2-2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 8/95	
Volatile Organic Compounds - mg/l																
Benzene	0.013	U	0.014	UB	0.021		0.086		0.049	J	0.046	J	0.14		0.24	D
Ethylbenzene	0.013	U	0.014	U	0.013	U	0.014	U	0.011	J	0.009	J	0.023		0.058	
Styrene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Tetrachloroethene	NA		NA		NA		NA		NA		NA		0.01	U	0.01	U
Toluene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.003	J	0.002	J
Trichloroethene	NA		NA		NA		NA		NA		NA		0.01	U	0.01	U
1,2,4-Trimethylbenzene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	NA		NA	
Total Xylenes	0.026	U	0.028	U	0.026	U	0.028	U	0.026	UJ	0.026	UJ	0.024		0.029	
Semivolatile Organic Compounds - mg/l																
Acenaphthene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.003	J	0.002	J
Acenaphthylene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.01	UJ	0.01	U	0.01	U
Anthracene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Benzo(a)anthracene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Benzo(a)pyrene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Benzo(b)fluoranthene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Bis(2-ethylhexyl)phthalate	NA		NA		NA		NA		NA		NA		0.01	U	0.01	U
Chrysene	0.013	U	0.014	UJ	0.013	U	0.014	UJ	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Dibenzofuran	0.013	UJ	0.014	UJ	0.013	UJ	0.014	UJ	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Fluoranthene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Fluorene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
1-Methylnaphthalene	0.013	U	0.014	UJ	0.013	U	0.009	J	0.013	UJ	0.013	UJ	NA		NA	
2-Methylnaphthalene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
2-Methylphenol	NA		NA		NA		NA		NA		NA		0.01	U	0.01	U
4-Methylphenol	NA		NA		NA		NA		NA		NA		0.01	U	0.01	U
Naphthalene	0.013	U	0.014	UJ	0.013	U	0.018	J	0.013	UJ	0.013	UJ	0.009	J	0.01	U
Phenanthrene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U
Phenol	NA		NA		NA		NA		NA		NA		0.006	J	0.002	J
Pyrene	0.013	U	0.014	U	0.013	U	0.014	U	0.013	UJ	0.013	UJ	0.01	U	0.01	U

APPENDIX A-5
ANALYTICAL DATA FOR GROUNDWATER
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Location:	MW-8S		MW-8S2		MW-8D		MW-8D2		CWW-1		CWW-2		CWW-1-2		CWW-2-2	
Date:	Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 3/95		Atlantic - 3/95		Atlantic - 7/95		Atlantic - 8/95	
Inorganics - ug/l																
Aluminum	51	U	14.4	U	21.6	U	14.4	U	12	U	12	U	14.4	U	22.1	U
Arsenic	1.4	U	1.3	U	1.4	U	1.3	U	1.4	U	1.4	U	1.3	U	1.3	U
Barium	73.5	B	69.7	B	86.5	B	94.8	B	73.5	B	65.6	B	72.4	B	77.2	B
Beryllium	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U	0.2	U
Calcium	126000		126000		125000		124000		127000		127000		113000		121000	
Chromium	1.8	UJ	1.6	U	1.8	UJ	1.6	U	1.8	UJ	1.8	UJ	1.6	U	1.6	U
Cobalt	1.6	U	2	U	1.4	U	2	U	1.4	U	1.6	U	2	U	2	UJ
Copper	1.1	J	4.7	U	1.1	UJ	4.7	U	9.1	B	1.8	J	6.9	B	7.1	B
Cyanide	114		146		17.6		25.8		5	U	8	B	5.2		5	UJ
Iron	232		68.8	B	25.2	J	37.7	U	108		10.6	J	33.4	U	43.7	B
Lead	0.9	U	1.6	UJ	0.9	U	1.6	UJ	1.6	B	1.1	B	1.6	UJ	2.2	B
Magnesium	19700		18400		22700		23200		24700		23100		23600		24800	
Manganese	661		936		89.4		77.4		83.1		25.9		41.8		43.4	
Nickel	3.7	U	5.4	U	3.7	U	5.4	U	3.7	U	3.7	U	5.4	U	5.4	U
Potassium	7710		5880		6450		6730		6470		6370		6300		6370	
Selenium	2.9	B	2.8	B	2.9	B	3.5	B	2.6	U	3	B	2.7	U	2.7	U
Sodium	35000		36100		36000		37000		38700		37500		37200		39300	
Thallium	4.2	U	4.1	U	4.2	U	4.1	U	4.2	U	4.2	U	4.1	U	7.5	U
Vanadium	2	B	2.3	U	1.8	B	1.8	U	1.8	B	2.4	B	3.5	U	3.6	B
Zinc	1.1	UJ	6.7	U	1.1	UJ	6.7	U	123		66.6	J	153	U	143	

NA - not analyzed
U - not detected at reported detection limit
J - estimated
B - detected value between instrument detection limit and CRDL
Table presents compounds detected at least once in groundwater.

APPENDIX B

SUMMARY STATISTICS FOR EXPOSURE MEDIA

TRESPASSER SCENARIO
SUMMARY STATISTICS FOR SHAWNEE CREEK SEDIMENTS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	SUMMARY STATISTICS						
	Number Analyzed	Number Detected	Minimum Detected mg/kg	Maximum Detected mg/kg	Average Concentration mg/kg	95% Upper Confidence Level (UCL) mg/kg	Reasonable Maximum Exposure (RME) Concentration[2] mg/kg
Benzo(a)anthracene	2	2	0.92	1	0.96	Greater than Maximum	1
Benzo(b)fluoranthene	2	2	1	1.3	1.15	Greater than Maximum	1.3
Benzo(g,h,i)perylene [1]	2	2	0.51	0.61	0.56	Greater than Maximum	0.61
Benzo(a)pyrene	2	2	0.67	0.74	0.71	Greater than Maximum	0.74
Inorganics							
Aluminum	2	2	6.98	9200	8.09	Greater than Maximum	9200
Arsenic	2	2	0.0104	16.1	0.01	Greater than Maximum	16.1
Copper	2	2	0.195	391	0.29	Greater than Maximum	391
Iron	2	2	25	27200	26.10	Greater than Maximum	27200
Manganese	2	2	0.686	1300	0.99	Greater than Maximum	1300

[1] Includes SD-3 (Atlantic), and SD-4 (Atlantic)
[2] RME concentration is minimum of the maximum concentration and 95% UCL

TRESPASSER SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	12	6	0.09	0.79	0.13	Greater than Max.	0.79
Ethylbenzene	12	3	0.08	0.25	0.07	Greater than Max.	0.25
Toluene	12	8	0.07	1.3	0.20	Greater than Max.	1.3
1,2,4-Trimethylbenzene	8	5	0.41	1.4	0.48	Greater than Max.	1.4
Xylenes (Mixed)	12	7	0.16	3.28	0.60	Greater than Max.	3.3
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	12	5	0.17	0.26	0.15	0.21	0.21
Acenaphthylene	12	11	0.080	3.8	1.1	Greater than Max.	3.8
Anthracene	12	9	0.20	1.3	0.54	Greater than Max.	1.3
Benzo(ghi)perylene	12	11	0.20	9.0	2.7	Greater than Max.	9.0
Fluoranthene	12	10	0.19	6.6	2.4	Greater than Max.	6.6
Fluorene	12	6	0.060	0.49	0.19	0.34	0.34
1-Methylnaphthalene	8	6	0.12	0.76	0.36	Greater than Max.	0.76
2-Methylnaphthalene	12	9	0.090	1.0	0.49	Greater than Max.	1.0
Naphthalene	12	10	0.060	1.7	0.72	Greater than Max.	1.7
Phenanthrene	12	10	0.070	3.4	1.2	Greater than Max.	3.4
Pyrene	12	11	0.14	12	4.4	Greater than Max.	12
Carcinogenic PAHs							
Benzo(a)anthracene	12	10	0.20	4.6	2.0	Greater than Max.	4.6
Benzo(a)pyrene	12	11	0.10	6.2	2.7	Greater than Max.	6.2
Benzo(b)fluoranthene	12	9	0.47	6.7	1.9	Greater than Max.	6.7

TRESPASSER SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Benzo(k)fluoranthene	12	10	0.36	6.1	2.0	Greater than Max.	6.1
Chrysene	12	10	0.20	5.5	2.3	Greater than Max.	5.5
Dibenzo(ah)anthracene	12	0	0	0	0.11	Greater than Max.	ND
Indeno(123-cd)pyrene	12	10	0.17	5.9	2.0	Greater than Max.	5.9
Phthalates							
bis(2-Ethylhexyl)phthalate	4	2	0.11	0.25	0.42	Greater than Max.	0.25
Other SVOCs							
Dibenzofuran	12	4	0.09	0.38	0.14	0.20	0.20
Inorganic Compounds							
Aluminum	12	12	2060	10700	5509	7956	7956
Arsenic	12	12	2	14	5.9	8.85	9
Barium	12	10	16	221	73	Greater than Max.	221
Beryllium	12	5	0.060	0.54	0.23	0.38	0.38
Cadmium	12	2	4.7	5.8	1.2	3.43	3.427486028
Chromium (Total)	10	8	1.9	32	11	Greater than Max.	32
Copper	12	12	6.1	112	41.1	Greater than Max.	112
Cyanide	12	8	0.69	48	7.0	46	46
Iron	12	12	4710	41800	17897	30748	30748
Lead	12	12	5.0	634	125	Greater than Max.	634
Manganese	12	12	93	1700	431	1033	1033
Nickel	12	9	5.6	23	14	Greater than Max.	23
Thallium	12	3	0.15	0.26	0.41	Greater than Max.	0.26
Vanadium	12	8	4.5	24	11.1	23.89	24
Zinc	12	12	15	410	161	Greater than Max.	410

[1] Includes samples S-1, S-2, S-3, S-4, SS-1, SS-2, SS-3, SS-4, SS-5, SS-6, SS-7, SS-8

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

TRESPASSER SCENARIO
SUMMARY STATISTICS FOR SHAWNEE CREEK SEDIMENTS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

COMPOUNDS	SUMMARY STATISTICS						
	Number Analyzed	Number Detected	Minimum Detected mg/kg	Maximum Detected mg/kg	Average Concentration mg/kg	95% Upper Confidence Level (UCL) mg/kg	Reasonable Maximum Exposure Point Concentration[4] mg/kg
Benzo(a)anthracene	2	2	0.92	1	0.96	Greater than Maximum	1
Benzo(b)fluoranthene	2	2	1	1.3	1.15	Greater than Maximum	1.3
Benzo(g,h,i)perylene[3]	2	2	0.51	0.61	0.56	Greater than Maximum	0.61
Benzo(a)pyrene	2	2	0.67	0.74	0.71	Greater than Maximum	0.74
Inorganics							
Aluminum	2	2	6980	9200	8090	Greater than Maximum	9200
Arsenic	2	2	10.4	16.1	13	Greater than Maximum	16.1
Copper	2	2	195	391	293	Greater than Maximum	391
Iron	2	2	25000	27200	26100	Greater than Maximum	27200
Manganese	2	2	686	1300	993	Greater than Maximum	1300

[1] Includes samples SD-3 and SD-4 (SD-4 is a duplicate of SD-2)
[2] The reasonable maximum exposure point concentration is the minimum of the maximum detected concentration and the 95% UCL
[3] Evaluated qualitatively

INDUSTRIAL WORKER SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	7	3	0.09	0.79	0.15	Greater than Max.	0.79
Ethylbenzene	7	2	0.08	0.25	0.06	Greater than Max.	0.25
Toluene	7	3	0.07	1.3	0.24	Greater than Max.	1.3
1,2,4-Trimethylbenzene	3	2	0.41	1.4	0.63	Greater than Max.	1.4
Xylenes (Mixed)	7	3	0.16	3.28	0.61	Greater than Max.	3.3
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	7	2	0.17	0.24	0.15	Greater than Max.	0.24
Acenaphthylene	7	6	0.190	3.8	1.5	Greater than Max.	3.8
Anthracene	7	5	0.25	1.3	0.66	Greater than Max.	1.3
Benzo(ghi)perylene	7	6	0.20	9.0	3.3	Greater than Max.	9.0
Fluoranthene	7	6	0.19	6.6	2.6	Greater than Max.	6.6
Fluorene	7	3	0.060	0.47	0.18	Greater than Max.	0.47
1-Methylnaphthalene	3	2	0.38	0.76	0.40	Greater than Max.	0.76
2-Methylnaphthalene	7	5	0.260	1.0	0.59	Greater than Max.	1.0
Naphthalene	7	6	0.060	1.7	0.85	Greater than Max.	1.7
Phenanthrene	7	6	0.070	3.4	1.4	Greater than Max.	3.4
Pyrene	7	6	0.45	12	5.1	Greater than Max.	12
Carcinogenic PAHs							
Benzo(a)anthracene	7	6	0.20	4.6	2.4	Greater than Max.	4.6
Benzo(a)pyrene	7	6	0.47	6.2	3.2	Greater than Max.	6.2
Benzo(b)fluoranthene	7	5	1.1	6.7	2.5	Greater than Max.	6.7

INDUSTRIAL WORKER SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Benzo(k)fluoranthene	7	6	0.36	6.1	2.4	Greater than Max.	6.1
Chrysene	7	6	0.20	5.5	2.7	Greater than Max.	5.5
Dibenzo(ah)anthracene	7	0	0	0	0.13	Greater than Max.	ND
Indeno(123-cd)pyrene	7	6	0.17	5.9	2.5	Greater than Max.	5.9
Phthalates							
bis(2-Ethylhexyl)phthalate	4	2	0.11	0.25	0.42	Greater than Max.	0.25
Other SVOCs							
Dibenzofuran	7	2	0.1	0.15	0.14	Greater than Max.	0.15
Inorganic Compounds							
Aluminum	7	7	2060	7920	5194	Greater than Max.	7920
Arsenic	7	7	2	10	5.6	Greater than Max.	10
Barium	7	6	16	146	65	Greater than Max.	146
Beryllium	7	4	0.240	0.54	0.27	Greater than Max.	0.54
Cadmium	7	2	4.7	5.8	1.8	Greater than Max.	5.8
Chromium (Total)	6	5	1.9	32	11	Greater than Max.	32
Copper	7	7	6.1	83.5	32.1	Greater than Max.	84
Cyanide	7	5	0.69	25	4.5	Greater than Max.	25
Iron	7	7	4710	41800	18514	Greater than Max.	41800
Lead	7	7	5.0	634	141	Greater than Max.	634
Manganese	7	7	93	807	334	Greater than Max.	807
Nickel	7	6	5.6	23	14	Greater than Max.	23
Thallium	7	1	0.21	0.21	0.62	Greater than Max.	0.21
Vanadium	7	5	4.5	24	9.9	Greater than Max.	24
Zinc	7	7	15	410	163	Greater than Max.	410

[1] Includes samples S-1, S-2, S-3, S-4, SS-2, SS-3, SS-4

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

INDUSTRIAL WORKER SCENARIO
SUMMARY STATISTICS FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	9	4	0.094	1.49	0.45	Greater than Maximum	1.49
Ethylbenzene	9	6	0.012	29	5.7	Greater than Maximum	29
Toluene	9	6	0.022	2.9	0.51	Greater than Maximum	3
1,2,4-Trimethylbenzene	8	5	2.9	46	11.8	Greater than Maximum	46
Xylenes (Mixed)	9	7	0.07	30	7.3	Greater than Maximum	30
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	9	7	0.08	85	24	Greater than Maximum	85
Acenaphthylene	9	8	0.11	30	10	Greater than Maximum	30
Anthracene	9	7	0.72	53	13	Greater than Maximum	53
Benzo(ghi)perylene	9	6	0.21	11	2.5	Greater than Maximum	11
Fluoranthene	9	7	0.38	92	26	Greater than Maximum	92
Fluorene	9	6	4	59	16	Greater than Maximum	59
1-Methylnaphthalene	8	6	0.15	92	36	Greater than Maximum	92
2-Methylnaphthalene	9	5	18	130	24	Greater than Maximum	130
Naphthalene	9	6	13	190	41	Greater than Maximum	190
Phenanthrene	9	8	0.14	160	39	Greater than Maximum	160
Pyrene	9	8	0.09	150	43	Greater than Maximum	150
Carcinogenic PAHs							
Benzo(a)anthracene	9	7	0.87	47	15	Greater than Maximum	47
Benzo(a)pyrene	9	7	0.21	35	10.2	Greater than Maximum	35
Benzo(b)fluoranthene	9	7	1	39	10.5	Greater than Maximum	39

INDUSTRIAL WORKER SCENARIO
SUMMARY STATISTICS FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Benzo(k)fluoranthene	9	5	0.13	18	2.1	Greater than Maximum	18
Chrysene	9	7	3.6	52	16	Greater than Maximum	52
Dibenzo(ah)anthracene	9	1	0.15	0.15	0.10	0.1	0.13
Indeno(123-cd)pyrene	9	5	1.1	15	3.3	Greater than Maximum	15
Other SVOCs							
Dibenzofuran	9	3	1.3	18	3.6	Greater than Maximum	18
Inorganic Compounds							
Aluminum	9	9	4760	21400	12700	20351	20351
Arsenic	9	6	3.6	18	5.8	Greater than Maximum	18
Barium	9	4	53.6	191	53	139	139
Beryllium	9	2	0.54	3.9	0.74	1.9	1.9
Cadmium	4	0	0	0	0.03	Greater than Maximum	ND
Chromium (Total)	9	9	6.3	18.3	11	14	14
Copper	9	9	9.9	55	24	41	41
Cyanide	9	2	3.8	45	5.7	Greater than Maximum	45
Iron	9	9	13600	37400	24433	32147	32147
Lead	9	8	3.5	140	23	Greater than Maximum	140
Manganese	9	9	56.2	2490	598	Greater than Maximum	2490
Nickel	9	4	15.8	29	14	Greater than Maximum	29
Thallium	9	3	2.4	3	1.61	2.4	2.4
Vanadium	9	9	6	22	16	Greater than Maximum	22
Zinc	9	9	31.5	115	69	103	103

[1] Includes samples SUB-4, SB-14A, SB-2B, SB-3A, SB-4A, SB-5A, SB-5B, SB-6A, SB-6B

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

ON-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Volatile Organic Compounds							
Benzene	7	3	0.09	0.79	0.15	Greater than Max.	0.79
Ethylbenzene	7	2	0.08	0.25	0.06	Greater than Max.	0.25
Toluene	7	3	0.07	1.3	0.24	Greater than Max.	1.3
1,2,4-Trimethylbenzene	3	2	0.41	1.4	0.63	Greater than Max.	1.4
Xylenes (Mixed)	7	3	0.16	3.28	0.61	Greater than Max.	3.3
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	7	2	0.17	0.24	0.15	Greater than Max.	0.24
Acenaphthylene	7	6	0.190	3.8	1.5	Greater than Max.	3.8
Anthracene	7	5	0.25	1.3	0.66	Greater than Max.	1.3
Benzo(ghi)perylene	7	6	0.20	9.0	3.3	Greater than Max.	9.0
Fluoranthene	7	6	0.19	6.6	2.6	Greater than Max.	6.6
Fluorene	7	3	0.060	0.47	0.18	Greater than Max.	0.47
1-Methylnaphthalene	3	2	0.38	0.76	0.40	Greater than Max.	0.76
2-Methylnaphthalene	7	5	0.260	1.0	0.59	Greater than Max.	1.0
Naphthalene	7	6	0.060	1.7	0.85	Greater than Max.	1.7
Phenanthrene	7	6	0.070	3.4	1.4	Greater than Max.	3.4
Pyrene	7	6	0.45	12	5.1	Greater than Max.	12
Carcinogenic PAHs							
Benzo(a)anthracene	7	6	0.20	4.6	2.4	Greater than Max.	4.6
Benzo(a)pyrene	7	6	0.47	6.2	3.2	Greater than Max.	6.2
Benzo(b)fluoranthene	7	5	1.1	6.7	2.5	Greater than Max.	6.7

ON-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Benzo(k)fluoranthene	7	6	0.36	6.1	2.4	Greater than Max.	6.1
Chrysene	7	6	0.20	5.5	2.7	Greater than Max.	5.5
Dibenzo(ah)anthracene	7	0	0	0	0.13	Greater than Max.	ND
Indeno(123-cd)pyrene	7	6	0.17	5.9	2.5	Greater than Max.	5.9
Phthalates							
bis(2-Ethylhexyl)phthalate	4	2	0.11	0.25	0.42	Greater than Max.	0.25
Other SVOCs							
Dibenzofuran	7	2	0.1	0.15	0.14	Greater than Max.	0.15
Inorganic Compounds							
Aluminum	7	7	2060	7920	5194	Greater than Max.	7920
Arsenic	7	7	2	10	5.6	Greater than Max.	10
Barium	7	6	16	146	65	Greater than Max.	146
Beryllium	7	4	0.240	0.54	0.27	Greater than Max.	0.54
Cadmium	7	2	4.7	5.8	1.8	Greater than Max.	5.8
Chromium (Total)	6	5	1.9	32	11	Greater than Max.	32
Copper	7	7	6.1	83.5	32.1	Greater than Max.	84
Cyanide	7	5	0.69	25	4.5	Greater than Max.	25
Iron	7	7	4710	41800	18514	Greater than Max.	41800
Lead	7	7	5.0	634	141	Greater than Max.	634
Manganese	7	7	93	807	334	Greater than Max.	807
Nickel	7	6	5.6	23	14	Greater than Max.	23
Thallium	7	1	0.21	0.21	0.62	Greater than Max.	0.21
Vanadium	7	5	4.5	24	9.9	Greater than Max.	24
Zinc	7	7	15	410	163	Greater than Max.	410

[1] Includes samples S-1, S-2, S-3, S-4, SS-2, SS-3, SS-4

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

ON-SITE CONSTRUCTION WORKER SCENARIO
SUMMARY STATISTICS FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	9	4	0.094	1.49	0.45	Greater than Maximum	1.49
Ethylbenzene	9	6	0.012	29	5.7	Greater than Maximum	29
Toluene	9	6	0.022	2.9	0.51	Greater than Maximum	3
1,2,4-Trimethylbenzene	8	5	2.9	46	11.8	Greater than Maximum	46
Xylenes (Mixed)	9	7	0.07	30	7.3	Greater than Maximum	30
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	9	7	0.08	85	24	Greater than Maximum	85
Acenaphthylene	9	8	0.11	30	10	Greater than Maximum	30
Anthracene	9	7	0.72	53	13	Greater than Maximum	53
Benzo(ghi)perylene	9	6	0.21	11	2.5	Greater than Maximum	11
Fluoranthene	9	7	0.38	92	26	Greater than Maximum	92
Fluorene	9	6	4	59	16	Greater than Maximum	59
1-Methylnaphthalene	8	6	0.15	92	36	Greater than Maximum	92
2-Methylnaphthalene	9	5	18	130	24	Greater than Maximum	130
Naphthalene	9	6	13	190	41	Greater than Maximum	190
Phenanthrene	9	8	0.14	160	39	Greater than Maximum	160
Pyrene	9	8	0.09	150	43	Greater than Maximum	150
Carcinogenic PAHs							
Benzo(a)anthracene	9	7	0.87	47	15	Greater than Maximum	47
Benzo(a)pyrene	9	7	0.21	35	10.2	Greater than Maximum	35
Benzo(b)fluoranthene	9	7	1	39	10.5	Greater than Maximum	39

ON-SITE CONSTRUCTION WORKER SCENARIO
SUMMARY STATISTICS FOR SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Benzo(k)fluoranthene	9	5	0.13	18	2.1	Greater than Maximum	18
Chrysene	9	7	3.6	52	16	Greater than Maximum	52
Dibenzo(ah)anthracene	9	1	0.15	0.15	0.10	0.1	0.13
Indeno(123-cd)pyrene	9	5	1.1	15	3.3	Greater than Maximum	15
Other SVOCs							
Dibenzofuran	9	3	1.3	18	3.6	Greater than Maximum	18
Inorganic Compounds							
Aluminum	9	9	4760	21400	12700	20351	20351
Arsenic	9	6	3.6	18	5.8	Greater than Maximum	18
Barium	9	4	53.6	191	53	139	139
Beryllium	9	2	0.54	3.9	0.74	1.9	1.9
Cadmium	4	0	0	0	0.03	Greater than Maximum	ND
Chromium (Total)	9	9	6.3	18.3	11	14	14
Copper	9	9	9.9	55	24	41	41
Cyanide	9	2	3.8	45	5.7	Greater than Maximum	45
Iron	9	9	13600	37400	24433	32147	32147
Lead	9	8	3.5	140	23	Greater than Maximum	140
Manganese	9	9	56.2	2490	598	Greater than Maximum	2490
Nickel	9	4	15.8	29	14	Greater than Maximum	29
Thallium	9	3	2.4	3	1.61	2.4	2.4
Vanadium	9	9	6	22	16	Greater than Maximum	22
Zinc	9	9	31.5	115	69	103	103

[1] Includes samples SUB-4, SB-14A, SB-2B, SB-3A, SB-4A, SB-5A, SB-5B, SB-6A, SB-6B

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

ON-SITE CONSTRUCTION WORKER SCENARIO
SUMMARY STATISTICS FOR SURFACE AND SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	16	7	0.09	1.49	0.32	Greater than Max.	1.49
Ethylbenzene	16	8	0.012	29	3.2	Greater than Max.	29
Toluene	16	9	0.022	2.9	0.4	Greater than Max.	3
1,2,4-Trimethylbenzene	11	7	0.41	46	8.8	Greater than Max.	46
Xylenes (Mixed)	16	10	0.070	30	4.4	Greater than Max.	30
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	16	9	0.080	85	14	Greater than Max.	85
Acenaphthylene	16	14	0.110	30	6.3	Greater than Max.	30
Anthracene	16	12	0.25	53	7.4	Greater than Max.	53
Benzo(ghi)perylene	16	12	0.2	11	2.9	Greater than Max.	11
Fluoranthene	16	13	0.19	92	16	Greater than Max.	92
Fluorene	16	9	0.060	59	8.8	Greater than Max.	59
1-Methylnaphthalene	11	8	0.15	92	26	Greater than Max.	92
2-Methylnaphthalene	16	10	0.260	130	14	Greater than Max.	130
Naphthalene	16	12	0.060	190	23	Greater than Max.	190
Phenanthrene	16	14	0.070	160	22	Greater than Max.	160
Pyrene	16	14	0.090	150	26	Greater than Max.	150
Carcinogenic PAHs							
Benzo(a)anthracene	16	13	0.2	47	9.2	Greater than Max.	47
Benzo(a)pyrene	16	13	0.21	35	7.2	Greater than Max.	35
Benzo(b)fluoranthene	16	12	1	39	7.0	Greater than Max.	39

ON-SITE CONSTRUCTION WORKER SCENARIO
SUMMARY STATISTICS FOR SURFACE AND SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Benzo(k)fluoranthene	16	11	0.13	18	2.2	Greater than Max.	18.0
Chrysene	16	13	0.2	52	10.4	Greater than Max.	52
Dibenzo(ah)anthracene	16	1	0.15	0.15	0.11	0.14	0.14
Indeno(123-cd)pyrene	16	11	0.17	15	3.0	Greater than Max.	15
Phthalates							
bis(2-Ethylhexyl)phthalate	4	2	0.11	0.25	0.42	Greater than Max.	0.25
Other SVOCs							
Dibenzofuran	16	5	0.1	18	2.1	9.2	9.2
Inorganic Compounds							
Aluminum	16	16	2060	21400	9416	14313	14313
Arsenic	16	13	2	18	5.7	10.6	10.6
Barium	16	10	16	191	58	122	122
Beryllium	16	6	0.24	3.9	0.53	0.81	0.81
Cadmium	7	2	4.7	5.8	1.8	Greater than Max.	5.8
Chromium (Total)	15	14	1.9	32	11	18	18
Copper	16	16	6.1	84	28	49	49
Cyanide	16	7	0.69	45	5.19	21	21
Iron	16	16	4710	41800	21844	32983	32983
Lead	16	15	3.5	634	75	Greater than Max.	634
Manganese	16	16	56	2490	483	940	940
Nickel	16	10	5.6	29	14	23	23
Thallium	16	4	0.21	3	1.17	Greater than Max.	3.0
Vanadium	16	14	4.5	24	13	20	20
Zinc	16	16	15	410	110	223	223

[1] Includes samples S-1, S-2, S-3, S-4, SS-2, SS-3, SS-4, SUB-4, SB-14A, SB-2B, SB-3A, SB-4A, SB-5A, SB-5B, SB-6A, SB-6B

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

OFF-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SUBSURFACE SOIL
Between the Susquehanna River and the waste water treatment plant
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analyzed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	4	2	1.2	3.9	1.31	Greater than Maximum	3.9
Ethylbenzene	4	3	0.12	1.1	0.4	Greater than Maximum	1
Toluene	4	3	0.16	22	5.80	Greater than Maximum	22
1,2,4-Trimethylbenzene	4	3	0.33	4.4	1.4	Greater than Maximum	4
Xylenes (Mixed)	4	4	0.36	4.7	1.6	Greater than Maximum	4.7
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	4	4	0.3	76	24	Greater than Maximum	76
Acenaphthylene	4	4	1.3	87	26	Greater than Maximum	87
Anthracene	4	4	1.1	15	6	Greater than Maximum	15
Benzo(ghi)perylene	4	4	1	4.4	3.0	Greater than Maximum	4.4
Fluoranthene	4	4	3.6	19	10	Greater than Maximum	19
Fluorene	4	4	0.79	49	15	Greater than Maximum	49
1-Methylnaphthalene	4	4	0.52	67	22	Greater than Maximum	67
2-Methylnaphthalene	4	4	0.45	22	11	Greater than Maximum	22
Naphthalene	4	4	0.95	59	19	Greater than Maximum	59
Phenanthrene	4	4	3.9	62	23	Greater than Maximum	62
Pyrene	4	4	5.5	29	14	Greater than Maximum	29
Carcinogenic PAHs							
Benzo(a)anthracene	4	4	2.3	11	6	Greater than Maximum	11
Benzo(a)pyrene	4	4	3	9.3	5.6	Greater than Maximum	9.3
Benzo(b)fluoranthene	4	4	1.8	7.6	4.1	Greater than Maximum	7.6
Benzo(k)fluoranthene	4	4	0.23	6.6	2.4	Greater than Maximum	6.6
Chrysene	4	4	2.7	10	6	Greater than Maximum	10

OFF-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SUBSURFACE SOIL
Between the Susquehanna River and the waste water treatment plant
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	Number Analzyed [1]	Number Detected	Minimum (mg/kg)	Maximum (mg/kg)	Average (mg/kg)	95% Upper Confidence Limit (UCL) (mg/kg)	Reasonable Maximum Exposure Point Concentration [2] (mg/kg)
Dibenzo(ah)anthracene	4	2	0.24	0.36	0.19	Greater than Maximum	0.36
Indeno(123-cd)pyrene	4	4	0.5	6	3.2	Greater than Maximum	6
Other SVOCs							
Dibenzofuran	4	3	0.59	6.5	2.7	Greater than Maximum	6.5
Inorganic Compounds							
Aluminum	2	2	12100	16300	14200	16300	16300
Arsenic	2	2	6.7	6.9	6.8	7	7
Barium	2	2	66.1	119	93	119	119
Beryllium	2	2	0.81	1.6	1.21	1.6	1.6
Cadmium	1	0	0	0	0.12	ND	ND
Chromium (Total)	2	2	19.6	21	20	21	21
Copper	2	2	13.5	25	19	25	25
Cyanide	2	1	5.8	5.8	3.1	5.8	6
Iron	2	2	22000	25600	23800	25600	25600
Lead	2	2	19.4	38.8	29	38.8	38.8
Manganese	2	2	375	847	611	847	847
Nickel	2	1	13.5	14	11	14	14
Thallium	2	0	0	0	0.88	ND	ND
Vanadium	2	2	21.6	35	28	35	35
Zinc	2	2	43.6	75.2	59	75	75

[1] Includes samples SB-10A, SB-11A, TP-F, TP-G

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

OFF-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SUBSURFACE SOIL
Between Front St. and the railroad tracks
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Volatile Organic Compounds							
Benzene	4	2	0.09	0.22	0.12	Greater than Maximum	0.22
Ethylbenzene	4	1	11	11	2.8	Greater than Maximum	11
Toluene	4	2	0.07	1.4	0.40	Greater than Maximum	1
1,2,4-Trimethylbenzene	4	1	30	30	7.6	Greater than Maximum	30
Xylenes (Mixed)	4	3	0.08	7.5	2.0	Greater than Maximum	7.5
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	4	1	140	140	35	Greater than Maximum	140
Acenaphthylene	4	2	0.39	160	40	Greater than Maximum	160
Anthracene	4	2	0.3	99	25	Greater than Maximum	99
Benzo(ghi)perylene	4	2	2.6	7	2.4	Greater than Maximum	7
Fluoranthene	4	2	4.1	49	13	Greater than Maximum	49
Fluorene	4	1	57	57	14	Greater than Maximum	57
1-Methylnaphthalene	4	2	0.13	240	60	Greater than Maximum	240
2-Methylnaphthalene	4	2	0.18	24	6	Greater than Maximum	24
Naphthalene	4	2	0.38	41	10	Greater than Maximum	41
Phenanthrene	4	2	0.59	170	43	Greater than Maximum	170
Pyrene	4	2	5.2	83	22	Greater than Maximum	83
Carcinogenic PAHs							
Benzo(a)anthracene	4	2	3.6	47	13	Greater than Maximum	47
Benzo(a)pyrene	4	2	3.6	24	6.9	Greater than Maximum	24
Benzo(b)fluoranthene	4	2	3	21	6.0	Greater than Maximum	21
Benzo(k)fluoranthene	3	1	3.2	3.2	1.1	Greater than Maximum	3.2
Chrysene	4	2	3.2	44	12	Greater than Maximum	44

OFF-SITE CONSTRUCTION WORKER
SUMMARY STATISTICS FOR SUBSURFACE SOIL
Between Front St. and the railroad tracks
UGI Columbia Former MGP Site

	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed [1]**	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration [2] (mg/kg)**
Dibenzo(ah)anthracene	4	1	8.4	8.4	2.16	Greater than Maximum	8.40
Indeno(123-cd)pyrene	3	1	3.1	3.1	1.1	Greater than Maximum	3.1
Other SVOCs							
Dibenzofuran	4	1	15	15	3.8	Greater than Maximum	15
Inorganic Compounds							
Aluminum	3	3	3010	12300	6623	16300	16300
Arsenic	3	1	18.9	18.9	6.9	7	7
Barium	3	2	73.4	154	80	119	119
Beryllium	3	1	2.1	2.1	0.83	1.6	1.6
Cadmium	2	0	0	0	0.08	ND	ND
Chromium (Total)	3	3	5	9.8	7	21	21
Copper	3	2	5.8	68	25	25	25
Cyanide	3	1	0.98	0.98	0.5	5.8	6
Iron	3	3	9650	37200	18897	25600	25600
Lead	3	3	5	46.6	19	38.8	38.8
Manganese	3	3	114	1040	499	847	847
Nickel	3	1	16.9	17	8	14	14
Thallium	3	0	0	0	0.80	ND	ND
Vanadium	3	1	15.3	15	7	35	35
Zinc	3	3	18.3	194	79	75	75

[1] Includes samples SB-7A, SB-8A, SB-8B, TP-A

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

HYPOTHETICAL OFF-SITE RESIDENT
SUMMARY STATISTICS FOR
GROUNDWATER AND SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	GROUNDWATER SUMMARY STATISTICS					
COMPOUND [1]	**Number Analyzed** [2]	**Number Detected**	**Minimum (mg/L)**	**Maximum (mg/L)**	**Average (mg/L)**	**95% Upper Confidence Limit (UCL) (mg/L)**
Volatile Organic Compounds						
Benzene	20	10	0.021	39	3.1	Greater than Maximum
Ethylbenzene	20	7	0.011	3.5	0.37	2.046078748
Toluene	20	5	0.002	9.5	0.9	2.632986764
1,2,4-Trimethylbenzene	18	4	0.016	0.47	0.057	0.11
Xylenes (Mixed)	20	5	0.024	2.9	0.31	0.6
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthene	20	8	0.002	0.15	0.048	Greater than Maximum
Acenaphthylene	20	2	0.052	0.18	0.038	0.10
Anthracene	20	1	0.01	0.01	0.027	Greater than Maximum
Benzo(ghi)perylene	0	NA	0	0	NA	Greater than Maximum
Fluoranthene	20	0	0	0	0.027	Greater than Maximum
Fluorene	20	2	0.023	0.043	0.030	Greater than Maximum
1-Methylnaphthalene	18	6	0.037	0.75	0.11	0.35
2-Methylnaphthalene	20	3	0.099	0.94	0.10	0.39
Naphthalene	20	7	0.009	8.2	0.8	6.313288096
Phenanthrene	20	3	0.017	0.036	0.030	Greater than Maximum
Pyrene	20	0	0	0	0.027	Greater than Maximum
Carcinogenic PAHs						
Benzo(a)anthracene	20	0	0	0	0.027	Greater than Maximum
Benzo(a)pyrene	20	0	0	0	0.027	Greater than Maximum
Benzo(b)fluoranthene	20	0	0	0	0.027	Greater than Maximum
Benzo(k)fluoranthene	0	NA	0	0	NA	Greater than Maximum
Chrysene	20	0	0	0	0.027	Greater than Maximum

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	GROUNDWATER SUMMARY STATISTICS					
COMPOUND [1]	Number Analyzed [2]	Number Detected	Minimum (mg/L)	Maximum (mg/L)	Average (mg/L)	95% Upper Confidence Limit (UCL) (mg/L)
Dibenzo(ah)anthracene	0	NA	0	0	NA	Greater than Maximum
Indeno(123-cd)pyrene	0	NA	0	0	NA	Greater than Maximum
Phthalates						
bis(2-Ethylhexyl)phthalate	2	0	0	0	ND	ND
Other SVOCs						
Dibenzofuran	20	2	0.048	0.065	0.012	0.014
Inorganic Compounds						
Aluminum	20	2	0.253	0.364	0.042	0.065
Arsenic	20	0.0	0.0	0.0	0.0009	Greater than Maximum
Barium	20	2	0.427	0.541	0.09	0.12
Beryllium	20	0.0	0.0	0.0	0.0001	Greater than Maximum
Cadmium	0	NA	0.0	0.0	NA	Greater than Maximum
Chromium (Total)	20	0.0	0.0	0.0	0.0009	Greater than Maximum
Copper	20	4	0.0011	0.0042	0.0022	0.004
Cyanide	20	14	0.0052	0.223	0.062	Greater than Maximum
Iron	20	12	0.0106	24.5	3	Greater than Maximum
Lead	20	1	0.0082	0.0082	0.0012	0.0015
Manganese	20	18	0.0259	2.97	0.69	Greater than Maximum
Nickel	20	0	0	0	0.0024	Greater than Maximum
Thallium	20	0	0	0	0.0022	Greater than Maximum
Vanadium	20	0	0	0	0.0012	Greater than Maximum
Zinc	20	6	0.0026	0.143	0.024	Greater than Maximum

[1] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

[2] Includes samples MW-2R, MW-2R2, MW-5, MW-52, MW-6S, MW-6S2, MW-6D, MW-6D2, MW-7S, MW-7S2, MW-7D, MW-7D2 MW-8S, MW-8S2, MW-8D, MW-8D2, CWW-1, CWW1-1, CWW-2, CWW2-2

[3] Includes samples SB-10A, SB-11A, TP-F, TP-G

[4] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

HYPOTHETICAL OFF-SITE RESIDENT
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	SUBSURFACE SOIL SUMMARY STATISTICS						Groundwater	Subsurface Soil
COMPOUND [1]	Number Analzyed [1]	Number Detected	Minimum (mg/kg)	Maximum (mg/kg)	Average (mg/kg)	95% Upper Confidence Limit (UCL) (mg/kg)	Reasonable Maximum Exposure Point Concentration [4] (mg/L)	Reasonable Maximum Exposure Point Concentration [4] (mg/kg)
Volatile Organic Compounds								
Benzene	4	2	1.2	3.9	1.31	Greater than Maximum	39	3.9
Ethylbenzene	4	3	0.12	1.1	0.4	Greater than Maximum	2.046078748	1
Toluene	4	3	0.16	22	5.80	Greater than Maximum	2.632986764	22
1,2,4-Trimethylbenzene	4	3	0.33	4.4	1.4	Greater than Maximum	0.11	4
Xylenes (Mixed)	4	4	0.36	4.7	1.6	Greater than Maximum	0.6	4.7
Semivolatile Organic Compounds								
Non-Carcinogenic PAHs								
Acenaphthene	4	4	0.3	76	24	Greater than Maximum	0.15	76
Acenaphthylene	4	4	1.3	87	26	Greater than Maximum	0.10	87
Anthracene	4	4	1.1	15	6	Greater than Maximum	0.010	15
Benzo(ghi)perylene	4	4	1	4.4	3.0	Greater than Maximum	0.0	4.4
Fluoranthene	4	4	3.6	19	10	Greater than Maximum	ND	19
Fluorene	4	4	0.79	49	15	Greater than Maximum	0.043	49
1-Methylnaphthalene	4	4	0.52	67	22	Greater than Maximum	0.35	67
2-Methylnaphthalene	4	4	0.45	22	11	Greater than Maximum	0.39	22
Naphthalene	4	4	0.95	59	19	Greater than Maximum	6.313288096	59
Phenanthrene	4	4	3.9	62	23	Greater than Maximum	0.04	62
Pyrene	4	4	5.5	29	14	Greater than Maximum	ND	29
Carcinogenic PAHs								
Benzo(a)anthracene	4	4	2.3	11	6	Greater than Maximum	ND	11
Benzo(a)pyrene	4	4	3	9.3	5.6	Greater than Maximum	ND	9.3
Benzo(b)fluoranthene	4	4	1.8	7.6	4.1	Greater than Maximum	ND	7.6
Benzo(k)fluoranthene	4	4	0.23	6.6	2.4	Greater than Maximum	0.0	6.6
Chrysene	4	4	2.7	10	6	Greater than Maximum	ND	10

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	SUBSURFACE SOIL SUMMARY STATISTICS						Groundwater	Subsurface Soil
COMPOUND [1]	Number Analzyed [3]	Number Detected	Minimum (mg/kg)	Maximum (mg/kg)	Average (mg/kg)	95% Upper Confidence Limit (UCL) (mg/kg)	Reasonable Maximum Exposure Point Concentration [4] (mg/L)	Reasonable Maximum Exposure Point Concentration [4] (mg/kg)
Dibenzo(ah)anthracene	4	2	0.24	0.36	0.19	Greater than Maximum	0	0.36
Indeno(123-cd)pyrene	4	4	0.5	6	3.2	Greater than Maximum	0	6
Phthalates								
bis(2-Ethylhexyl)phthalate	0	NA	0	0	NA	Greater than Maximum	ND	0
Other SVOCs								
Dibenzofuran	4	3	0.59	6.5	2.7	Greater than Maximum	0.014	6.5
Inorganic Compounds								
Aluminum	2	2	12100	16300	14200	16300	0.1	16300
Arsenic	2	2	6.7	6.9	6.8	7	ND	7
Barium	2	2	66.1	119	93	119	0.12	119
Beryllium	2	2	0.81	1.6	1.21	1.6	ND	1.6
Cadmium	1	0	0	0	0.12	0.0	NA	ND
Chromium (Total)	2	2	19.6	21	20	21	ND	21
Copper	2	2	13.5	25	19	25	0.004	25
Cyanide	2	1	5.8	5.8	3.1	5.8	0.22	5.8
Iron	2	2	22000	25600	23800	25600	24.5	25600
Lead	2	2	19.4	38.8	29	38.8	0.0015	38.8
Manganese	2	2	375	847	611	847	3.0	847
Nickel	2	1	13.5	14	11	14	ND	14
Thallium	2	0	0	0	0.88	0.0	ND	1.05
Vanadium	2	2	21.6	35	28	35	ND	35
Zinc	2	2	43.6	75.2	59	75	0.1430	75.2

[1] Not all compounds were selected as
that were selected as COPCs.
[2] Includes samples MW-2R, MW-2R2,
MW-8S, MW-8S2, MW-8D, MW-8D2,
[3] Includes samples SB-10A, SB-11A,
[4] The reasonable maximum exposure

HYPOTHETICAL ON-SITE RESIDENT SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
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	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Volatile Organic Compounds							
Benzene	7	3	0.09	0.79	0.15	Greater than Max.	0.79
Ethylbenzene	7	2	0.08	0.25	0.06	Greater than Max.	0.25
Toluene	7	3	0.07	1.3	0.24	Greater than Max.	1.3
1,2,4-Trimethylbenzene	3	2	0.41	1.4	0.63	Greater than Max.	1.4
Xylenes (Mixed)	7	3	0.16	3.28	0.61	Greater than Max.	3.3
Semivolatile Organic Compounds							
Non-Carcinogenic PAHs							
Acenaphthene	7	2	0.17	0.24	0.15	Greater than Max.	0.24
Acenaphthylene	7	6	0.190	3.8	1.5	Greater than Max.	3.8
Anthracene	7	5	0.25	1.3	0.66	Greater than Max.	1.3
Benzo(ghi)perylene	7	6	0.20	9.0	3.3	Greater than Max.	9.0
Fluoranthene	7	6	0.19	6.6	2.6	Greater than Max.	6.6
Fluorene	7	3	0.060	0.47	0.18	Greater than Max.	0.47
1-Methylnaphthalene	3	2	0.38	0.76	0.40	Greater than Max.	0.76
2-Methylnaphthalene	7	5	0.260	1.0	0.59	Greater than Max.	1.0
Naphthalene	7	6	0.060	1.7	0.85	Greater than Max.	1.7
Phenanthrene	7	6	0.070	3.4	1.4	Greater than Max.	3.4
Pyrene	7	6	0.45	12	5.1	Greater than Max.	12
Carcinogenic PAHs							
Benzo(a)anthracene	7	6	0.20	4.6	2.4	Greater than Max.	4.6
Benzo(a)pyrene	7	6	0.47	6.2	3.2	Greater than Max.	6.2
Benzo(b)fluoranthene	7	5	1.1	6.7	2.5	Greater than Max.	6.7

HYPOTHETICAL ON-SITE RESIDENT SCENARIO
SUMMARY STATISTICS FOR SURFACE SOIL
UGI Columbia Former MGP Site
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	SUMMARY STATISTICS						
COMPOUND [3]	**Number Analzyed** [1]	**Number Detected**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Average (mg/kg)**	**95% Upper Confidence Limit (UCL) (mg/kg)**	**Reasonable Maximum Exposure Point Concentration** [2] **(mg/kg)**
Benzo(k)fluoranthene	7	6	0.36	6.1	2.4	Greater than Max.	6.1
Chrysene	7	6	0.20	5.5	2.7	Greater than Max.	5.5
Dibenzo(ah)anthracene	7	0	0	0	0.13	Greater than Max.	ND
Indeno(123-cd)pyrene	7	6	0.17	5.9	2.5	Greater than Max.	5.9
Phthalates							
bis(2-Ethylhexyl)phthalate	4	2	0.11	0.25	0.42	Greater than Max.	0.25
Other SVOCs							
Dibenzofuran	7	2	0.1	0.15	0.14	Greater than Max.	0.15
Inorganic Compounds							
Aluminum	7	7	2060	7920	5194	Greater than Max.	7920
Arsenic	7	7	2	10	5.6	Greater than Max.	10
Barium	7	6	16	146	65	Greater than Max.	146
Beryllium	7	4	0.240	0.54	0.27	Greater than Max.	0.54
Cadmium	7	2	4.7	5.8	1.8	Greater than Max.	5.8
Chromium (Total)	6	5	1.9	32	11	Greater than Max.	32
Copper	7	7	6.1	83.5	32.1	Greater than Max.	84
Cyanide	7	5	0.69	25	4.5	Greater than Max.	25
Iron	7	7	4710	41800	18514	Greater than Max.	41800
Lead	7	7	5.0	634	141	Greater than Max.	634
Manganese	7	7	93	807	334	Greater than Max.	807
Nickel	7	6	5.6	23	14	Greater than Max.	23
Thallium	7	1	0.21	0.21	0.62	Greater than Max.	0.21
Vanadium	7	5	4.5	24	9.9	Greater than Max.	24
Zinc	7	7	15	410	163	Greater than Max.	410

[1] Includes samples S-1, S-2, S-3, S-4, SS-2, SS-3, SS-4

[2] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

[3] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds that were selected as COPCs.

HYPOTHETICAL ON-SITE RESIDENT
SUMMARY STATISTICS FOR
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UGI Columbia Former MGP Site
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	GROUNDWATER SUMMARY STATISTICS					
COMPOUND [1]	Number Analyzed [2]	Number Detected	Minimum (mg/L)	Maximum (mg/L)	Average (mg/L)	95% Upper Confidence Limit (UCL) (mg/L)
Volatile Organic Compounds						
Benzene	5	3	9.6	23	9.3	Greater than Maximum
Ethylbenzene	5	2	3.9	4.7	2.20	Greater than Maximum
Tetrachloroethene	1	1	0.005	0.005	0.0050	Greater than Maximum
Toluene	5	3	1.5	7	2.9	Greater than Maximum
Trichloroethene	1	1	0.003	0.003	0.0030	Greater than Maximum
1,2,4-Trimethylbenzene	4	2	0.35	0.44	0.201	Greater than Maximum
Xylenes (Mixed)	5	3	1.9	3.7	1.75	Greater than Maximum
Semivolatile Organic Compounds						
Non-Carcinogenic PAHs						
Acenaphthene	5	3	0.092	0.75	0.211	Greater than Maximum
Acenaphthylene	5	3	0.051	0.49	0.134	Greater than Maximum
Anthracene	5	1	0.32	0.32	0.081	Greater than Maximum
Benzo(ghi)perylene	0	NA	0	0	NA	Greater than Maximum
Fluoranthene	5	1	0.28	0.28	0.073	Greater than Maximum
Fluorene	5	2	0.023	0.12	0.044	Greater than Maximum
1-Methylnaphthalene	4	2	0.34	0.47	0.22	Greater than Maximum
2-Methylnaphthalene	5	3	0.49	2.6	0.77	Greater than Maximum
Naphthalene	5	3	6.4	8.1	4.4	Greater than Maximum
Phenanthrene	5	3	0.016	1.2	0.264	Greater than Maximum
Pyrene	5	1	0.72	0.72	0.161	Greater than Maximum
Carcinogenic PAHs						
Benzo(a)anthracene	5	1	0.19	0.19	0.055	Greater than Maximum
Benzo(a)pyrene	5	1	0.15	0.15	0.047	Greater than Maximum
Benzo(b)fluoranthene	5	1	0.13	0.13	0.043	Greater than Maximum

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GROUNDWATER AND SUBSURFACE SOIL
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	GROUNDWATER SUMMARY STATISTICS					
COMPOUND [1]	Number Analyzed [2]	Number Detected	Minimum (mg/L)	Maximum (mg/L)	Average (mg/L)	95% Upper Confidence Limit (UCL) (mg/L)
Benzo(k)fluoranthene	0	NA	0	0	NA	Greater than Maximum
Chrysene	5	1	0.14	0.14	0.045	Greater than Maximum
Dibenzo(ah)anthracene	0	NA	0	0	NA	Greater than Maximum
Indeno(123-cd)pyrene	0	NA	0	0	NA	Greater than Maximum
Phthalates						
bis(2-Ethylhexyl)phthalate	1	1	0.069	0.069	0.069	Greater than Maximum
Other SVOCs						
Dibenzofuran	5	2	0.03	0.081	0.026	Greater than Maximum
Inorganic Compounds						
Aluminum	5	1	9.57	9.57	1.9	Greater than Maximum
Arsenic	5	0	0	0	0.0018	Greater than Maximum
Barium	5	1	0.177	0.177	0.17	Greater than Maximum
Beryllium	5	0	0	0	0.00058	Greater than Maximum
Cadmium	0	NA	0	0	NA	Greater than Maximum
Chromium (Total)	5	0	0	0	0.0017	Greater than Maximum
Copper	5	1	0.031	0.031	0.0074	Greater than Maximum
Cyanide	5	4	0.0149	0.136	0.038	Greater than Maximum
Iron	5	5	0.13	41.7	14	Greater than Maximum
Lead	5	1	0.0189	0.0189	0.0043	Greater than Maximum
Manganese	5	3	0.298	1.43	0.42	Greater than Maximum
Nickel	5	1	0.0232	0.0232	0.0065	Greater than Maximum
Thallium	5	0	0	0	0.0027	Greater than Maximum
Vanadium	5	1	0.0188	0.0188	0.0044	Greater than Maximum
Zinc	5	1	0.0772	0.0772	0.018	Greater than Maximum

[1] Not all compounds were selected as COPCs. See the risk-based screening tables and Table 12 for a summary of compounds tha

[2] Includes samples MW-3S, MW-3D, MW-3D2, MW-4, MW-9S2

[3] Includes samples SUB-4, SB-14A, SB-2B, SB-3A, SB-4A, SB-5A, SB-5B, SB-6A, SB-6B

[4] The reasonable maximum exposure point concentration is the minimum of the maximum concentration and the 95% UCL

HYPOTHETICAL ON-SITE RESIDENT
SUMMARY STATISTICS FOR
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UGI Columbia Former MGP Site
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	SUBSURFACE SOIL SUMMARY STATISTICS						Groundwater	Soil
COMPOUND [1]	Number Analzyed [3]	Number Detected	Minimum (mg/kg)	Maximum (mg/kg)	Average (mg/kg)	95% Upper Confidence Limit (UCL) (mg/kg)	Reasonable Maximum Exposure Point Concentration [4] (mg/L)	Reasonable Maximum Exposure Point Concentration [4] (mg/kg)
Volatile Organic Compounds								
Benzene	9	4	0.094	1.49	0.45	Greater than Maximum	23	1.49
Ethylbenzene	9	6	0.012	29	5.7	Greater than Maximum	4.7	29
Tetrachloroethene	0	NA	0	0	NA	Greater than Maximum	0.0050	0.0
Toluene	9	6	0.022	2.9	0.51	Greater than Maximum	7	3
Trichloroethene	0	NA	0	0	NA	Greater than Maximum	0.0030	0.00
1,2,4-Trimethylbenzene	8	5	2.9	46	11.8	Greater than Maximum	0.44	46
Xylenes (Mixed)	9	7	0.07	30	7.3	Greater than Maximum	3.7	30
Semivolatile Organic Compounds								
Non-Carcinogenic PAHs								
Acenaphthene	9	7	0.08	85	24	Greater than Maximum	0.75	85
Acenaphthylene	9	8	0.11	30	10	Greater than Maximum	0.49	30
Anthracene	9	7	0.72	53	13	Greater than Maximum	0.320	53
Benzo(ghi)perylene	9	6	0.21	11	2.5	Greater than Maximum	0.0	11
Fluoranthene	9	7	0.38	92	26	Greater than Maximum	0.280	92
Fluorene	9	6	4	59	16	Greater than Maximum	0.120	59
1-Methylnaphthalene	8	6	0.15	92	36	Greater than Maximum	0.47	92
2-Methylnaphthalene	9	5	18	130	24	Greater than Maximum	2.60	130
Naphthalene	9	6	13	190	41	Greater than Maximum	8.1	190
Phenanthrene	9	8	0.14	160	39	Greater than Maximum	1.20	160
Pyrene	9	8	0.09	150	43	Greater than Maximum	0.720	150
Carcinogenic PAHs								
Benzo(a)anthracene	9	7	0.87	47	15	Greater than Maximum	0.190	47
Benzo(a)pyrene	9	7	0.21	35	10.2	Greater than Maximum	0.150	35
Benzo(b)fluoranthene	9	7	1	39	10.5	Greater than Maximum	0.130	39

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	SUBSURFACE SOIL SUMMARY STATISTICS						Groundwater	Soil
COMPOUND [1]	Number Analyzed [3]	Number Detected	Minimum (mg/kg)	Maximum (mg/kg)	Average (mg/kg)	95% Upper Confidence Limit (UCL) (mg/kg)	Reasonable Maximum Exposure Point Concentration [4] (mg/L)	Reasonable Maximum Exposure Point Concentration [4] (mg/kg)
Benzo(k)fluoranthene	9	5	0.13	18	2.1	Greater than Maximum	0.0	18
Chrysene	9	7	3.6	52	16	Greater than Maximum	0.140	52
Dibenzo(ah)anthracene	9	1	0.15	0.15	0.10	0.1	0	0.13
Indeno(123-cd)pyrene	9	5	1.1	15	3.3	Greater than Maximum	0	15
Phthalates								
bis(2-Ethylhexyl)phthalate	0	NA	0	0	NA	Greater than Maximum	0.069	0
Other SVOCs								
Dibenzofuran	9	3	1.3	18	3.6	Greater than Maximum	0.081	18
Inorganic Compounds								
Aluminum	9	9	4760	21400	12700	20351	9.6	20351
Arsenic	9	6	3.6	18	5.8	Greater than Maximum	ND	18
Barium	9	4	53.6	191	53	139	0.18	139
Beryllium	9	2	0.54	3.9	0.74	1.9	ND	1.9
Cadmium	4	0	0	0	0.03	Greater than Maximum	NA	ND
Chromium (Total)	9	9	6.3	18.3	11	14	ND	14
Copper	9	9	9.9	55	24	41	0.031	41
Cyanide	9	2	3.8	45	5.7	Greater than Maximum	0.14	45
Iron	9	9	13600	37400	24433	32147	41.7	32147
Lead	9	8	3.5	140	23	Greater than Maximum	0.0189	140
Manganese	9	9	56.2	2490	598	Greater than Maximum	1.4	2490
Nickel	9	4	15.8	29	14	Greater than Maximum	0.0232	29
Thallium	9	3	2.4	3	1.61	2.4	ND	2.4
Vanadium	9	9	6	22	16	Greater than Maximum	0.0188	22
Zinc	9	9	31.5	115	69	103	0.077	103

[1] Not all compounds were selected asit were selected as COPCs.
[2] Includes samples MW-3S, MW-3D, I
[3] Includes samples SUB-4, SB-14A, S
[4] The reasonable maximum exposure

APPENDIX C

APPENDIX C

Modeling for Inhalation and Dermal Exposure

ORIGINAL (Red)

APPENDIX C
DERMAL EXPOSURE - ADHERENCE FACTORS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Table 6-11 Mean Soil Adherence by Activity and Body Region

Activity	N*	Body Part (mg/cm²) Hands	Arms	Legs	Face	Feet
Outdoor						
Soccer No. 1	8	0.11 0.066-0.18	0.011 0.0058-0.019	0.031 0.010-0.093	0.012 0.0083-0.016	-
Soccer No. 2	8	0.035 0.011-0.11	0.0043 0.0022-0.0083	0.014 0.0034-0.055	0.016 0.011-0.022	-
Soccer No. 3	7	0.019 0.013-0.028	0.0029 0.0014-0.0060	0.0081 0.0052-0.013	0.012 0.0078-0.018	-
Grounds Keeper No. 1	2	0.15	0.0050	-	0.0021	0.018
Grounds Keeper No. 2	5	0.090 0.040-0.24	0.0021 0.00065-0.0067	0.0012 0.00063-0.0021	0.010 0.0045-0.023	-
Grounds Keeper No. 3	7	0.030 0.014-0.065	0.0023 0.0012-0.0043	0.0009 0.00044-0.0019	0.0047 0.0021-0.010	0.0041 -
Grounds Keeper No. 4	7	0.046 0.025-0.082	0.014 0.0079-0.023	0.0008 0.00035-0.0018	0.0029 0.0018-0.0044	0.018 -
Grounds Keeper No. 5	8	0.032 0.021-0.049	0.023 0.0098-0.052	0.0010 0.0008-0.0014	0.0037 0.0019-0.0073	-
Irrigation Installers	6	0.19 0.12-0.31	0.018 0.0053-0.062	0.0054 0.0029-0.010	0.0063 0.0047-0.0086	-
Rugby Players	8	0.40 0.26-0.62	0.27 0.18-0.40	0.36 0.23-0.55	0.059 0.026-0.13	-
Farmers No. 1	4	0.41 0.20-0.84	0.059 0.0094-0.37	0.0059 0.0012-0.028	0.018 0.011-0.030	-
Farmers No. 2	6	0.47 0.33-0.69	0.13 0.056-0.29	0.057 0.0088-0.16	0.041 0.013-0.13	-
Reed Gatherers	4	0.66 0.25-1.7	0.086 0.011-0.12	0.16 0.0047-5.4	-	0.63 0.028-14
Kids-in-mud No. 1	6	35 15-84	11 1.7-73	36 18-75	-	24 6.2-93
Kids-in-mud No. 2	6	58 24-140	11 2.6-44	9.5 4.0-23	-	6.7 0.47-94
Indoor						
Tae Kwon Do	7	0.0062 0.0036-0.011	0.0019 0.0006-0.0062	0.0020 0.0011-0.0034	-	0.0024 0.0012-0.0049
Greenhouse Workers	2	0.043 -	0.0064 -	0.0015 -	0.0051 -	-

* N = number of subjects
Source: Kissel et al., 1996

[From: Kissel et al. 1996]

APPENDIX C INHALATION EXPOSURE FACTORS - VOLATILE EMISSION FACTOR
UGI Columbia Former MGP Site
Columbia, PA

					(n-θ_w)	(Table 37, EPA 1996d)	(Table 36, EPA 1996d)	(EQ 1994)	(Table 37, EPA 1996d)	$(1-\rho_b/\rho_s)$	(EPA 1991)		(Table 3, EPA 1996d)	(Table 39, EPA 1996d)	(EPA 1996d)	(EPA 1991)
					(L_{wc}/L_{soil})	(cm^2/s)	(Dimensionless)	(L_{water}/L_{soil})	(cm2/s)	(L_{pore}/L_{soil})	(g/cm3)	($K_{oc} \times f_{oc}$)	(g/m2-s per kg/m3)	(cm^3/g)	(g/g)	(sec)
			VF	D_a	θ_a	D_i[c]	H'[d]	θ_w	D_w[e]	n	ρ_b	K_d	Q/C	K_{oc} (geometric mean)[a,b]	F_{oc}	T
	Volatile Organic Compounds															
71-43-2	Benzene		2912	0.00201917	0.28	8.80E-02	2.28E-01	0.15	9.80E-06	0.43	1.5	0.372	71.87	62	0.006	9.50E+08
100-41-4	Ethylbenzene		4347	0.00090619	0.28	7.50E-02	3.23E-01	0.15	7.80E-06	0.43	1.5	1.224	71.87	204	0.006	9.50E+08
127-18-4	Tetrachloroethene		3339	0.0015355	0.28	7.20E-02	7.54E-01	0.15	8.20E-06	0.43	1.5	1.59	71.87	265	0.006	9.50E+08
108-88-3	Toluene		3721	0.0012368	0.28	8.70E-02	2.72E-01	0.15	8.60E-06	0.43	1.5	0.84	71.87	140	0.006	9.50E+08
79-01-6	Trichloroethene		2717	0.00231853	0.28	7.90E-02	4.22E-01	0.15	9.10E-06	0.43	1.5	0.5658	71.87	94.3	0.006	9.50E+08
	1,2,4-Trimethylbenzene		10821	0.0001462	0.28	0.064997	0.249	0.15	7.83E-06	0.43	1.5	5.586	71.87	931	0.006	9.50E+08
1330-20-7	Xylenes (Mixed)[a]		5265	0.00061759	0.28	7.69E-02	3.14E-01	0.15	8.44E-06	0.43	1.5	1.866	71.87	311	0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
	Semivolatile Organic Compoun		---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
	Non-Carcinogenic PAHs		---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
83-32-9	Acenaphthene		190482	4.7184E-07	0.28	4.21E-02	6.36E-03	0.15	7.69E-06	0.43	1.5	29.388	71.87	4898	0.006	9.50E+08
208-96-8	Acenaphthylene		69115	3.584E-06	0.28	0.0442592	0.0238	0.15	7.44E-06	0.43	1.5	15	71.87	2.50E+03	0.006	9.50E+08
120-12-7	Anthracene		730179	3.2111E-08	0.28	3.24E-02	2.67E-03	0.15	7.74E-06	0.43	1.5	140.958	71.87	23493	0.006	9.50E+08
191-24-2	Benzo(ghi)perylene		69916961	3.5022E-12	0.28	0.0203	2.95E-10	0.15	0.0000052	0.43	1.5	9600	71.87	1.60E+06	0.006	9.50E+08
206-44-0	Fluoranthene		2168187	3.6418E-09	0.28	3.02E-02	6.60E-04	0.15	6.35E-06	0.43	1.5	294.576	71.87	49096	0.006	9.50E+08
86-73-7	Fluorene		400076	1.0696E-07	0.28	3.63E-02	2.61E-03	0.15	7.88E-06	0.43	1.5	46.242	71.87	7707	0.006	9.50E+08
	1-Methylnaphthalene		52856	6.128E-06	0.28	0.0484	0.033	0.15	7.75E-06	0.43	1.5	13.398	71.87	2233	0.006	9.50E+08
91-57-6	2-Methylnaphthalene		46858	7.7973E-06	0.28	0.0484	0.042	0.15	7.75E-06	0.43	1.5	13.398	71.87	2233	0.006	9.50E+08
91-20-3	Naphthalene		45279	8.3505E-06	0.28	5.90E-02	1.98E-02	0.15	7.50E-06	0.43	1.5	7.146	71.87	1191	0.006	9.50E+08
85-01-8	Phenanthrene		58038	5.0825E-06	0.28	0.0333	0.248	0.15	7.47E-06	0.43	1.5	84	71.87	1.40E+04	0.006	9.50E+08
129-00-0	Pyrene		3200479	1.6714E-09	0.28	2.72E-02	4.51E-04	0.15	7.24E-06	0.43	1.5	407.952	71.87	67992	0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
	Carcinogenic PAHs		---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
56-55-3	Benzo(a)anthracene		9351579	1.9577E-10	0.28	5.10E-02	1.37E-04	0.15	9.00E-06	0.43	1.5	2145.222	71.87	357537	0.006	9.50E+08
50-32-8	Benzo(a)pyrene		24840098	2.7746E-11	0.28	4.30E-02	4.63E-05	0.15	9.00E-06	0.43	1.5	5812.644	71.87	968774	0.006	9.50E+08
205-99-2	Benzo(b)fluoranthene		4854902	7.2635E-10	0.28	2.26E-02	4.55E-03	0.15	5.56E-06	0.43	1.5	7380	71.87	**1.23E+06**	0.006	9.50E+08
207-08-9	Benzo(k)fluoranthene		40844218	1.0262E-11	0.28	2.26E-02	3.40E-05	0.15	5.56E-06	0.43	1.5	7380	71.87	**1.23E+06**	0.006	9.50E+08
218-01-9	Chrysene		2853068	2.1032E-09	0.28	2.48E-02	3.88E-03	0.15	6.21E-06	0.43	1.5	2388	71.87	**3.98E+05**	0.006	9.50E+08
53-70-3	Dibenzo(ah)anthracene		73388373	3.1787E-12	0.28	2.02E-02	6.03E-07	0.15	5.18E-06	0.43	1.5	10734.606	71.87	1789101	0.006	9.50E+08
193-39-5	Indeno(123-cd)pyrene		59366979	4.8575E-12	0.28	1.90E-02	6.56E-05	0.15	5.66E-06	0.43	1.5	20820	71.87	3.47E+06	0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
	Phthalates		---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
117-81-7	bis(2-Ethylhexyl)phthalate		19109974	4.688E-11	0.28	3.51E-02	4.18E-06	0.15	3.66E-06	0.43	1.5	666.738	71.87	111123	0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
	Other SVOCs		---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08
132-64-9	Dibenzofuran		680144	3.7009E-08	0.28	0.0269911	0	0.15	5.93E-06	0.43	1.5	0.936	71.87	156	0.006	9.50E+08
			---	---	0.28			0.15		0.43	1.5	0	71.87		0.006	9.50E+08

[a]Expressed values are for p-Xylenes

[b]Geometric mean is used except where the values are in bold; bold values are calculated.

[c]Values for acenaphthylene, benzo(ghi)perylene, 1-methylnaphthalene, 2-methylnaphthalene, phenanthrene and dibenzofurans estimated using EPA 1994 (CHEM8 Database). Values for 1,2,4-Trimethylbenzene is estimated based on the values for cumene.

[d]Values for acenaphthylene, benzo(ghi)perylene, 1-methylnaphthalene, 2-methylnaphthalene, phenanthrene and dibenzofurans are estimated using EPA 1994(CHEM8 Database). Values for 1,2,4-Trimethylbenzene are based on values in NAS 1981 (NRC-The Alkylbenzenes)

[e]Values for acenaphthylene, benzo(ghi)perylene, 1-methylnaphthalene, 2-methylnaphthalene, phenanthrene and dibenzofurans estimated using EPA 1994 (CHEM8 Database). Values for 1,2,4-Trimethylbenzene is estimated based on the values for cumene.

[f]Values for acenaphthylene, chrysene, benzo(ghi)perylene, 1,2,4-Trimethylbenzene, and dibenzofurans are based on K_{ow} values in ATSDR 1990, 1991 and calculations in EPA 1996d. Values for methylnaphthalene are based on estimates in EPA 1994 (CHEM8 Database)

JRL(NM) (Rev)

APPENDIX C
INHALATION EXPOSURE FACTORS - PARTICULATE EMISSION FACTOR
UGI Columbia
Columbia, PA

Particulate Emission Factor for Fugitive Dust

PEF (m^3/kg) = 1.04E+09

Variable	Value	Units	Source
Q/C (Dispersion)	71.87	g/m^2-s per kg/m^3	EPA 1996 (Table 3); Harrisburg - 1 Acre
V (fraction of Vegetative Cover)	0.5	unitless	EPA 1991
Um (mean annual windspeed)	4.69	m/s	EQ 1994
Ut (equivalent threshold value of windspeed)	11.32	m/s	EPA 1991
F(x) (function dependent on U_m/U_t)	0.194	unitless	EPA 1991

APPENDIX C
DERMAL EXPOSURE ADHERENCE FACTORS AND ASSUMPTIONS
UGI Columbia Former MGP Site

Columbia, PA

Trespasser

		cm2		
total skin area		15300	(50th pecentile, table 4b-3,4b-4, page 4-30 thru 4-31; EPA 1989)	
		male	female	average of combined year & category
	11-12	1.23	1.3	15314
	12-13	1.34	1.4	
	13-14	1.47	1.48	
	14-15	1.61	1.55	
	15-16	1.7	1.57	
	16-17	1.76	1.6	
	17-18	1.8	1.63	
(from table 4-3, p4-13)				
Average of 12-14 and 16-18 yrs				
		% of Total	**Multiplier (divide by 100)**	**Fractional Skin Area (cm2)**
forearms = arms		14.1	0.141	**2157**
hands		5.33	0.0533	**815**
feet		7.32	0.0732	**1120**
lower legs = legs		31.73	0.3173	**4855**
head		8.56	0.0856	**1310**
neck		NA	(Only data available for trunk = torso + neck)	

Kissel 1996 (page 121); Soccer No.1-3 (mg/cm2); 95% UCL

Hands	Arms	Legs	Faces (=Head)	Feet (=Hand)
0.18	0.019	0.093	0.016	0.18
0.11	0.0083	0.055	0.022	0.11
0.028	0.006	0.013	0.018	0.028
0.11	**0.011**	**0.054**	**0.019**	**0.11**

APPENDIX C
DERMAL EXPOSURE ADHERENCE FACTORS AND ASSUMPTIONS
UGI Columbia Former MGP Site

ORIGINAL (Red)

Industrial Worker — Columbia, PA

		cm2		
total skin area		**18150**	(50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)	
		male	female	
		1.94	1.69	
		average:	18150	
(adult; 50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)				
		(m2)	**(m2)**	**(cm2)**
		female	**male**	**average**
forearms = arms		0.23	0.291	2605
hands		0.0817	0.099	904
head		0.111	0.13	1205
neck		(Only data available for trunk = torso + neck)		
Kissel 1996 (page 121); Groundskeeper #2-5 (mg/cm2); 95% UCL				
Hands	Arms	Faces (=Head)		
0.24	0.0067	0.023		
0.065	0.0043	0.01		
0.082	0.023	0.0044		
0.05	0.052	0.007		
0.11	**0.022**	**0.011**		

Construction Worker

		cm2		
total skin area		**18150**	(50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)	
		male	female	
		1.94	1.69	
		average:	18150	
(adult; 50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)				
		female	**male**	**average**
forearms = arms		0.23	0.291	2605
hands		0.0817	0.099	904
head		0.111	0.13	1205
neck		(Only data available for trunk = torso + neck)		
Kissel 1996 (page 121); Irrigation Worker (mg/cm2)				
Hands	Arms	Faces (=Head)		
0.31	0.062	0.0086		

ORIGINAL (Red)

APPENDIX C
DERMAL EXPOSURE ADHERENCE FACTORS AND ASSUMPTIONS
UGI Columbia Former MGP Site
Columbia, PA

Resident - Child

	cm2			
total skin area	6880	(50th pecentile, table 4b-3,4b-4, page 4-30 thru 4-31; EPA 1989)		
	male	female	average of combined year & category	
2-3	0.603	0.579	6880	
3-4	0.664	0.649		
4-5	0.731	0.706		
5-6	0.793	0.779		
(from table 4-3, p4-13)				
Average of 1-5 year olds				
	% of Total	Multiplier (divide by 100)	Fractional Skin Area (cm2)	
forearms = arms	13.38	0.1338	**921**	
hands	5.61	0.0561	**386**	
feet	6.876	0.06876	**473**	
lower legs = legs	24.3	0.243	**1672**	
head	15.26	0.1526	**1050**	

Note: Per agreement with EPA, applied 1 mg/cm^2 dermal adherence rate for this receptor

Resident - teen

	cm^2			
total skin area	13118	(50th pecentile, table 4b-3,4b-4, page 4-30 thru 4-31; EPA 1989)		
	male	female	average of combined year & category	
6-7	0.866	0.843	13118	
7-8	0.936	0.917		
8-9	1	1		
9-10	1.07	1.06		
10-11	1.18	1.17		
11-12	1.23	1.3		
12-13	1.34	1.4		
13-14	1.47	1.48		
14-15	1.61	1.55		
15-16	1.7	1.57		
16-17	1.76	1.6		
17-18	1.8	1.63		
(from table 4-3, p4-13)				
Average of 6-7, 9-10, 12-14 and 16-18 yrs				
	% of Total	Multiplier (divide by 100)	Fractional Skin Area (cm2)	
forearms = arms	13.6	0.14	**1788**	
hands	5.2	0.052	**685**	
feet	7.3	0.073	**956**	
lower legs = legs	30.5	0.30	**3994**	
head	9.9	0.099	**1298**	
neck	NA			

Kissel 1996 (page 121); Soccer No.1-3 (Only data available for trunk = torso + neck)

Hands	Arms	Legs	Faces (=Head)	Feet (=Hand)
0.18	0.019	0.093	0.016	0.18
0.11	0.0083	0.055	0.022	0.11
0.028	0.006	0.013	0.018	0.028
0.11	**0.011**	**0.054**	**0.019**	**0.106**

APPENDIX C
DERMAL EXPOSURE ADHERENCE FACTORS AND ASSUMPTIONS
UGI Columbia Former MGP Site

ORIGINAL (Red)

Columbia, PA

Resident - Adult

		cm^2			
		18150	(50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)		
total skin area		male	female		
		1.94	1.69		
		average:	18150		
(adult; 50th pecentile, table 4b-1,4b-2, page 4-28 thru 4-29; EPA 1989)					
		female	**male**	**average**	
forearms		0.23	0.291	2605	
hands		0.0817	0.099	904	
head		0.111	0.13	1205	
lower legs		0.218	0.256	2370	
feet		0.114	0.131	1225	
neck					
		(Only data available for trunk = torso + neck)			
Kissel 1996 (page 121); Soccer No.1-3		(Only data available for trunk = torso + neck)			
Hands	Arms	Legs	Faces (=Head)	Feet (=Hand)	
0.18	0.019	0.093	0.016	0.18	
0.11	0.0083	0.055	0.022	0.11	
0.028	0.006	0.013	0.018	0.028	
0.11	**0.011**	**0.054**	**0.019**	**0.106**	

Resident - 6 to 30 year old

			cm2		
			15634	(50th pecentile, table 4b-3,4b-4, page 4-30 thru 4-31; EPA 1989)	
			male	**female**	
		6-7	8660	8430	**15634**
		7-8	9360	9170	
		8-9	10000	10000	
		9-10	10700	10600	
		10-11	11800	11700	
		11-12	12300	13000	
		12-13	13400	14000	
		13-14	14700	14800	
		14-15	16100	15500	
		15-16	17000	15700	
		16-17	17600	16000	
		17-18	18000	16300	
		18-19	18150	18150	
		19-20	18150	18150	
		20-21	18150	18150	
		21-22	18150	18150	
		22-23	18150	18150	
		23-24	18150	18150	
		24-25	18150	18150	
		25-26	18150	18150	
		26-27	18150	18150	
		27-28	18150	18150	
		28-29	18150	18150	
		29-30	18150	18150	
		(6-18)	(18-30)		
forearms		1788	2605	**2197**	
hands		685	904	**795**	
head		1298	1205	**1251.5**	
lower legs		3994	2370	**3182**	
feet		956	1225	**1090.5**	

APPENDIX D

TOXICITY PROFILES

VOLATILE ORGANIC COMPOUNDS

BENZENE

(Benzol, Phenyl Hydride, Coal Naphtha, Cyclohexatriene)
CAS No. 71-43-2

A. Potential Sources and Exposure

Benzene is a volatile constituent in gasoline and used as a solvent in industry. Consumer products which contain benzene include gasoline, glues, adhesives, household cleaning products, paint strippers, and cigarette smoke. Given benzene's high volatility, the most common route of exposure is inhalation of benzene vapors.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	78.11 g/mol
Water Solubility	1791 mg/L at 25°C
Vapor Pressure	95.19 mm Hg at 25°C
K_{oc}	65
K_{ow}	135
Henry's Law Constant	5.43 x 10^{-3} atm-m^3/mol at 25°C

C. Toxicity

Acute exposure to benzene may result in nausea, vomiting, ataxia, and excitement. Benzene depresses central nervous system function. Narcotic effects, similar to those produced by toluene "glue sniffing," may occur. Chronic exposure to benzene is known to cause adverse hematological effects (ATSDR, 1989).

Several epidemiological studies have associated occupational benzene exposure with an increased incidence of leukemia (USEPA, 1984). As a result, EPA classifies benzene as a Group A - known human carcinogen. Until recently, studies in animals did not conclusively support the evidence that benzene is a human leukemogen. For instance, IARC (1982) concluded that there is only limited evidence that benzene is carcinogenic in experimental animals. However, a recent study (NTP, 1984) found that "under the conditions of these studies, there was clear evidence of carcinogenicity of benzene" for F344/N rats and B6C3F1 mice of both sexes.

Based on epidemiological studies, the USEPA estimated unit cancer risks for benzene of $2.9x10^{-2}$ $(mg/kg/day)^{-1}$ for the oral and inhalation routes of exposure (IRIS, 1992). There is

no inhalation RfD available for benzene although it has been demonstrated that exposure may result in systemic effects as well as carcinogenicity. Development of an inhalation RfD is under review by an EPA work group (IRIS, 1992).

D. Toxicokinetics

Benzene is readily absorbed through ingestion, moderately absorbed through inhalation, and poorly through intact skin. Once in the bloodstream, benzene is distributed throughout the body, with the concentration in any one compartment dependent on the degree of blood perfusion of tissue. Highly perfused tissues such as kidney, liver, and bone marrow can accumulate benzene. Since benzene is lipid-soluble, it accumulates in adipose tissue, but the rate of accumulation is slow since fat is poorly perfused.

The metabolites of benzene which include phenol, catechol, hydroquinone and conjugated phenolic compounds, are responsible for benzene's toxic effects. The primary site of benzene metabolism is the liver via the cytochrome P450 mixed function oxidase system. Some benzene metabolism may also occur in the bone marrow via the same enzyme system. However, most absorbed benzene is excreted unchanged in the expired air. Sulfate and glucuronide conjugation occur, allowing its metabolites to be excreted in the urine (USEPA, 1980).

Several studies have indicated that approximately 50% of the airborne benzene is absorbed after inhalation exposure (Teisinger et. al., 1952, as cited in NIOSH, 1974; Srbova et. at., 1950; Hunter, 1966; Nomiyama and Nomiyama, 1974, as cited in Johnson, 1979). For dermal absorption, the controlling factor is contact time with the skin. One study (Susten et al., 1990) estimated the non-occluded dermal absorption of benzene in hairless mice to be approximately 1% of the applied dose in 4 hours (or 6% in 24 hours), with volatilization occurring rapidly. The oral absorption efficiency of pure benzene is estimated to be essentially 100% (Parke and Williams, 1953, Sabourin et al., 1986, Turkall et al., 1988).

E. Ecological Effects

Acute toxicity data are available for two freshwater invertebrate species and six fish species (USEPA, 1980). The 48-hour effect concentrations ranged between 203,000 ug/L and 620,000 ug/L for invertebrates. The 96-hour test results for fish species range from 5,300 ug/L (rainbow trout) to 386,000 ug/L (mosquito fish). No bioconcentration factor is available for benzene.

For saltwater species, acute values generally range between 10,900 ug/L and 924,000 ug/L (USEPA, 1980). Fish are generally more sensitive than marine invertebrates. Striped bass exhibited LC_{50} values as low as 5,100 ug/L.

Studies have been conducted in which marine organisms were exposed to the water-soluble

fraction (WSF) of oil and these data have been useful in evaluating the toxicity of light aromatic hydrocarbons as a group (the WSF contains mainly benzene, xylene, and toluene). In terms of total light aromatics, the 96-hour LC_{50} values ranged between 1,000-3,000 ug/L for marine species (USEPA, 1980). Chronic effects in marine species for benzene include survival reduction and stress at 700 ug/L in Pacific herring (USEPA, 1980).

It should be noted that based on studies of the effects of oil on aquatic/marine organisms, chronic or sublethal toxic effects can occur at low concentrations (e.g., on the order of a few ug/L). The studies reviewed do not focus on specific hydrocarbon compounds but on classes of compounds (e.g., soluble hydrocarbons). However, they do provide some insights into the potential for sublethal and chronic effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

• OSHA Permissible Exposure Limit (PEL)	10 ppm
Ceiling	25 ppm
Acceptable peak (during 8 hour period)	50 ppm
• ACGIH Threshold Limit Value-Time Weighted Average	10 ppm (32 mg/m^3)

Drinking Water:

• USEPA Maximum Contaminant Level (MCL)	5 ug/L
• USEPA Maximum Contaminant Level Goal (MCLG) (for carcinogens)	0 mg/L
• Drinking Water Health Advisories:	
Ten-day Health Advisory	0.235 mg/L

Water:

• Ambient Water Quality Criteria, Freshwater:	
Acute	5300 ug/L
• Ambient Water Quality Criteria, Marine:	
Acute	5100 ug/L
Chronic	700 ug/L

G. Toxicity Factors

Cancer Potency Factor: 2.9×10^{-2} $(mg/kg/day)^{-1}$ for the oral and inhalation routes of exposure (IRIS, 1992).

RfDs currently not available.
Various state guidelines may differ from federal regulations and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological profile for benzene. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

International Agency for Research on Cancer (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 29: Some industrial chemicals and dyestuffs. Lyon, France: World Health Organization.

Johnson, H.L. 1979. Systemic Absorption of Benzene. Evaluation of Experimental Data and Comparison of Dermal and Inhalation Exposure. (Submitted to OSHA as Exhibit Number 231-4).

National Institute for Occupational Safety and Health (NIOSH). 1974. Criteria for a Recommended Standard: Occupational Exposure to Benzene. p. 26.

National Toxicology Program (NTP). 1984. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Draft Report. NTP-TR-289. NIH Publication No. 84-2545. NTP-84-072.

Parke, D.V. and Williams, R.T. 1953. Studies in Detoxication. 49. The Metabolism of Benzene Containing [^{14} C]-Benzene. *Biochem. J.* 54:231-238.

Sabourin, P., et al. 1986. Effect of Dose on Absorption and Excretion of [^{14} C]-Benzene Administered Orally or by Inhalation. *The Toxicologist.* 6:163.

Susten, A.S. et al. 1990. In Vivo Percutaneous Absorption Studies of Volatile Organic Solvents in Hairless Mice II. Toluene, Ethylbenzene and Aniline. *J. Appl. Toxicol.* 10:217-225.

Turkall, R.M. 1988. Soil Absorption Alters Kinetics and Bioavailability of Benzene in Orally Exposed Male Rats. *Arch. Environ. Contam. Toxicol.* 17:159-164.

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for

Benzene. EPA 440/5-80-018. Washington, D.C.: U.S.Environmental Protection Agency.

U.S. Environmental Protection Agency (USEPA). 1984. Health Effects Assessment for Benzene. EPA/540/1/86/037. Washington, D.C.: U.S. Environmental Protection Agency.

ETHYLBENZENE

(Ethylbenzol, Phenylethane)
CAS No. 100-41-4

A. Potential Sources and Exposure

Ethylbenzene is a colorless solvent that smells like gasoline. It is a component of automotive and aviation gasoline. Significant quantities of ethylbenzene are present in mixed xylenes which are frequently used in paints, agricultural sprays and gasoline blends. Individuals may be exposed to ethylbenzene by inhaling its vapors emitted at filling stations or in occupational settings where petroleum products or by-products are manufactured, or during high atmospheric smog generated by motor vehicles.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	106.16 g/mol
Water Solubility	152 mg/L at 25°C
Vapor Pressure	9.35 mm Hg at 25°C
K_{oc}	871
log K_{ow}	1.4
Henry's Law Constant	8.44 x 10^{-3} atm-m^3/mol at 20°C

C. Toxicity

Humans exposed to low levels of ethylbenzene in air for short periods of time may experience eye and throat irritation. Exposure to higher levels may cause more severe effects such as central nervous system depression, decreased movement and dizziness, and mucous membrane irritation. USEPA has estimated an oral subchronic RfD of 1.0 mg/kg-day for this chemical. The oral RfD was derived from a subchronic 182-day oral bioassay with rats. The critical toxic effects which were noted in the study include histological changes in the liver and kidney. A chronic RfD value of 1 x 10^{-1} mg/kg-day has been estimated via the oral route. An inhalation RfD of 1 x 10^{-1} mg/kg-day was derived from a subchronic developmental study using rats and rabbits. The rats and rabbits were exposed to concentrations of 0, 100, or 1000 ppm (434 or 4342 mg/m^3) for 6-7 hours/day, 7 days/week during days 1-19 and 1-24 days of gestation. Skeletal effects in rats were observed at both 434 and 4342 mg/m^3. Slightly reduced litter size in rabbits occurred at 4342 mg/m^3. Both adverse effects were used to determine a LOAEL of 1000 ppm. An uncertainty factor of 300 was used to derive the inhalation RfD.

Ethylbenzene is not classifiable as to human or animal carcinogenicity (Group D) due to a

lack of appropriate animal bioassays and human studies.

D. Toxicokinetics

Ethylbenzene is readily absorbed via inhalation, ingestion, or dermal absorption. Following exposure, ethylbenzene is distributed throughout the body, with the highest levels detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al., 1980). Ethylbenzene undergoes a variety of microsomally-mediated side-chain hydroxylations to yield the major metabolites, mandelic acid and phenylglyoxylic acid (Engstrom et. al., 1984). The oxidation products are conjugated following urinary excretion which appears to within 2 days of exposure (ATSDR, 1990).

E. Ecological Effects

The available data for ethylbenzene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 32,000 ug/L and for marine life at 430 ug/L. Effects would occur at lower concentrations among freshwater and marine species that are more sensitive than those tested. No definitive data are available concerning the chronic toxicity of ethylbenzene to sensitive freshwater life. Data concerning the toxicity of ethylbenzene to domestic animals or terrestrial wildlife were not available.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL) 100 ppm (435 mg/m^3)
  Short Term Exposure Limit (STEL) 125 ppm (545 mg/m^3)

Drinking Water:

- USEPA Maximum Contaminant Level (MCL) 0.7 mg/L
- USEPA Maximum Contaminant Level Goal(MCLG) 0.68 mg/L
- Drinking Water Health Advisories:
  - Drinking Water Equivalent Level (DWEL) 3.4 mg/L
  - One-day Health Advisory 32 mg/L
  - Ten-day Health Advisory 3.2 mg/L
  - Long-term Health Advisory 0.97 mg/L
  - Lifetime Health Advisory 0.68 mg/L

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute 32,000 ug/L

Chronic	No data available

- Ambient Water Quality Criteria, Marine:

Acute	430 ug/L
Chronic	No data available

G. Toxicity Factors

Cancer Slope Factors:

USEPA has not derived cancer slope factors for this non-carcinogenic compound.

Reference Doses (mg/kg-day):

	Subchronic	Chronic
Oral	1.0	0.1
Inhalation	1.0	1.0

Various state guidelines may differ from federal regulations and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological profile for ethylbenzene. U.S. Department of Health and Human Services.

Chin, B.J. et. al., 1980. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methylethylbenzene isomers in rats. *Bull. Env. Contam. Toxicol.* 24:477-483.

Engstrom, K. et al., 1984. Urinary disposition of ethylbenzene and m-xylene in man following separate and combined exposure. *Xenobiotics* 15:281-286.

Integrated Risk Information System, (IRIS). 1992. on-line database.

U.S. Environmental Protection Agency (USEPA). 1980. Ambient water quality for ethylbenzene. USEPA 440/5-80-048. Washington, D.C.: U.S. Environmental Protection Agency.

TETRACHLOROETHYLENE

GENERAL BACKGROUND INFORMATION

The major use for tetrachloroethylene (perchloroethylene, PCE) is in the dry-cleaning industry. Its popularity in this area is due to its nonflammability, ease of recovery for reuse and its compatibility with various fabrics. It is also used in cold cleaning and vapor degreasing of metals. Its remaining uses are as a chemical intermediate in the synthesis of fluorocarbons, various manufacturing and industrial processes as well as medicinal uses (IRP, 1985).

PHARMACOKINETICS

PCE is readily absorbed by humans through the lungs into the blood. Pulmonary uptake is proportional to ventilation rate, duration of exposure and (at lower concentrations of PCE) to the concentration of PCE in the inspired air (Hake and Stewart, 1977). PCE is also rapidly aborbed following oral administration, but is poorly absorbed following dermal exposure (see section on Relative Absorption Factors). Distribution occurs rapidly with the highest concentrations of PCE achieved in tissues of high fat content (ATSDR, 1990). Metabolism of PCE is believed to be mediated by the microsomal mixed function oxidase enzyme system involving the formation of an epoxide intermediate. Major metabolites of PCE are trichloroacetic acid and trichloroethanol. Unmetabolized PCE is excreted largely by exhalation with urinary excretion of metabolites representing a small percentage (ATSDR, 1990).

HUMAN TOXICOLOGICAL PROFILE

Stewart et al. (1977) found that exposure of 11 subjects to a mean PCE concentration of 101 ppm for 7 hours produced symptoms of headache, dizziness, difficulty in speaking, and sleepiness. Long-term exposed subjects are also reported to experience effects such as short-term memory defects, ataxia, irritability, disorientation, and sleep disturbances (USEPA, 1985). PCE causes hepatotoxicity in humans. A number of reports of liver damage after inhalation of PCE in acute or chronic exposure situations have been documented (Hake and Stewart, 1977). PCE ingestion in humans results in symptoms indicative of liver damage, including elevated SGOT and SGPT levels, hepatomegaly, and fatty degeneration of the liver cells (Koppel et al., 1985).

Male and female rats treated via stomach tube showed symptoms of tremors, ataxia, CNS depression, and finally, death (Hayes et al., 1986). Moderate fatty degeneration of the liver was observed in mice 1 day after a single 4-hour exposure to 200 ppm PCE, but not 3 days after exposure (Kylin et al., 1963). Chronic exposure in animals has been found to damage the CNS, producing symptoms such as hypertrophy and proliferation of astroglial cells in the brain (Rosengren et al., 1986). In this study, there was a decreased DNA content observed in the brain of gerbils exposed continuously to PCE concentrations as low as 60 ppm. It was suggested that this may represent the development of brain atrophy. Rowe et al. (1952) exposed rats, rabbits, guinea pigs, and monkeys to PCE vapors at levels of 100 to 400 ppm, 7 hour/day, 5 days/week for 6 months. Only guinea pigs showed adverse effects due to exposure. These effects included increased liver weights with some fatty degeneration, a slight increase in hepatic lipid content, and the presence of several small hepatic fat vacuoles. PCE also causes renal effects in rodents. Groups of rats and mice of each sex were exposed to PCE in corn oil by gavage 5 days/week for 78 weeks (NCI, 1977). Toxic nephropathy occurred at all dose levels in both sexes of rats and mice. PCE has been found to be fetotoxic, but not teratogenic at concentrations that are also maternally toxic (Schwetz, 1975). Fetotoxicity was usually expressed by decreased fetal weight and delayed skeletal ossification. There is some evidence that PCE causes adverse effects on reproductive systems. The finding of abnormal sperm in mice exposed to 500 ppm PCE is an indication of chemical effects on the sperm. However, definitive evidence that PCE or its metabolites reached germinal tissue and damaged DNA is not provided (U.S. EPA, 1985).

GENOTOXICITY

In vitro studies of PCE genotoxicity have been performed in prokaryotic, eukaryotic and mammalian cells. The results using prokaryotic systems were all negative, whereas in studies using yeast or mammalian cells, the results were mixed (Bronzetti et al., 1983; Price et al., 1978). NTP (1985) conducted inhalation carcinogenicity studies in F344/N rats and B6C3F1 mice of each sex for 6 hours/day, 5 days/week for 103 weeks. There were increases in mononuclear cell leukemia in rats and hepatocellular adenomas and carcinomas in mice. In chronic oral studies (NCI, 1977), PCE produced hepatocellular carcinomas in mice, but not in rats.

Epidemiological studies of dry-cleaning and laundry workers have determined significant excesses in mortality due to cancers of the lung, cervix, kidney, skin and colon (Blair et al., 1979; Kaplan, 1980). Although these studies suggest an association between chronic occupational exposure to PCE and increased cancer risk, the evidence is inconclusive, because workers were exposed to other solvents as well. Considering the inconclusive evidence for

carcinogenicity in humans, the U.S. EPA places PCE in Group B2, meaning that is considered a probable human carcinogen.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR) (1990) Toxicological profile for tetrachloroethylene. U.S. Public Health Service.

Blair, A., Decoufle, P. and Grauman, D. (1979) *Causes of death among laundry and dry cleaning workers*. Am. J. Pub. Health 69:508-511.

Bronzetti, G.C., et al. (1983) *Genetic and biochemical studies on perchloroethylene in vitro and in vivo*. Mutat. Res. 116:323-331.

Hake, C.L. and Stewart, R.D. (1977) *Human exposure to tetrachloroethylene: inhalation and skin contact*. Environ. Health Persp. 21:231-238.

Hayes, J.R., Condie, L.W. and Borzelleca, J.F. (1986) *The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats*. Fund. Appl. Toxicol. 7:119-125.

The Installation Restoration Program Toxicology Guide (IRP). Vol 1. (1985) Arthur D. Little, Inc. Cambridge, MA.

Kaplan, S.D. (1980) Dry-Cleaning workers exposed to perchloroethylene. A retrospective cohort mortality study. Cincinnati, OH.

Koppel, C., Arndt, I., Arendt, U. and Koeppe, P. (1985) *Acute tetrachloroethylene poisoning: Blood elimination kinetics during hyperventilation therapy*. J. Toxicol. Clin. Toxicol. 23:103-116.

Kylin, B., Reichard, H., Sumegi, I. and Ylhner, S. (1963) *Hepatotoxicity of inhaled trichloroethylene, tetrachloroethylene, and chloroform -single exposure*. Acta. Pharmacol. Toxicol. 20:16-26.

National Cancer Institute (NCI) (1977) Bioassay of tetrachloroethylene for possible carcinogenicity. DHEW Publication No. (NIH) 77-813.

National Toxicology Program (NTP) (1986) Toxicology and carcinogenesis of tetrachloroethylene (perchloroethylene) in F344/N rats and B6C3F1 mice (inhalation studies). NTP Technical Report No. 311. DHHS Publication No. (NIH) 86-2567.

Price, P.J., Hassett, C.M. and Mansfield, J.I. (1978) *Transforming activities of trichloroethylene and proposed industrial alternatives*. In Vitro 14(3):290-293.

Rosengren, L.E., Kjellstrand, P. and Haglid, K.G.(1986) *Tetrachloroethylene: levels of DNA and S-100 in the gerbil CNS after chronic exposure*. Neurobehav. Toxicol. Teratol. 8:201-206.

Rowe, V.K., McCollister, D.D., Spencer, H.C., Adams, E.M. and Irish, D.D. (1952) *Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects*. AMA Arch. Ind. Hyg. Occup. Med. 5:566-579.

Schwetz, B.A., Leong, B.K.J. and Gehring, P.J. (1975) *The effect of maternally inhaled trichloroethylene, perchloroethylene, methylchloroform, and methylene chloride on embryonal and fetal development in mice and rats*. Toxicol. Appl. Pharmacol. 32:84-96.

Stewart, R.D., Hake, C.L. and Wu, A. (1977) Effects of perchloroethylene/drug interaction on behavior and neurological function. Final Report.

U.S. Environmental Protection Agency (U.S. EPA) (1985) Health assessment document for tetrachloroethylene (perchloroethylene). Final Report. Washington, DC.

TOLUENE

(Methylbenzene, Methylbenzol, Phenylmethane, Toluol)
CAS No. 108-88-3

A. Potential Exposure

Toluene is a clear, volatile, colorless liquid with a sweet odor and is used in industry in the refining of gasoline, chemical manufacturing, and manufacture of paints, lacquers, adhesives, rubber and in leather tanning processes. Humans may be exposed to toluene from drinking water, food, air, occupational settings, and consumer products. Toluene exposure in humans occurs primarily through breathing the chemical in the workplace or during deliberate glue sniffing or solvent abuse. Toluene vapors are emitted from automobile exhausts, paints, gasoline, rubber cement and adhesives.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	92.1 g/mol
Water Solubility	534.8 mg/l at 25°C
Vapor Pressure	28.4 mm Hg at 25°C
Koc	300
log Kow	2.73
Henry's Law constant	5.94×10^{-3} atm-m^3/mol

C. Toxicity

In humans, acute exposure to high levels of toluene vapors may cause central nervous system (CNS) depression (ATSDR, 1989). Acute exposures may result in reversible depression of the CNS, neurological dysfunction, impaired performance, and narcosis. Chronic exposure has been reported to result in permanent CNS effects such as ataxia, tremors, and impaired speech, hearing and vision (ATSDR, 1989). Inhalation of 200-300 ppm lasting from 1-10 years may cause loss of coordination, impaired memory and thinking ability. Liver, kidney, cardiovascular, immunological and respiratory disturbances have been noted at high doses or prolonged exposures (ATSDR, 1989). The oral reference dose (RfD) for toluene is 0.2 mg/kg/day, based on the observed changes in liver and kidney weights in rats that received subchronic exposure to toluene by gavage (IRIS, 1992).

Toluene has been tested for carcinogenicity in a number of dermal application and inhalation studies using experimental animals. The lack of positive mutagenic and

carcinogenic animal data and no human data regarding carcinogenicity classifies toluene in Group D - Not Classifiable as to Carcinogenicity (IRIS, 1992).

D. Pharmacokinetics

Toluene is readily absorbed after inhalation or oral exposure and to a lesser extent via dermal contact (ATSDR, 1989). No studies evaluating toluene absorption after oral exposure have been reported (ATSDR, 1989).

Toluene is distributed into the tissues of the body according to their lipid content and blood perfusion: the greater the lipid content or blood perfusion of body tissue, the greater the amount of toluene deposited and retained there. The relative concentration of toluene in body tissues is as follows: adipose > bone marrow > brain, liver > lung, kidney, heart, muscle (ATSDR, 1989).

Toluene is metabolized by the mixed function oxidase system to benzyl alcohol, which is subsequently oxidized to benzaldehyde and benzoic acid. Benzoic acid is then conjugated to glycine to form hippuric acid (ATSDR, 1989). Inducers of the mixed function oxidase system decrease toluene toxicity whereas inhibitors enhance toluene toxicity. This suggests that toxicity is due to toluene directly and not to its metabolite(s).

Toluene is eliminated primarily via urinary excretion of its major metabolite, hippuric acid. After inhalation exposure to toluene, a large portion of the absorbed toluene is also exhaled (ATSDR, 1989).

E. Environmental Effects

Toluene is highly volatile and only slightly soluble in seawater. The LC_{50}s for toluene in marine invertebrates ranged from 3,700 ug/l using bay shrimp to 1,050,000 ug/l for the Pacific oyster (USEPA, 1980). In the admittedly limited data base, marine fish also exhibited a wide range of sensitivities. Striped bass had a LC_{50} value of 6,300 ug/l while sheepshead minnow were very resistant with LC_{50}s between 277,000 and 485,000 ug/l. However, fish exposed to toluene can eliminate it through their gills relatively rapidly upon exposure to uncontaminated water (Thomas and Rice, 1981). Potera (1975) examined the relationship of the 24-hour LC_{50} and the variables, temperature, salinity, and life stage of the grass shrimp. His data indicate that the LC_{50} value is not particularly sensitive to these variables.

Sheepshead minnow exhibited chronic effects on hatching and survival of embryo-larvae at 5,000 ug/l and represents the marine chronic criterion value (USEPA, 1980). The response of plants to toluene was variable even within species. Skeletonema costatum, a

common estuarine diatom, exhibited effects on growth or photosynthesis at 8,000 ug/l and at concentrations greater than 433,000 ug/l (USEPA, 1980). Kelp have exhibited effects on photosynthesis at 10,000 ug/l (USEPA, 1980).

Of five fresh water organisms (including four fish species) tested with toluene, the cladoceran, *Daphnia magna*, was most resistant to any acute effects. The EC_{50} and LC_{50} values ranged from 12,700 ug/l to 313,000 ug/l. The species acute toxicity value of 17,500 ug/l for bluegill from static 96-hour static LC_{50} was selected as the freshwater acute criterion. No chronic tests were available for freshwater species. The freshwater alga tested were relatively insensitive to toluene with EC_{50} values of 245,000 ug/l or greater.

F. Federal Regulations, Standards and Guidelines

Air:

- OSHA Permissible Exposure Limit (PEL) 100 ppm (375 mg/m^3)
  STEL 150 ppm (560 mg/m^3)

- NIOSH Recommended Exposure Limit (REL) 100 ppm (375 mg/m^3)

Drinking Water:

- USEPA Maximum Contaminant Level (MCL) 2.0 mg/l

- USEPA Maximum Contaminant Level Goal (MCLG) 1.0 mg/l

- USEPA Secondary Maximum Contaminant Level 0.04 mg/l (proposed)

- Drinking Water Health Advisories:
  - One-day Health Advisory (child) 20 mg/l
  - Ten-day Health Advisory (child) 2 mg/l
  - Longer-term Health Advisory (child) 3 mg/l
  - Longer-term Health Advisory (adult) 10 mg/l
  - Drinking Water Equivalent Level (DWEL) 7 mg/l
  - Lifetime Health Advisory 1.0 mg/l

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute 17,500 ug/l
  - Chronic No Data Available

- Ambient Water Quality Criteria, Marine:

Acute	6,300 ug/l
Chronic	5,000 ug/l

G. Toxicity Factors

Cancer Potency Factors:

Cancer Potency Factors not derived for this non-carcinogenic compound.

Reference Doses (mg/kg-day):

	Subchronic	Chronic
Oral	2.0	0.2
Inhalation	2.0	2.0

Various state guidelines may differ from federal regulations and should be consulted.

H. Massachusetts Standards & Guidelines

Air:

- Allowable Ambient Limit (AAL) 2.72 ppb (10.24 ug/m^3)
- Threshold Effects Limit (TEL 24 hr) 2.72 ppb (10.24 ug/m^3)
- Allowable Threshold Concentration 14 ppb (51 ug/m^3)

Drinking Water: 1.0 mg/l

Groundwater: 1.0 mg/l

I. References

Agency for Toxic Substances and Disease Registry. (ATSDR) 1989. Toxicological profile for Toluene. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database, accessed 1/13/92.

Potera, F.T. 1975. The effects of benzene, toluene, and ethylbenzene on several important members of the estuarine ecosystem. Ph. D. dissertation, Lehigh University.

Thomas, R.E. and S.D. Rice. 1981. Excretion of aromatic hydrocarbons and their metabolites by fresh water and salt water Dolly Varden char. In: Biological Monitoring of Marine Pollutants. pp. 425-448. Academic Press, NY.

U.S. Environmental Protection Agency, (USEPA). 1980. Ambient Water Quality Criteria for Toluene. Washington, D.C.

TRICHLOROETHYLENE

GENERAL BACKGROUND INFORMATION

Trichloroethylene (TCE) is widely used as an industrial solvent, particularly in metal degreasing, which consumes about 90% of TCE produced annually in the U.S. TCE is also used for dry-cleaning, as a low-temperature heat exchange fluid, as a fumigant, as a diluent in paints and adhesives, in aerospace operations, and in textile processing. Previously, TCE was used as an extractant in food-processing. These uses were discontinued in 1975 due to evidence of possible carcinogenic activity. Its earlier use in anesthetics was also discontinued (IRP, 1985).

PHARMACOKINETICS

Absorption of TCE from the gastrointestinal and respiratory tracts is extensive. TCE is extensively metabolized in humans to trichloroethanol, trichloroethanol glucuronide, and trichloroacetic acid. Although the liver is the primary site of TCE metabolism, there is evidence for extrahepatic metabolism in the lungs and kidneys (ATSDR, 1988).

HUMAN TOXICOLOGICAL PROFILE

TCE is assumed to be responsible for the deaths of four men employed at degreasing operations using TCE as the solvent (Kleinfeld and Tabershaw, 1954). Toxicological analysis revealed TCE in varying concentrations in various tissues. Kleinfeld and Tabershaw (1954) reported that, despite treatment, a man died 11 days after he accidentally drank an unknown quantity of TCE. TCE has been shown to affect the central nervous system. Short-term exposure to high concentrations of TCE caused dizziness, headache, nausea, confusion, facial numbness, blurred vision, and, at very high levels, unconsciousness. Longer exposures cause ataxia, decreased appetite, sleep disturbances, and trigeminal neuropathy (U.S. EPA, 1985). Information regarding hepatotoxicity in humans is limited and derived from acute overexposures. U.S. EPA (1985) has concluded that it is unlikely that chronic exposure to trichloroethylene at concentrations found or expected in ambient air would result in liver damage.

MAMMALIAN TOXICOLOGICAL PROFILE

In laboratory animals, the acute toxicity of trichloroethylene is low. Oral LD_{50} values of 4920 mg/kg in the rat, 3200 mg/kg in the mouse and 2800 mg/kg in the dog have been reported. In a study by Baker (1958), several dogs died within 20 minutes of being exposed to TCE at 30,000 ppm. Rats exposed to 20,000 ppm for 5 hours died (Adams, 1951). A 2-year study

in rats conducted by the NTP (1986a) showed decreased survival due to TCE treatment. Deaths were attributed to toxic nephrosis. Behavioral changes were observed in rats at TCE vapor concentrations as low as 100 ppm (Silverman and Williams, 1975). Liver enlargement is the most commonly observed hepatic effect seen in TCE-exposed animals (Kjellstrand et al., 1983). Mice, especially males, appear to be particularly sensitive to the hepatotoxic effects of TCE. The only reproductive effects observed were reduced testis and epididymis weights in rats exposed to dietary TCE (NTP, 1986b). There were no effects of reproductive system histology, fertility, or other reproductive performance parameters in treated males or females in these studies.

GENOTOXICITY

Perocco and Prodi (1981) found positive results for unscheduled DNA synthesis both with and without metabolic activation in human lymphocytes in vivo. Another study reported a significant increase in sister chromatid exchange in six workers exposed to TCE (Gu et al., 1981). In vitro mutagenicity tests in bacteria, yeasts, and molds demonstrated weak positive responses. Most of these tests required metabolic activation of the compound (Crebelli et al., 1985). TCE has been shown to be carcinogenic in animals. Inhalation and oral exposure produced liver and lung tumors in mice and kidney adenocarcinomas, testicular Leydig cell tumors, and possibly leukemia in rats. These studies are deemed sufficient to place TCE in CAG classification B2, probable human carcinogen (U.S. EPA, 1987). Further support that TCE is a probable human carcinogen comes from studies that indicate that metabolism is qualitatively similar in humans and test animals (U.S. EPA, 1987). The available carcinogenicity studies indicate that mice are more susceptible to TCE carcinogenicity than the rat. Factors contributing to this difference may be an increased rate of metabolic conversion to trichloracetic acid in mice, and the more pronounced trichloroacetic acid-mediated peroxisomal proliferation and cell proliferation in mice (Elcombe et al., 1985). The peroxisomal proliferation may lead to an increase in the reactive oxygen species and DNA damage, which may lead to hepatocellular carcinoma.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR) (1988) Toxicological profile for trichloroethylene. U.S. Public Health Service.

Adams, E.M., Spencer, H.C., Rowe, V.K., McCollister, D.D. and Irish, D.D. (1951) *Vapor toxicity of trichloroethylene determined by experiments on laboratory animals*. Arch. Ind. Hyg. Occup. Med. 4:469-481.

Baker, A.B. (1958) *The nervous system in trichloroethylene. An experimental study*. J. Neuropath. Exp. Med. 17:649-655.

Crebelli, R., Conti, G., Conti, L. and Caurere, A. (1985) *Mutagenicity of trichloroethylene, trichloroethanol, and chloral hydrate in Aspergillus nidulans*. Mutat. Res. 156:105-111.

Elcombe, C.R., Rose, M.S. and Pratt, I.S. (1985) *Biochemical, histological and ultrastructural changes in rat and mouse liver following the administration of trichloroethylene: Possible relevance to species differences in hepatocarcinogenicity.* Toxicol. Appl. Pharmacol. 79:365-376.

Gu, Z.W., Sele, B. and Jalbert, P. (1981) *Induction d'echanges entre les chromatides soeurs (SCE) par le trichloroethylene et ses metabolites.* Toxicol. Eur. Res. 3:63-67.

The Installation Restoration Program Toxicology Guide (IRP). 1986. Vol. 1. Arthur D. Little, Inc., Cambridge, MA.

Kjellstrand, P., Holmquist, B., Alm, P., Kanje, M., Romare, S., Jonsson, I., Mannson, L. and Bjerkamo, M. (1983) *Trichloroethylene: Further studies of the effects on body and organ weights and plasma butyryl cholinesterase activity in mice.* Acta Pharmacol. Toxicol. 53:375-384.

Kleinfeld, M. and Tabershaw, I.R. (1954) *Trichloroethene toxicity. Report of five fatal cases.* AMA: Arch. Ind. Hyg. Occup. Med. 10:134-141.

National Toxicology Program (NTP) (1986a) Toxicology and carcinogenesis studies of trichloroethylene in F344/N rats and B6C3F1 mice. NTP TR 243.

National Toxicology Program (NTP). (1986b) Trichloroethylene: Reproduction and fertility assessment in F344 rats when administered in the feed. Final Report. NTP-86-085.

Perocco, P. and Prodi, G. (1981) *DNA damage by haloalkanes in human lymphocytes cultured in vitro.* Cancer Lett. 13:213-218.

Silverman, A.P. and Williams, H. (1975) *Behaviour of rats exposed to trichloroethylene vapors.* Br. J. Ind. Med. 32:308-315.

U.S. Environmental Protection Agency (U.S. EPA) (1985) Health assessment document for trichloroethylene, Final Report. Washington, DC. NTIS PB85-249696.

U.S. Environmental Protection Agency (U.S. EPA) (1987) Addendum to the health assessment document for trichloroethylene: Update carcinogenicity assessment for trichloroethylene. EPA/600/8-82/006FA.

1,2,4-TRIMETHYLBENZENE
CAS No. 95-63-6

A. Potential Sources and Exposure

This compound, also called "pseudocumene", occurs in coal tars and petroleum. It is used in the manufacture of dyes, perfumes, resins, and pharmaceuticals. It is also used as a solvent, paint thinner, and as a vermifuge. It is one of the three isomers of trimethylbenzene. Although exposure to one isomer alone is possible, it is more likely that exposure would be to an isometric mixture of trimethylbenzene in coal tar or petroleum distillates.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	120.21 g/mol
Water Solubility	57 mg/L at 25°C.
Vapor Pressure	2.7×10^{-3} atm at 25°C.
K_{oc}	not available
log K_{ow}	3.65
Henry's Law Constant	5.8×10^{-3} atm-m^3/mole

C. Toxicity

This compound is toxic by inhalation and dermal contact. Effects from exposure may include headache, fatigue, nausea, irritation of the skin, eyes, and mucous membranes, central nervous system depression, asthmatic bronchitis, chemical pneumonitis, or pulmonary edema.

An investigation of 27 persons who worked for years with a solvent containing 50% 1,2,4-trimethylbenzene and 30% 1,3,5-trimethylbenzene had nervousness, tension, anxiety, asthmatic bronchitis, a tendency to hypochromic anemia and deviation in coagulability of the blood. Workers exposed to paint thinner containing 80% 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene experienced disturbance of blood coagulation and a tendency to hematoma formation with low level of thrombocytes and erythrocytes. Seventy percent of the workers exposed to a high concentration of this mixture experienced headache, fatigue, and drowsiness.

Rats exposed to 1700 ppm of an isomeric mixture (1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene) in air for 10 to 21 days exhibited no fatalities or other adverse effects.

Exposure for 4 months to the same concentrations caused diminished weight gain and progressively increasing lymphopenia and neutrophilia and marked central nervous system depression.

Systemic intoxication due to absorption through the skin is not believed to be probable.

D. Toxicokinetics

In rats, two major metabolites are found in the urine after oral administration, 2,4-dimethylbenzoic acid and 3,4 dimethylhippuric acid.

E. Ecological Effects

Toxicity testing resulted in a 96-hour LC_{50} for the fathead minnow of 7.72 mg/L.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air: None available

Drinking Water: None available

Water:

Ambient Water Quality Criteria, Freshwater: None

Ambient Water Quality Criteria, Marine: None

G. Toxicity Factors

Reference Doses (mg/kg-day): None available
Cancer Slope Factors $(mg/kg\text{-}day)^{-1}$: None available

H. References

Hazardous Substance Database, 1995. On-line database.

Lewis, R.J., Sr., *Sax's Dangerous Properties of Industrial Materials*, Volume III, Eighth edition, Van Nostrand Reinhold, New York.

Mackay, D., W.Y. Shiu, and K.C. Ma, 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals,* Volume I, Lewis Publishers, Ann Arbor.

XYLENE
(Dimethylbenzene, Xylol)
CAS No. 1330-20-7

A. Potential Sources and Exposure

Xylene is a volatile constituent in gasoline and used as a solvent in industry. Consumer products which contain xylene include gasoline, glues, adhesives, household cleaning products, paint strippers, and cigarette smoke. Given xylene's high volatility, the most common route of exposure is inhalation of vapors.

B. Physical and Chemical Properties

Property	Value	Reference
Molecular Weight	106.16 g/mol	Verschueren, 1983
Water Solubility	175 mg/L at 20°C,insoluble, and 198mg/L at 25°C for 1,2-,1,3- and 1,4-xylene	Verschueren, 1983
Vapor Pressure	5,6, and 6.5 mm Hg for 1,2-,1,3 and 1,4-xylene at 20°C	Verschueren, 1983
K_{oc}	not available	----------
K_{ow}	589, 1585, and 1412 for 1,2-, 1,3-, and 1,4-xylene	Lyman et. al., 1982
Henry's Law Constant	5.1×10^{3} $atm\text{-}m^3/mol$	Lyman et. al., 1982

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the USEPA.

C. Toxicity

When inhaled at high concentrations, xylene causes CNS depression; it can also cause reddening of the face, disturbed vision, and salivation. There is some evidence suggesting that xylene sensitizes the myocardium to the endogenous neurohormone, epinephrine, and can precipitate heart failure and death.

Marks et. al. (1982) reported that xylene caused adverse reproductive effects in mice. In this study, mice were administered xylene three times per day on days 6-15 of gestation by oral gavage at doses from 0.52 to 4.13 g/kg/day. An increase in cleft palate formation in the offspring was noted at doses of 2.06 g/kg/day and higher. At these doses, an increase in liver weight and a decrease in fetal weight were also observed. No adverse effects were seen at a dose of 1.03 g/kg/day. Mirkova et. al. (1983) reported that xylene caused embryotoxic effects in rates after inhalation exposure. In this study, rats were exposed to xylene at a concentration, of 10, 50, or 500 mg/m^3, 6 hr/day, 5 days/wk from days 1 through 21 of gestation. Significant embryotoxic effects were produced after exposure to the two higher concentrations but not to the lowest concentration.

Workers chronically exposed to xylene display symptoms similar to those seen in acutely exposed individuals (Sandmeyer, 1981). In addition, there have been reports that disturbances in the blood can occur from xylene exposure. These effects, however, may be due to benzene contamination. The ACGIH has recommended a TLV for xylene of 100 ppm (ACGIH, 1980).

There are no studies to indicate that xylene is carcinogenic or mutagenic.

D. Toxicokinetics

Xylene is presumed to be absorbed after inhalation or oral exposure since toxicities have resulted after exposure by these routes. The rates at which xylene is absorbed have not been well characterized. The available data suggest that xylene toxicity is more severe after inhalation exposure than after oral exposure.

The major metabolite of xylene is methyl hippuric acid. This metabolite also represents the major urinary excretory product of xylene. Xylene can also be eliminated unchanged in the exhaled air.

E. Ecological Effects

Xylene is a volatile aromatic hydrocarbon. Xylene tends to not be persistent because it readily volatizes from surface water and surface soil environments. In aquatic environments some fraction of the chemical may become adsorbed onto organic particulate matter and settle to the bottom or remain in suspension. The chemical may be degraded by photodegradation processes in the atmosphere and shallow water or surface soil environments and by

microorganisms in soil and water.

Because xylenes are volatile chemicals, static acute bioassays tend to underestimate their toxicity. Flow-through bioassays have been used to determine the acute toxicity of xylenes to trout and yielded an LC50 value of 13,500 ug/L. This is lower than but similar to other acute toxicity values for which; these range up to about 30,000 ug/L. Acute toxicity of xylenes to marine organisms are similar though somewhat lower with LC50 values in the range of 2,000 to 10,000 ug/L. No data were found on the chronic toxicity of xylene. Sublehal effects (avoidance) has been observed with fish larvae which responded to concentrations of xylene on the order of 100 ug/l.

Because of the volatility and low toxicity of xylenes, effects upon wild birds and mammals are expected to be minimal. No specific studies have been conducted to formally evaluate this.

Reported Levels In Ambient Air (ng/m^3) (Singh et. al., 1983)

St. Louis	5,451
Oakland	9,864
Pittsburgh	10,120
Chicago	10,406
Riverside	14,411
Denver	17,912
Houston	22,269
Staten Island	25,224
Phoenix	25,872
Los Angeles	28,295

Reported Levels In Indoor Air (ng/m^3) (USEPA, 1987)

Elizabeth-Bayonne	
Winter	49,000
Summer	49,000
Fall	68,000
New Jersey	
Winter	38,800
Summer	27,000
Fall	71,000
Los Angeles	
February	41,000
May	31,400

Contra Costa
June 14,700

F. Federal Regulations, Standards, Guidelines and Criteria

Air: the Federal Standard and the ACGIH, 1983/1984 TWA value is 100 ppm (435 mg/m3) for all isomers. NIOSH recommends adherence to the present federal standard of 100 ppm as a time weighted average.

Ambient Water Quality: No criteria set by USEPA.

Drinking Water:Maximum Contamiment Leve l(MCL) 440 ug/L

G. Toxicity Factors

Reference Doses (mg/kg-day):

Oral 2 (chronic)

H. References

American Conference of Governmental Industrial Hygienists (ACGIH). 1980. Documentation of threshold limit values. Fourth Edition.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of chemical property estimation methods. McGraw-Hill Book Co., NY.

Marks, T.A., T.A. LeDoux and J.A. Moore. 1982. Teratogenicity of a commercial xylene mixture in the mouse. *J. Toxicol. Environ. Health* 9:97-105.

Mirkova, E., C. Zaikov, G. Antov., A. Mikhailova, L. Khinkova and I. Benchev. 1983. Prenatal toxicity of xylene. *J. Hyg. Epidemiol.*, Microbiol. Immunol. 27:337-343.

Sandmeyer, E.E. 1981. Aromatic hydrocarbons. In: Patty's Industrial Hygiene and Toxicology. Editors, G.D. Clayton and F.E. Clayton. Volume 2B. Third Edition. John Wiley and Sons, NY.

U.S. Environmental Protection Agency (U.S. EPA). 1984. Summary of current acceptable daily intakes (ADIs) for oral exposure. Cincinnati, OH.

Verschueren, K. 1983. Handbook of environmental data on organic chemicals. Second Edition. Van Nostrand Reingold Co., NY.

SEMI-VOLATILE ORGANIC COMPOUNDS

POLYCYCLIC AROMATIC HYDROCARBONS
(PAHs, polynuclear hydrocarbons)

Since the polycyclic aromatic hydrocarbons (PAHs) are rarely found individually in the environment, and the effects on the environment and human health are not well defined for discrete PAHs, the reader is asked to refer to this toxicity profile for general information on the PAHs and to use the individual toxicity profiles for specific compounds.

A. Potential Sources and Exposure

The polycyclic aromatic hydrocarbons (PAHs) are a group of compounds that are formed during the incomplete combustion of coal, oil, gas, wood and other organic compounds. Natural sources of PAHs include forest fires and volcanic eruptions. PAHs are ubiquitous in soil and are rarely found as individual compounds. The greatest exposure sources of PAHs to humans are active or passive inhalation of the compounds in tobacco smoke, wood smoke and contaminated air. Exposure may also occur through ingestion of grilled or smoked foods, contaminated water or foods and through skin contact with soot, tars or contaminated sediments.

B. Physical and Chemical Properties

The PAHs have been categorized by the number of aromatic rings in their chemical structure as well as by their carcinogenicity in laboratory animals. Although naphthalene is a two-ringed structure, it is frequently categorized as a PAH. The other compounds are listed below and are three, four or five-ringed structures. PAHs are commonly found in the environment are solids at room temperature and are virtually insoluble in water.

2-Ringed PAH	3-Ringed PAHs	4-Ringed PAHs	> 4-Ringed PAHs
naphthalene	Acenaphthene	Benzo(a)anthracene	Benzo(b)fluoranthene
	Acenaphthylene	Benzo(a)pyrene	Benzo(k)fluoranthene
	Anthracene	Chrysene	Benzo(ghi)perylene
	Fluorene	Fluoranthene	Dibenz(a,h)anthracene
	Phenanthrene	Pyrene	Indeno(1,2,3-cd)pyrene

C. Toxicity

Within the large class of PAHs, many structure-activity relationship studies have been done to relate chemical structure to carcinogenic activity. Each of the environmentally relevant PAHs

No standard fresh water toxicity tests have been reported for polycyclic aromatic hydrocarbons (except naphthalene) as a class or specific compounds. There are some data for bioconcentration during tests with model ecosystems, or for short exposure periods.

Lu et. al. (1977) conducted studies with benzo(a)pyrene in a terrestrial-aquatic model ecosystem and observed bioconcentration factors after 3 days ranging from 930 for the mosquitofish to 134,248 for *Daphnia pulex*. Bioconcentration factors for *Daphnia magna* and *Hexagenia* sp. for a shorter time were 200 to 3,500. English sole and white suckers from populations with high frequencies of neoplasia had elevated levels of PAHs in their stomach contents.

Some PAH metabolites are carcinogenic, mutagenic, or teratogenic to organisms. Rather than enhancing detoxification, metabolism of some carcinogenic PAHs in induced animals could result in a higher steady-state level of toxic products (Stegeman, 1981). Although studies with various carcinogens have demonstrated that chemicals can cause cancer in aquatic species, most attempts to demonstrate carcinogenesis by PAHs in aquatic species have produced equivocal results (Pliss and Khudoley, 1975). Although recently there has been some evidence that PAH can cause cancer in aquatic animals, there is to date no direct evidence of a single specific PAH induction of carcinogenesis in aquatic species (Neff, 1979; Stegeman, 1981).

Studies in the Duwamish River, Boston Harbor, and Hudson River have identified populations of Dover sole and Atlantic tomcod with very high incidences of hepatocellular carcinoma (Varanasi, 1989), and higher incidences of similar diseases have been reported for other environments. Although the etiology of such diseases in fish is uncertain, there is reason to suspect that the chemical environment is responsible, and PAHs have not been exonerated (Stegeman, 1981). Bottom sediments in the areas that these fish populations inhabited contained elevated levels of PAHs.

The impacts of concern in the terrestrial environment include both direct toxicity and food-chain impacts. The toxic effects of PAHs in mammals can be inferred from the extensive toxicity testing work performed on laboratory animals. As with humans, the basic conclusion is that exposure to PAHs are only slightly to moderately toxic by acute exposure, but longer exposures to certain PAHs can result in cancer. Biomagnification in animal food chains is unlikely, however, since PAHs are readily metabolized.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL) for the benzene soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene, and pyrene). 0.2 mg/m^3

H. References

Integrated Risk Information System (IRIS) on-line database, accessed 1/13/92.

International Agency for Research on Cancer (IARC). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Volume 32: Polynuclear aromatic compounds, Part 1, Chemical environmental and experimental data, Lyon, France: World Health Organization.

Lu, P.Y., R.L. Metcals, N. Plummer, and D. Mandel. 1987. *Arch. Env. Contam. Toxicol.* 6:129-142.

Neff, J.M., 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. Applied Science Publishers, London.

Pliss, G.B., and V.V.Khudoley. 1975. *J. Natl. Cancer Inst.* 55:129.

Steggeman, J.J., 1981. PAHs and their Metabolism in the Marine Environment. In: PAHs and Cancer, Vol. 3, edited by H.V. Gelboin and P. Tso. Academic Press, NY. (pp1-60).

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for Polycyclic Aromatic Hydrocarbons. EPA 440/5-80-069. Washington, DC.

_____. 1982. An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons. Volume IV. Final Draft Report. Washington, DC.

_____. 1984. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons. EPA-540/1-86/013. Cincinnati, OH.

Varanasi, U. 1989. Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. CRC Press, Cleveland, OH.

ACENAPHTHENE
CAS No. 83-32-9

A. Potential Sources and Exposure

Acenaphthene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	154.2 g/mol
Water Solubility	3.42 mg/L at 25°C
Vapor Pressure	1.55×10^{-3} mm Hg at 20°C
K_{oc}	4600 mL/g
log K_{ow}	4
Henry's Law Constant	9.1×10^{-5} atm-m^3/mole

C. Toxicity

Acenaphthene has been shown to be irritating to the skin and mucous membranes and to cause vomiting following ingestion.

A review of the reported literature indicates that there are no conclusive experiments demonstrating the carcinogenic potential of acenaphthene. Studies using several different bacterial test systems provide no evidence of mutagenicity. No information concerning its teratogenicity or reproductive toxicity is available.

The oral RfD of 0.06 mg/kg-day for acenaphthene is based on subchronic study in mice. Four groups of CD-1 mice (20/sex/group) were gavaged daily with acenaphthene for 90 days. Liver weight changes accompanied by microscopic alterations (cellular hypertrophy) were noted in both mid- and high-dose animals and seemed to be dose-dependent. The LOAEL of 350 mg/kg/day is based on hepatotoxicity; the NOAEL is 175 mg/kg/day.

D. Toxicokinetics

Like other PAH compounds, acenaphthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No data were located on the

absorption of acenaphthene in laboratory animals or humans. In the absence of data it is assumed that 100 percent of acenaphthene is absorbed via the oral or inhalation exposure routes.

E. Ecological Effects

In aquatic acute toxicity tests EC50 values of 41,200 and 1,700 ug/L are reported for the cladoceran *Daphnia magna* and the bluegill, respectively. In saltwater species, the acute toxicity (96-hr LC50) values for shrimp and sheepshead minnow are 970 ug/L and 2,230 ug/L respectively. A chronic value of 710 ug/L is reported for the sheepshead minnow, yielding an acute:chronic ratio of 3:1.

A bioconcentration factor of 387 has been determined for bluegill sunfish.

A study summarizing the toxicity of a variety of compounds to wild and domestic bird species indicates that the LD50 of acenaphthene for redwinged blackbird is greater than 100 mg/kg.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for the PAHs for information regarding federal regulations, standards and guidelines.

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Subchronic	Chronic
Oral	0.6	0.06

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

ACENAPHTHYLENE
CAS No. 208-96-8

A. Potential Sources and Exposure

Acenaphthylene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	152.2 g/mol
Water Solubility	3.93 ppm at 24°C
Vapor Pressure	2.9×10^{-2} at 20°C
K_{oc}	2500 mL/g
log K_{ow}	3.7
Henry's Law Constant	1.48×10^{-3} atm-m^3/mol

C. Toxicity

Little information regarding the acute or chronic toxicity of acenaphthylene is available.

There are no long-term studies in the literature that adequately evaluate the carcinogenicity of acenaphthylene, nor are there any data from epidemiologic studies which correlate acenaphthylene exposure with an increased risk of cancer. A skin-painting study in mice produced negative results (IRIS, 1992). Structurally, acenaphthylene is similar to other low molecular weight polycyclic aromatic hydrocarbons that are considered to be noncarcinogenic. Acenaphthylene is classified as a Group D carcinogen by the USEPA based on the lack of human carcinogenicity data and inadequate data from animal bioassays.

Positive results have been reported from a single mutagenicity test in which acenaphthylene was tested in a strain of Salmonella typhimurium in the presence of liver microsomal activation (USEPA, 1982). Other tests in Salmonella have been negative (IRIS, 1992). There is currently no RfD for acenaphthylene, although based on structure-activity relationships with anthracene, an oral RfD of 0.3 mg/kg-day is recommended (USEPA, ORD Memo, 1992).

D. Toxicokinetics

Like other PAH compounds, acenaphthylene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No data were located on the absorption of

acenaphthylene in laboratory animals or humans. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects. A no effect level of 5 mg/L was observed for trout in an acute (24 hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for the PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses (mg/kg-day):

	Chronic
Oral	0.3

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

ANTHRACENE
(Paranaphthalene)
CAS. No 120-12-7

A. Potential Sources and Exposure

Anthracene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property (a)	Value	Reference
Molecular Weight	178.2	Mabey et. al., 1982
Water Solubility	1.29 ppm at 25°C $4.5x10^{-2}$ ppm at 25°C	Verschueren 1983 Mabey et. al., 1982
Vapor Pressure	$1.7x10^{-5}$ mmHg at 20°C	Mabey et. al., 1982
K_{oc}	1.4×10^{4}	Mabey et. al., 1982
K_{ow}	2.8×10^{4}	Mabey et. al., 1982
Henry's Law Constant	8.6×10^{-5} atm^{-3}/mol at 25°C	Mabey et. al., 1982

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the USEPA.

C. Toxicity

No epidemiological studies were identified which examined possible human health effects resulting from exposure to anthracene. Few reports of health effects in humans resulting from anthracene exposure exist. It is reported that three cases of epithelioma (any tumor derived from epithelium) of the hand, cheek and wrist occurred in men handling crude anthracene in an alizarin factory (Kennaway, 1924 as cited in IARC 1983). In another instance it was reported that in studies on the treatment of psoriasis, anthracene solubilized in an alcohol

N-methyl-2-pyrrolidone vehicle, induced photosensitive reactions when administered topically in low concentrations (~0.25%) to humans in combination with U.V. radiation (Urbanek, 1980; Walter, 1980 as cited in IARC, 1983).

Anthracene has been tested for carcinogenicity in a number of different species, using a variety of routes of administration, with primarily negative results. There is no evidence that anthracene is active in short-term tests. IARC,(1983) concludes that the available data provide no evidence that anthracene is carcinogenic to experimental animals.

D. Toxicokinetics

In their review of polycyclic aromatic hydrocarbons, the USEPA (1982) note that anthracene appears to be converted to 1,2-dihydroanthracene-1,2-diols and their glucuronides. In an investigation in which anthracene was incubated with rat liver preparations (Akhtar et. al., 1979 as cited in IARC, 1983), the major metabolite was identified as the 1,2-dihydrodiol. It has also been reported that the 1,2-dihydrodiol, 9,10-anthraquinone, 9,10-dihydrodiol, and 2,9,10-trihydroxyanthracene have been identified as metabolites in rat urine, together with conjugates consistent with the formation of the 1,2-oxide (Sims, 1964 as cited in IARC, 1983).

E. Ecological Effects

The profile for benzo(a)pyrene provides a generic description of the potential environmental effects of PAHs as a class of compounds. A no effect level of 5 mg/L was observed for trout in an acute (24 hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

Reported Levels In Sediments:

	mg/kg	Reference
Penobscot Bay, ME, outer region	.0069	Johnson et.al., 1985
Buzzards Bay, New Bedford, MA	.0070 - .0080	Giger and Blumer, 1974
Penobscot Bay, ME, inner region	.0234	Johnson et. al., 1985
New York Bight	.0391	Reid et. al., 1982
The Graves, Boston MA	.0420	Shiaris and Jambard-Sweet, 1986
Boston Harbor	.0725	
Buzzards Bay New Bedford, MA	.1700	Giger and Blumer, 1974
Boston Harbor Aquarium/Fort Point	.2450	Shiaris and Jambard-Sweet, 1986
Boston Harbor	.2833	Mass DEQE, (1985)
Buzzards Bay,		

New Bedford, MA	.3400	Giger and Blumer, 1974
Chelsea River, MA	.4110	Shiaris and Jambard-Sweet, 1986
Long Island Sound	.4550	Reid et. al., 1982
Savern Estuary, U.K.	2.4	John et. al., 1979

Reported Levels In Soils:

	Concentration (mg/kg)	Reference
Anthracene	0.008-0.017	USEPA (1976)

Reported Levels In Air:

Averages for		
Residential	0.03-0.83	Radian Corp., 1983
Rural	0.4	Radian Corp., 1983
Urban	0.068-0.278	White and Vanderslice, 1980
Urban	0.1-1.3	USEPA,1980
Detroit	1.2	White and Vanderslice, 1980

F. Federal Regulations, Standards, Guidelines and Criteria

Water Quality: USEPA (1980) has recommended criteria related to specific incremental lifetime risk levels of 10E-5, 10E-6, and 10E-7. The corresponding water concentrations assuming ingestion of water and aquatic organisms are 28ng/L, 2.8ng/L, and 0.28ng/L respectively. If the above estimates are made for consumption of aquatic organisms only, the levels are 311 ng/L, 31.1 ng/L, and 3.1 ng/L respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to benzo(a)pyrene. The carcinogenic potency of B(a)P and other PAHs is currently being reevaluated by a number of groups. With regard to protection of aquatic life, no numerical criteria have been developed.

OSHA Limit: An 8-hour time weighted average (TWA) concentration limit of 0.2 mg/m^3 has been set for the benzene soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene, and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample determined as a 10-hour TWA.

G. Toxicity Factors

Reference Doses (mg/kg-day):

	Subchronic	Chronic
Oral	0.3	3

State guidance may differ from federal guidance and should be consulted.

H. References

International Agency for Research on Cancer (IARC). 1983. Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data, IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans, Volume 32.

Mabey, W.R., J.H. Smith, R.T. Podell, H.L. Johnson, T. Mill, T.W. Chou, J. Gates, I.W. Partridge, H. Jaber, and D. Vandenberg. 1982. Aquatic Fate Process Data for Organic Priority Pollutants, Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, D.C., USEPA Contracts 68-01-3867 and 68-03-2981.

Sedman, R. 1986. Derivation of Applied Action Levels (AALS) for acenaphthene, State of California. Department of Health Services.

U.S. Environmental Protection Agency (USEPA). 1982. An Exposure and Risk Assessment for Benzo(a)pyrene and Other Polycyclic Aromatic Hydrocarbons: Volume III, Anthracene, Acenaphthene, Fluoranthene, Fluorene, Phenantherene, and Pyrene, Final Draft Report, Office of Water Regulations and Standards, Washington, D.C.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Second Edition. Van Nostrand Reinhold Co., New York.

BENZO(a)ANTHRACENE

(Benz(a)anthracene; 1,2-Benz(a)anthracene; Benzo(a)phenanthrene)
CAS No. 56-55-3

A. Potential Sources and Exposure

Benzo(a)anthracene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	228.3 g/mol
Water Solubility	5.7×10^{-3} mg/L at 20°C
Vapor Pressure	2.20×10^{-8} mm Hg at 20°C
K_{oc}	1,380,000 mg/L
log K_{ow}	5.6
Henry's Law Constant	1.16×10^{-6} atm-m^3/mol

C. Toxicity

Although there are no human data that specifically link exposure to benzo(a)anthracene to human cancers, benzo(a)anthracene is a component of mixtures that have been associated with human cancer. These include coal tar residues, coke oven emissions and cigarette smoke (IRIS, 1992).

Several studies indicate that benzo(a)anthracene is carcinogenic in animals, and IARC has evaluated that evidence as sufficient to establish the carcinogenicity of benzo(a)anthracene in animals (IARC, 1983). Benzo(a)anthracene administration caused an increase in the incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973); and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and Edgecomb, 1952) and intraperitoneal injection assays (Wislocki et al, 1986). A CPF has not been developed by the USEPA. Based on the work of Bingham and Falk (1969), ICF Clement (1987) estimated that benzo(a)anthracene has a relative potency - to benzo(a)pyrene - of 0.145. (Potency is approximately 14.5% of that of benzo(a)pyrene). This value can be used in the relative potency approach for estimating carcinogenic risk.

Extensive testing for mutagenicity has been documented (IARC, 1983) with mostly positive results (IRIS, 1992).

D. Toxicokinetics

Some benzo(a)anthracene metabolites have been shown to induce mutations, cell transformation, and to bind to nucleic acids. The metabolites of benzo(a)anthracene are mutagenic and tumorigenic (Sims and Grover, 1974, 1981; Conney, 1982 as cited in IARC, 1983).

Nucleic acid (DNA) adducts are formed in the skin from the metabolites 3,4-diol-1,2-epoxide and 8,9-diol-10,11-epoxide (Sims and Grover 1974, 1981; Conney 1982 as cited in IARC 1983). No information is available regarding dermal or oral absorption coefficient, although benzo(a)anthracene was reported to be readily transported across the gastrointestinal mucosa (Rees et. al., 1971 as cited in USEPA, 1984).

Benzo(a)anthracene induced benzo(a)pyrene hydroxylase in rat placenta (Welch et. al., 1969 as cited in IARC, 1983).

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

Hinga et. al. (1980) examined the biogeochemistry of C-14 labelled benzo(a)anthracene in an enclosed marine ecosystem. The experiment was conducted for 230 days. At the end of the experiment, 29% of the chemical had been respired to CO_2, while the remaining extractable activity (43%) was evenly divided between parent compound and intermediate metabolic products. Total C-14 activity was removed from the water with a half-life of about 52 hrs, while the C-14 parent compound had a half-life of 24 hrs. The chemical became associated with the sediments and was mixed deeper into the sediments by benthic animal activity. The authors made a rough calculation of the half-life in sediments and noted stated that half-lives on the order of 1.2 to 3 years may be calculated. They further point out, however, that the occurrence of benzo(a)anthracene at some depth in natural sediments suggests that a fraction of the compound and perhaps some of its metabolites may persist indefinitely.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

Air:

OSHA Permissable Exposure Limit (PEL) 0.2 mg/m^3 for the benzene soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene, and pyrene).

NIOSH Recommended Exposure Limit (REL) 0.1 mg/m^3 10-hour TWA for coal tar, coal tar pitch, creosote and mixtures of these substances of the cyclohexane-extractable fraction.

Drinking Water:

USEPA MCL 0.0002 mg/L (Proposed, 1990)
with a maximum lifetime individual risk of 1 x 10^{-4}

Water and Fish Consumption: 2.8 x 10^{-3} ug/L

USEPA MCLG (proposed, 1990) 0 mg/L

This value is based on carcinogenic PAHs as a class of compounds.

Water:

USEPA Marine Acute LEC 300 ug/L

With regard to protection of aquatic life, no numerical criteria have been developed.

Food:

USEPA Fish Consumption Only: 3.11 x 10^{-2} ug/L

G. Toxicity Factors

Reference Doses (mg/kg/day) are currently not available.

Cancer Potency Factors (IRIS, 1992):
Oral exposure: 5.8 (mg/kg-dy)$^{-1}$

H. References

Bingham, E., H.L. Falk. 1969. Environment carcinogens, The modifying effect of carcinogens on the threshold response. *Arch. Environ. Health.* 19:779-783.

Hinga, K.R., M. Pilson, R.F. Lee, J.W. Farrington, K. Tjessem, and A.C. Davis, Biogeochemistry of benzoanthracene in an enclosed marine ecosystem. *Envir. Sci. & Tech.* 14:1136-1143.

International Agency for Research on Cancer (IARC). 1983. Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Volume 32. Polynuclear Aromatic

Compounds. Part I. Chemical, environmental and experimental data. World Health Organization, Lyon, France.

International Agency for Research on Cancer (IARC). 1973. Certain Monographs on the Evaluation of Carcinogenic Risk to Humans. Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. Volume 3. World Health Organization, Lyon, France.

U.S. Environmental Protection Agency (U.S. EPA). 1984. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons. EPA-540/1-86/013. Washington, D.C.

BENZO[b]FLUORANTHENE

GENERAL BACKGROUND INFORMATION

Benzo[b]fluoranthene (BbF) is a member of the class of compounds referred to as polycyclic aromatic hydrocarbons (PAHs). PAHs contain two or more aromatic rings. PAHs are ubiquitous in nature and are both naturally occurring and man-made. Exposure to BbF can come from air, water, or soil. As a PAH, BbF is present in the emissions from industrial plants that produce coal tar, cooking plants, asphalt production plants, and home heating with wood and coal. BbF is also present in charcoal-broiled foods and cigarette smoke (ATSDR, 1990).

PHARMACOKINETICS

No data on the absorption, distribution or excretion of BbF were identified. BbF is metabolized under *in vitro* incubation conditions to phenol and dihydrodiol metabolites (Amin et al., 1982). The general metabolic pathways elucidated for benzo(a)pyrene are also active on BbF (Cooper et al., 1983; Levin et al., 1982; Grover et al., 1986). The reactive metabolites associated with the tumorigenic effects of BbF may not be the diol epoxides (Amin et al., 1982; Amin et al., 1985). As for the other PAHs, the material excreted is expected to consist primarily of dihydrodiol and phenol conjugates (Grover et al., 1986).

HUMAN TOXICOLOGICAL PROFILE

The database for human toxicity is very limited. There are no studies correlating exposure to BbF and cancer or systemic toxicity. The only data implicating BbF as a carcinogen come from carcinogenicity studies using a mixture of PAHs.

MAMMALIAN TOXICOLOGICAL PROFILE

The database on the toxicity of BbF is limited. Intratracheal administration of BbF to rats resulted in an increase in respiratory tract tumors (Deutsch-Wenzel et al., 1983). BbF has caused skin tumors in mice following dermal application (Wynder and Hoffman, 1959). The skin tumor initiating ability of BbF has been demonstrated in mice using a standard initiation/promotion protocol with either croton oil or phorbol myristate acetate as a tumor promotor (Amin et al., 1985; LaVoie et al., 1979, 1982).

GENOTOXICITY

The genotoxicity of BbF has been shown equivocally in three *in vitro* studies. BbF has been shown to be mutagenic in *Salmonella typhimurium* in the presence of an exogenous rat-liver preparation (LaVoie et al., 1979). Mutagenic activity has been reported in another similar study (Hermann, 1981). Negative results were reported by Mossanda (1979). The results cannot support an unequivocal determination regarding the genotoxicity of BbF at this time.

REFERENCES

Agency for Toxic Substance and Disease Registry (ATSDR) (1990) Toxicological profile for benzo(b)fluoranthene. U.S. Public Health Service.

Amin, S., LaVoie, E.J. and Hecht, S.S. (1982) *Identification of metabolites of benzo(a)fluoranthene*. Carcinogenesis 3:171-174.

Amin, S., Huie, K., Hussaur, N., Geddie, J.E. and Hecht, S.S. (1985) *Mutagenicity and tumor initiating activity of methylated benzo(b)fluoranthenes*. Carcinogenesis 6:1023-1025.

Cooper, C.S., Grover, P.L. and Sims, P. (1983) *The metabolism and activation of benzo(a)pyrene*. In: Bridges, J.W., Chase, L.F. eds. Progress in drug metabolism, Vol.7. John Wiley and Sons, New York, pps. 295-396.

Deutsch-Wenzel, R.P., Brune, H., Grimmer, O., Dettbarn, G. and Misfield, J. (1983) *Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons*. J. Natl.Cancer Inst. 71:539-544.

Grover, P.L. (1986) *Pathways involved in the metabolism and activation of polycyclic aromatic hydrocarbons*. Xenobiotica. 16:915-931.

Hermann, M. (1981) *Synergistic effects of individual polycyclic aromatic hydrocarbons and the mutagenicity of their mixtures*. Mutat. Res. 90:399-409.

LaVoie, E.J., Amin, S., Hecht, S.S., Furuya, K. and Hoffmann, D. (1979) *On the metabolic activation of the environmental carcinogens benzo(j)fluoranthene and benzo(b)fluoranthene*. Proc. Am. Assoc. Ca. 20:81.

LaVoie, E.J., Amin, S., Hecht, S.S., Furuya, K. and Hoffmann, D. (1982) *Tumour initiating activity of dihydrodiols of benzo(b)fluoranthene, benzo(j)fluoranthene, and benzo(k)fluoranthene*. Carcinogenesis 3:49-52.

Levin, W., Wood, A., Chang, R.L. et al. (1982) *Oxidative metabolism of polycyclic aromatic hydrocarbons to ultimate carcinogens*. Drug Metab. Rev. 13:555-580.

Mossanda, K., Poncelet, F., Fousasin, A. and Mercier, M. (1979) *Detection of mutagenic polycyclic aromatic hydrocarbons in African smoked fish*. Food Cosmet. Toxicol. 17:141-143.

Wynder, E.L. and Hoffmann, D. (1959) *The carcinogenicity of benzofluoranthenes*. Cancer 12:1194-1199.

BENZO(k)FLUORANTHENE
(11,12-Benzo(k)fluoranthene)
CAS No. 207-08-9

A. Potential Sources and Exposure

Benzo(k)fluoranthene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	252.3 g/mol
Water Solubility	4.3×10^{-3} mg/L at 25°C
Vapor Pressure	5.0×10^{-7} mm Hg at 20°C
K_{oc}	5.5×10^{5} mL/g
log K_{ow}	6.06
Henry's Law Constant	3.94×10^{-5} atm-m^3/mol

C. Toxicity

Although there are no human data that specifically link exposure to benzo(k)fluoranthene to human cancers, benzo(k)fluoranthene is a component of complex mixtures that have been associated with human cancer. These include soot, coke oven omissions, and cigarette smoke (USEPA as cited in IRIS, 1992). IARC concluded that there is sufficient evidence for the carcinogenicity of benzo(k)fluoranthene in experimental animals. Benzo(k)fluoranthene has been administered by skin painting, subcutaneous injection, and intrapulmonary injection. USEPA has classified benzo(k)fluoranthene as a probable human carcinogen (B2).

D. Toxicokinetics

Like other PAH compounds, benzo(k)fluoranthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information was available regarding DNA adduct formation or absorption factors. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for the PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses and Cancer Slope Factors for this compound are currently not available. The toxicity of benzo(k)fluoranthene is evaluated relative to the toxicity of benzo(a)pyrene. Based on the work of Deutsch-Wenzel et. al. (1983), ICF Clement (1986) estimated that the potency of benzo(k)fluoranthene relative to benzo(a)pyrene is approximately 0.066. This number can be used in the relative potency approach to estimate a CSF.

H. References

For references, see the Polycyclic Aromatic Hydrocarbon toxicity profile.

BENZO(ghi)PERYLENE
(1,12-Benzoperylene)
CAS No. 191-24-2

A. Potential Sources and Exposure

Benzo(ghi)perylene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	276.3 g/mol
Water Solubility	7×10^{-4} mg/L at 25°C
Vapor Pressure	1.03×10^{-10} mm Hg at 25°C
K_{oc}	1.6×10^{6} mg/L
log K_{ow}	6.51
Henry's Law Constant	5.34×10^{-8} atm-m^3/mol

C. Toxicity

Although there are no human data that specifically link exposure to benzo(ghi)perylene to human cancers, it is found in complex mixtures that have been associated with human cancer. These include soot, coke oven emissions, and cigarette smoke (USEPA as cited in IRIS, 1992).

IARC (1983) and USEPA (IRIS, 1992) concluded that the available data are inadequate to evaluate the carcinogenic potential of benzo(ghi)perylene and is classified as a Group D carcinogen by the USEPA based on the lack of human carcinogenicity data and inadequate data from animal bioassays. Based on the study by Deutsch-Wenzel et. al. (1983), in which benzo(g,h,i) perylene increased lung tumor incidence when implanted into rat lungs, ICF Clement (1986) reported that the potency of this compound relative to benzo(a)pyrene was 0.022.

Negative tumorgenicity results were obtained for benzo(ghi)perylene in skin painting studies using mice (Wynder and Hoffman, 1959; Hoffman and Wynder, 1966; Muller, 1968; Van Duuren et al., 1973; as cited in IRIS, 1992). Mutations due to benzo(ghi)perylene were evident in in vitro bacterial mutagenicity tests (IARC, 1983).

D. Toxicokinetics

No data are available regarding the formation of carcinogenic metabolites, DNA adduct formation, enzyme induction, or absorption.

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses are currently not available.

H. References

For references, see the PAH toxicity profile.

BENZO(a)PYRENE

(Benzo(d,e,f) chrysene, 3,4-Benzopyrene, 6,7-Benzopyrene)
CAS No. 50-32-8

A. Potential Sources and Exposure

Benzo(a)pyrene (B(a)P) is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	252.3 g/mol
Water Solubility	1.2×10^{-3} mg/L at 20°C
Vapor Pressure	5.60×10^{-9} mm Hg at 25°C
K_{oc}	5,500,000 mL/g
log K_{ow}	6.06
Henry's Law Constant	1.55×10^{-6} atm-m^3/mol

C. Toxicity

Lung and skin tumors have been induced in humans by mixtures of PAHs known to contain benzo(a)pyrene (cigarette smoke, roofing tar and coke oven emissions). It is not possible, however, to conclude from this information that benzo(a)pyrene is the responsible agent (IRIS, 1992).

Benzo(a)pyrene is a complete carcinogen when applied to the skin of mice, rats, and rabbits (IARC, 1973). Subcutaneous or intramuscular benzo(a)pyrene injection has been shown to result in local tumors in mice, rats, guinea pigs, monkeys and hamsters (IARC, 1973). Intratracheal instillation of benzo(a)pyrene products produced increased incidences of respiratory tract neoplasms in both male and female Syrian hamsters (Feron et al., 1973; Kobayashi, 1975 as cited in IRIS, 1992).

Benzo(a)pyrene administered orally to rats and hamsters produces stomach tumors. Neal and Ridgon (1967) administered dietary benzo(a)pyrene in a subchronic study to male and female CFW-Swiss mice. Stomach tumors were observed in mice consuming 20 or more mg/kg benzo(a)pyrene. Incidence was apparently related both to the dose and the number of administered doses.

Hamsters were chronically exposed to B(a)P by inhalation (Thyssen et al., 1981 as cited in IRIS, 1992) and were shown to develop respiratory tract tumors. Those hamsters in the highest dose group developed upper digestive tract tumors.

USEPA has classified B(a)P as a Group B2, or probable human carcinogen. The oral cancer slope factor is based on a dietary study in mice published by Neal and Rigdon (1967). The data were modeled by two procedures to provide three upper bound estimates. A linearized multistage procedure was applied to data by Brune et al., (1981) to provide the fourth estimate. The range is 4.5 to 9.0 $(mg/kg\text{-}day)^{-1}$, with a median of 6.3 $(mg/kg\text{-}day)^{-1}$. The geometric mean of the four risk estimates is 7.3 $(mg/kg\text{-}day)^{-1}$.

D. Toxicokinetics

There are no toxicokinetic data for B(a)P in humans (USEPA, 1980). Animal data indicate that B(a)P is readily absorbed after exposure by inhalation or oral intake and distributes to many tissues in the body (USEPA, 1980). B(a)P in itself is not believed to be carcinogenic, but metabolized by the cytochrome P-450 dependent mixed function oxidase system, often referred to as the aryl hydrocarbon hydroxylase (AHH) system. The metabolism results in a more hydrophilic compound which is easier to excrete, although is carcinogenic. The hepatic metabolic pathway for B(a)P metabolism is readily inducible by exposure to a variety of chemicals, including B(a)P, and is found in most mammalian tissues. It catalyzes the formation of reactive epoxide intermediates as well as the ultimate carcinogenic form of B(a)P: the B(a)P-7,8-diol-9,10-epoxide (USEPA, 1982) which is capable of forming covalent bonds with cellular macromolecules such as DNA, RNA, and proteins. This covalent binding and subsequent alteration of structure and function may result in tumor formation.

Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards and guidelines, and criteria.

G. Toxicity Factors

Reference Doses are currently not available.

Cancer Potency Factors (IRIS, 1992):
Oral exposure: 7.3 $(mg/kg\text{-}dy)^{-1}$

H. References

For references, see the PAH toxicity profile.

CHRYSENE
CAS No. 218-01-9

A. Potential Sources and Exposure

Chrysene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	228.3 g/mol
Water Solubility	1.8×10^{-3} mg/l at 25°
Vapor Pressure	6.3×10^{-9} mm Hg at 25°C
K_{oc}	2.0×10^{5} ml/g
log K_{ow}	5.61
Henry's Law Constant	1.05×10^{-6} atm-m^3/mol

C. Toxicity

Although there are no human data that specifically link exposure to chrysene to human cancers, chrysene is a component of mixtures that have been associated with human cancers. These include coal tar, soots, coke oven emissions and cigarette smoke (IARC, 1983). USEPA has classified chrysene as a Group B2, or probable human carcinogen, on the basis of evidence of carcinogenicity from mouse skin painting and intraperitoneal chrysene injections in male mice which caused an increased incidence of liver tumors (Wislocki et al., 1986); Buening et al., 1979). In mouse skin painting assays, chrysene tested positive in both initiation and complete carcinogen studies (Wynder and Hoffman, 1959). The relative tumorigenic potency of chrysene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays tested using similar protocols (USEPA, 1984). The ranking was as follows: benzo(a)pyrene > dibenz(a,h)anthracene > benzo(b)fluoranthene > benz(a)anthracene > indeno(1,2,3-cd)pyrene > chrysene.

There is limited evidence that chrysene is mutagenic in short-term assays (IARC, 1983). There are no experimental data on the teratogenicity of chrysene in mammals. There is no information on the potential effects of chrysene on other endpoints of toxicity.

Based on the data of Wynder and Hoffman (1959), ICF Clement (1986) estimated that chrysene had a relative potency to B(a)P of approximately 0.0044. This number can be used in the relative potency method to estimate a cancer potency factor.

D. Toxicokinetics

Like other PAH compounds, chrysene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Several monohydroxyl and dihydrodiol derivatives of chrysene have been reported (IARC, 1983). Epoxides of the 1,2-dihydrodiol and 3,4-dihydrodiol have also been reported (IARC, 1983). The 1,2-dihydrodiol and 1,2-diol-3,4-epoxide have been shown to be mutagenic in bacterial and mammalian cells (Wood et. al., 1977), 1979 as cited in IARC, 1983) and inducers of pulmonary adenomas in newborn mice (Buening et. al., 1979; Chang et. al., 1983 as cited in IARC, 1983 as cited in IARC, 1983). In addition, the 1,2-dihydrodiol has been shown to be a tumor initiating agent on mouse skin (Levin et. al., 1978; Slaga et. al., 1980; Chang et. al., 1983 as cited in IARC, 1983). The 1,2-diol-3,4-epoxide is believed to be the metabolite of chrysene that forms adducts with DNA (Hodgson et. al., 1982; Vigny et. al., 1982 as cited in IARC, 1983).

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses and Cancer Slope Factors are currently not available.

H. References

For references, see the PAH toxicity profile.

DIBENZO(a,h)ANTHRACENE
(1,2,5,6-Dibenzanthracene, Dibenz(a,h)anthracene)
CAS No. 53-70-3

A. Potential Sources and Exposure

Dibenzo(a,h)anthracene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	278.36 g/mol
Water Solubility	5.0×10^{-4} mg/L at 25°C
Vapor Pressure	1.0×10^{-10} mm Hg at 20°C
K_{oc}	3.3×10^{6} mL/g
log K_{ow}	6.8
Henry's Law Constant	7.30×10^{-8} atm-m^3/mol

C. Toxicity

Although there are no human data that specifically link exposure to dibenzo[a,h]anthracene with human cancers, dibenzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (USEPA, 1984, 1990; IARC, 1984).

Dibenzo(a,h)anthracene [DB(a,h)A] has been tested for carcinogenicity in a variety of test species employing a number of different routes of exposure with positive results having been reported in the majority of studies. Little data were identified concerning toxic effects other than tumor induction in the various test species. USEPA has classified dibenzo(a)anthracene as group B2; probable human carcinogen, based on sufficient data from animal biossays. Dibenzo[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenzo[a,h]anthracene and some of its metabolites have induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

D. Toxicokinetics

Like other PAH compounds, DB(a,h)A is oxidized by liver enzymes to form water-soluble

derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients.

No quantitative data were located concerning the absorption of DB(a,h)A in experimental animals. The 5,6-oxide and the 1,2- 3,4- and 5,6-dihydrodiols have been detected as metabolites of DB(a.h)A after incubation in rat liver preparations (Selkirket et al., 1971; MacNicoll et. al., 1979, 1980 as cited in IARC, 1983) and mouse skin in organ culture (MacNicoll et al., 1980 as cited in IARC, 1983). The 5,6-oxide was found to bind to cellular macromolecules in mammalian cells (Kuroki, et. at., 1972 as cited in IARC, 1983). Nucleoside adducts have been detected in mouse skin following topical application of DB(a,h)A but were not characterized (Phillips et. al., 1979 as cited in IARC, 1983).

No information on the tissue distribution or excretion of DB(a,H)A could be located. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for the PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses and Cancer Slope Factors are currently not available for this compound. Based on the work of Wynder and Hoffman (1959), ICF Clement (1986) estimated that DB(a,h)A had a potency relative to benzo(a)pyrene of 1.11. This number can be used in the relative slope method to estimate a cancer slope factor.

H. References

For references, see the PAH toxicity profile.

FLUORANTHENE

(Idryl; 1,2-(1,8-Naphthylene)benzene; Benzo(jk)fluorene)
CAS No. 206-44-0

A. Potential Sources and Exposure

Fluoranthene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	202.30 g/mol
Water Solubility	0.206 mg/L at 25°C
Vapor Pressure	5.0×10^{-6} mm Hg
K_{oc}	3.8×10^{4} mL/g
log K_{ow}	4.9
Henry's Law Constant	6.5×10^{-6} atm-m^3/mol

C. Toxicity

Fluoranthene has been tested for carcinogenicity, with negative results, in several tests including skin painting studies (as cited in IARC, 1983) and a subcutaneous injection study (as cited in IARC, 1983). USEPA has not classified fluoranthene with regard to its carcinogenicity due to inadequate evidence (IRIS, 1992). However, equivocal evidence for mutagenicity of fluoranthene in short-term bacterial and mammalian tests has been reported (IRIS, 1992).

The RfD for oral exposure to fluoranthene is 0.04 mg/kg-day, based on a study in mice in which subchronic exposure by gavage was associated with kidney toxicity, increased liver weights and alterations in blood characteristics (IRIS, 1992).

A study of fluoranthene's developmental toxicity was performed in which intraperitoneal injection to pregnant mice resulted in an increased rate of fetal resorption (IRIS, 1992).

D. Toxicokinetics

Like other PAH compounds, fluoranthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients. Because of their high lipid solubility, PAHs are believed to be

distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

LaVoie and coworkers (1982 as cited in IARC, 1983) detected the 2,3-dihydrodiol metabolite of fluoranthene which is mutagenic in bacterial tests with an exogenous activation system.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for the PAHs for information regarding federal regulations, standards and guidelines.

G. Toxicity Factors

Reference Doses(mg/kg-dy):

	Subchronic	Chronic
Oral	0.4	0.04

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

FLUORENE
CAS No. 86-73-7

A. Potential Sources and Exposure

Fluorene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	166.7g/mol
Water Solubility	1.69 mg/L at 25°C
Vapor Pressure	7.1×10^{-4} mm Hg
K_{oc}	7300 mL/g
log K_{ow}	4.2
Henry's Law Constant	6.4×10^{-5} atm-m^3 /mole

C. Toxicity

Due to the lack of data on the toxicity of fluorene to humans, IARC (1983) concluded that the available data in experimental animals was inadequate to permit an evaluation of the carcinogenicity of fluorene. The USEPA's Carcinogen Assessment Group has classified fluorene in Group D: not classifiable as human carcinogen (IRIS, 1992).

The RfD for oral exposure to fluorene is 0.04 mg/kg-day, based on subchronic exposure to flluorene in mice by oral gavage. The LOAEL is 250 mg/kg-day based on hematological effects; the NOAEL is 125 mg/kg-day.

D. Toxicokinetics

Like other PAH compounds, fluorene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients.

Due to their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. Elimination of PAHs is primarily via the hepatobiliary tract.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards,guidelines, and criteria.

G. Toxicity Factors

Reference Doses (mg/kg-dy):

	Subchronic	Chronic
Oral	0.4	0.04

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

INDENO(1,2,3-cd)PYRENE

(2,3-Phenylenepyrene; 2,3-o-Phenylenepyrene)
CAS No. 193-39-5

A. Potential Sources and Exposure

Indeno(1,2,3-cd)pyrene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	276.34 g/mol
Water Solubility	6.20×10^{-1} ppm at 25°C
Vapor Pressure	1.0×10^{-10} mm Hg at 20°C
K_{oc}	1.6×10^{6} mL/g
log K_{ow}	6.5
Henry's Law Constant	6.86×10^{-8} atm-m^3/mol at 20°C

C. Toxicity

Although there are no human data that specifically link exposure to indeno(1,2,3-cd)pyrene to human cancers, indeno(1,2,3-cd)pyrene is a component of mixtures that have been associated with human cancer. USEPA has classified indeno(1,2,3-cd)pyrene as a B2 or probable human carcinogen, on the basis of positive results in mice and bacterial mutation assays. Indeno(1,2,3-cd)pyrene has produced tumors in mice following lung implants, subcutaneous injection, and dermal exposure (as cited in IRIS, 1992).

The relative tumorigenic potency of indeno(1,2,3-cd)pyrene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays conducted using similar protocols (USEPA, 1984). The ranking was as follows: B(a)P > dibenzo(ah)anthracene > benzo(b)fluoranthene > benzo(a)anthracene > indeno(1,2,3-cd)pyrene > chrysene.

Indeno(1,2,3-cd)pyrene induced mutations bacterial assays in Salmonella typhimurium strain TA 100 at a concentration of 20 ug/plate and in strain TA 98 at a concentration of 2 ug/plate in the presence of an exogenous metabolic activating system (IARC, 1983). Due to the equivocal mutagenicity testing data, IARC (1983) considered the available evidence inadequate to classify indeno(1,2,3-cd)pyrene as a mutagen.

D. Toxicokinetics

There are no toxicokinetic data of indeno(1,2,3-cd)pyrene in man (USEPA, 1980). In general, many polycyclic aromatic hydrocarbons (PAHs) can produce toxicity after inhalation, oral, or dermal exposure. Thus, it is believed that they are readily absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed function oxidase system and eliminated primarily via the hepatobiliary tract.

E. Ecological Effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses and Cancer Slope Factors are currently not available for this compound. At this time, the USEPA's Carcinogen Assessment Group has not estimated a CSF for indeno(1,2,3-cd) pyrene. Based on the study by Duetsch-Wenzel et. al. (1983), ICF Clement (1986) estimated that indeno(1,2,3-cd)pyrene has a relative potency to benzo(a)pyrene of 0.232. This value can be used in the relative potency method to estimate a cancer slope factor.

H. References

For references, see the PAH toxicity profile.

METHYLNAPHTHALENES
CAS Nos. 90-12-0 and 91-57-6

A. Potential Sources and Exposure

Methylnaphthalenes are used in the chemical synthesis of pesticides. Because of the insolubility of these chemicals, they are very rarely found in water in the environment. Very little is known about exposures and toxicity of these chemicals, however, the reader is referred to the toxicity profile for naphthalene which is believed to have similar properties to 1- and 2-methylnaphthalene.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	142.2 g/mol
Water Solubility	25 mg/L at 25°C , insoluble at 20°C
Vapor Pressure	9.0 at 25°C
K_{oc}	3.4
log K_{ow}	3.8
Henry's Law Constant	45 atm-m^3/mole

C. Toxicity

There are no data available on the toxicity of the methylnaphthalenes. However, based on its structural similarity to naphthalene, it is likely to behave similarly. The reader is referred to the toxicity profile for naphthalene.

D. Toxicokinetics

There are no data available on the toxicokinetic behavior of the methylnaphthalenes. However, based on its structural similarity to naphthalene, it is likely to behave similarly. The reader is referred to the toxicity profile for naphthalene.

E. Ecological Effects

There are little data available on the toxic effects of methylnaphthalene. The reader should consult the toxicity profile for naphthalene, a structurally similar compound.

F. Federal Regulations, Standards, Guidelines, and Criteria

There are none available for the methylnaphthalenes. Those standards that have been promulgated for naphthalene are often used as surrogate values for the methylnaphthalenes.

G. Toxicity Factors

The reader should consult the toxicity profile for naphthalene, a structurally similar compound.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological profile for Naphthalene and 2-Methylnaphthalene. U.S. Public Health Service.

Mackay, D., Shiu, W.Y. and R.C. Ma. 1992. Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Lewis Publishers, Chelsea, Michigan. 1992.

A. Potential Sources and Exposures

Naphthalene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information. Naphthalene is found in moth balls; exposure may arise through inhalation, dermal and ingestion routes.

B. Physical and Chemical Properties

Property	Value
Molecular Weight.	128.2 g/mol
Water Solubility	31.7 mg/L at 25°C
Vapor Pressure	8.2 x 10-2 mm Hg at 25°C
K_{oc}	940 mL/g
log K_{ow}	3.3
Henry's Law Constant	4.8 x 10^{-4} atm-m^3/mole

C. Toxicity

In humans, exposure to sufficient concentrations of naphthalene through inhalation, ingestion, or dermal contact may cause intravascular hemolysis or the less severe symptoms of eye irritation, headache, confusion, tremors, nausea, vomiting, abdominal pain, and bladder irritation (Sittig, 1985). In severe cases hematological effects have included red cell fragmentation, icterus, severe anemia, leukocytosis and dramatic decreases in hemoglobin, hemacrit, and red cell counts. Hemolysis can also lead to renal disease from precipitated hemoglobin (USEPA, 1982). Poisonings have occurred in humans as a result of the ingestion of moth balls as well as from clothing infants in materials that had been stored in moth balls. A study of workers exposed to naphthalene for a period of 5 years found corneal ulceration, cataracts, and some lenticular and general opacities in 8 of the 21 employees examined (Ghetti and Mariani, 1956, as cited in Sandmeyer, 1981).

The National Toxicology Program is currently evaluating naphthalene for carcinogenicity in mice by the inhalation route; final results are not yet available.

D. Toxicokinetics

Naphthalene is rapidly absorbed when inhaled but is more slowly absorbed by ingestion or through the intact skin. No quantitative data on absorption were located. The compound is metabolized (primarily in the liver) to more toxic agents. This occurs rapidly in the adult but very slowly in the newborn (Sittig, 1985). No data were located indicating naphthalene to be an hepatic enzyme inducer.

E. Ecological Effects

A variety of aquatic species has been exposed to naphthalene and most acute tests were under static procedures with unmeasured test concentrations. All but two LC50 effect levels for fish and invertebrate species are in the range of 2,300 to 8,900 ug/L. One embryo-larval test with the fathead minnow demonstrated adverse effects at a test concentration of 850 ug/L.

Daphnia magna is the only tested freshwater invertebrate species for which the acute toxicity of naphthalene has been determined (U.S. EPA, 1978). The reported 48-hour EC50 is 8,570 ug/L.

DeGraeve et al. (1980) conducted flow-through tests with measured concentrations for the rainbow trout and the fathead minnow. The trout appeared to be more sensitive with a 96-hour LC50 of 2,300 ug/L. The 96-hour LC50 for the fathead minnow tested at 14 degrees centigrade was 4,900 ug/L, at 24 degrees centigrade the LC50 was 8,900 ug/L. The LC50 of 150,000 ug/L for the mosquitofish appears to be atypical but the result cannot be discounted.

LC50 (96 h) values for the polycheate, *Neanthes arenaceodentata*, (Pacific oyster), and the grassshrimp are 3,800, 199,000 and 2,350 ug/L, respectively. The 24-hour LC50 values for one fish and two saltwater shrimp species range from 2,400 to 2,600 ug/L.

With the exception of the mosquitofish and the Pacific oyster, all LC50 and EC50 values, regardless of test method, fall within the narrow range of 2,300 to 8,900 ug/L for 9 freshwater and saltwater species.

Tests have been conducted to determine the chronic toxicity of naphthalene to ecological receptors. An embryo-larval test has been conducted with the fathead minnow and the resultant chronic value is 620 ug/l. When this concentration is divided by the geometric mean LC50 value of 6,600 ug/L for this species an acute-chronic ratio of II is obtained. No other species have been tested under chronic conditions.

There is only one reported test that determined an apparent equilibrium bioconcentration factor for naphthalene. After nine days, the bioconcentration factor for a copepod was 5,000. Bioconcentration data for other species for exposures of one hour to one day range from 32 to 77 and indicate that equilibrium does not occur rapidly when those results are compared to the nine-day value of 5,000.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL)
  8-hour Time Weighted Average: 50 ppm

- Drinking Water Health Advisories:

One-day Health Advisory (child)	0.5 mg/L
Ten-day Health Advisory (child)	0.5 mg/L
Longer-term Health Advisory (child)	0.4 mg/L
Longer-term Health Advisory (adult)	1 mg/L
Lifetime Health Advisory	0.02 mg/L
Drinking Water Equivalent Level	0.1 mg/L

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Chronic	Subchronic
Oral	0.04	0.04

H. References

For references, see the PAH toxicity profile.

INORGANIC COMPOUNDS

ALUMINUM
CAS No. 7429-90-5

A. Potential Sources and Exposure

Aluminum is not an essential element in the human diet. Humans are exposed to aluminum primarily via ingestion (diet and water). Exposure also occurs via inhalation, and skin absorption, and intravenously through kidney dialysis. Aluminum containing antacids and other medications are the major source of total aluminum exposure in humans. The average intake of aluminum ranges from 10 to 20 mg/day. The total amount of aluminum consumed through ingestion of food is approximately between 2 to 100 mg/day. This estimate includes natural content, intentional and unintentional additives. Ambient aluminum concentrations in raw water range from 10 to 1,000 ug/l. Alum-treated drinking water at the tap contains 100 to 500 ug/l normally, but higher values up to 2 mg/l are not uncommon.

B. Physical and Chemical Properties

Property	Value
Molecular weight	26.98 g/mole

C. Toxicity

The toxicity of aluminum is poorly understood. Aluminum can adversely affect the bone and the brain. High risk groups include children and adults with renal failure, preterm infants with immature kidney function, individuals with gastrointestinal inflammatory disease, and individuals with blood-brain barrier damage. At this time, no causative link has been established between exposure to aluminum and the development of Alzheimer's Disease. Aluminum can be a selective and potent neurotoxin. Aluminum has been linked with specific encephalopathy dialysis dementia.

D. Toxicokinetics

When ingested by healthy individuals, aluminum (Al) is only slightly absorbed. Gut uptake depends on Al species, pH, interaction with co-ingested compounds and individual metabolism. Very little is known about human absorption and metabolism of aluminum. Apparently, at some level of exposure between 125 to 1,000 mg/day, the body may absorb Al faster than it can excrete it and Aluminum may begin to accumulate. Under normal physiological conditions, almost all the aluminum entering the blood is eliminated in the urine.

E. Ecological Effects

Toxicity of aluminum/complexed aluminum species to aquatic biota is influenced by the pH

and temperature of the water and what type of ligand is complexed to aluminum. Several different aluminum (fulvic acid, fluoride, sulfate, organic silicates) complexes may present in natural waters. Aluminum can bioaccumulate in freshwater and marine plants. Under acidic conditions in a water environment, aluminum can form monomeric or polymeric hydrates which enhance its bioavailability. Dissolved aluminum is highly toxic to crop roots and forests. Total aluminum residues are usually low in invertebrate and fish species under pristine conditions. A number of chronic toxic effects on aquatic invertebrates and fish have been observed following exposure to different aluminum species. Most of the aquatic toxicity studies have been conducted at an acidic pH and there is generally an effect on the biological ion flux within the test organism. Acid soluble forms of aluminum appear to be the most toxic, to biota and using acid-soluble data, guidelines for the protection of aquatic species has been established (see next section).

F. Federal Regulations, Standard, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL)

metal dust	15 mg/m^3
respirable fraction, pyro powders, welding fumes	5 mg/m^3
soluble salts, alkyls	2 mg/m^3

- ACGIH Threshold Limit Value

metal dust & oxide	10 mg/m^3
pyro powders & welding fumes	5 mg/m^3
soluble salts & alkyls	2 mg/m^3

Drinking Water:
USEPA Secondary Maximum Contaminant Level 0.05 - 0.2 mg/l

Water:
Criteria to protect freshwater aquatic life when the pH for water ranges from 6.5 - 9.0.

- Ambient Water Quality Criteria, Freshwater

Acute	87 ug/l (four-day average)
	750 ug/l (one-hour limit)

Various state guidelines may differ from federal regulations and should be consulted.

H. Reference

Agency for Toxic Substances and Disease Registry (ATSDR), 1991. Draft Toxicological profile for Aluminum. U.S. Public Health Service.

Aluminum and Health, An Update on Alzheimer's Disease. May 1987. Environmental

Research, Health and Safety Aluminum Association, Inc.

United States Environmental Protection Agency, (USEPA) 1988. Ambient Water Quality Criteria for Aluminum, Office of Water. Washington, D.C.

ARSENIC
CAS No. 7439-96-5

A. Potential Sources and Exposure

Arsenic is a naturally-occurring metal that has been widely used in rat and ant poisons, herbicides, some medicines, and in arsenic-treated (pressure treated) wood. Some areas of the U.S. have unusually high natural levels of arsenic in rock, which can lead to high concentrations in soil and water. There are several forms of arsenic to which an individual might be exposed and the toxicity is dependent upon the type of arsenic compound.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	74.92 g/mol
Water Solubility	insoluble at 25°C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

C. Toxicity

The toxicity of arsenic depends upon its chemical form and route, dose, and duration of exposure. In general, arsenites (As^{3+}) are more toxic than arsenates, soluble arsenic compounds are more toxic than insoluble compounds, and inorganic arsenic compounds are more toxic than organic derivatives (ATSDR, 1992).

Arsenic is an irritant of the skin, mucous membranes, and gastrointestinal tract. Symptoms of acute toxicity include vomiting, diarrhea, convulsions, and a severe drop in blood pressure. Subchronic exposures may result in hyperpigmentation of the skin, persistent headache, and lethargy. Chronic exposures to inorganic arsenic compounds may lead to neurotoxicity of both the peripheral and central nervous systems as well as peripheral vascular disease and skin lesions.

The most potent forms of the compound are the trivalent arsenic compounds. These compounds can bind to sulfhydral groups on proteins and enzymes. Arsenic affects mitochondrial enzymes and impairs tissue respiration, which seems to be related to the cellular toxicity (Klaassen, 1995). Arsenic compounds are inducers of metallothionein.

The USEPA classifies arsenic as a Group A - Human Carcinogen based on epidemiological studies in which a causal association between exposure and skin cancer was observed

D. Toxicokinetics

Arsenic (trivalent or pentavalent insoluble forms) is well absorbed from the gastrointestinal tract. Limited data suggest nearly complete absorption of soluble forms of trivalent and pentavalent arsenic. Deposition of arsenic in the airway is dependent on particle size and chemical form. Excretion of absorbed arsenic is mainly via the urine. Arsenic has a predilection for the skin and is excreted by desquamation of skin and in sweat, particularly during periods of profuse sweating. It also concentrates in nails and hair. Dimethyl arsenic is the principal detoxication product (Klaassen, 1995).

E. Ecological Effects

Bioaccumulation
Arsenic is neither a major contaminant of aquatic plants nor does it normally concentrate in either freshwater or marine fish. Only in extreme cases of ambient pollution does it contaminate aquatic plants and there are few reports of tissue residues exceeding health guidelines in fish. However, some reports do demonstrate rather high levels in invertebrates, for example, exceeding 30 mg/kg.

Toxic Effects to Aquatic Organisms
Although insufficient data exist to determine the definitive acute toxicity to organisms, fresh or marine, work on the topic indicates that large doses of Arsenic (greater than 1 mg As/L) are required to induce acute toxic effects in both plants and invertebrates. Chronic effects for both invertebrates and fish exposed to inorganic Arsenic have been reported and require a relatively large dose, typically > 5 mg As/L.

Toxic Effects to Wildlife (tertiary)
To be absorbed by terrestrial plants, arsenic compounds must be in a mobile form in the soil. Unless located in an area where arsenic concentrations are exceptionally high, plants will distribute accumulated arsenic in nontoxic amounts throughout the plant body. Most plants will yield significantly less of a crop when concentrations become 3 to 28 mg/L of water soluble arsenic and 25 to 85 mg/kg of total arsenic. Air concentrations up to 3.9 μg As/m^3 have also been seen to have adverse effects on vegetation.

Effects on soil biota and insects remains limited but generally it is believed that soil microorganisms are capable of tolerating relatively high concentrations of arsenic.

In birds, signs of inorganic trivalent arsenite poisoning include muscular incoordination, debility, slowness, jerkiness, falling hyperactivity, fluffed feathers, drooped eyelid, huddled position, immobility, and seizures. Studies suggest that lethal acute inorganic arsenic

poisoning results in the destruction of blood vessels lining the gut thereby causing decreased blood pressure and subsequent shock.

Mammalian exposure to arsenic occurs primarily through ingestion. Acute episodes of poisoning are characterized by high mortality and morbidity. Signs of arsenic toxicosis include intense abdominal pain, staggering gait, extreme weakness, trembling, salivation, vomiting, diarrhea, prostration, collapse, and death. Chronic poisoning is infrequently seen due to the fact that excretion and detoxification are rapid.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	
Organic As Compounds	500 ug/m^3
Inorganic As Compounds	10 ug/m^3
Action Level	5 ug/m^3
ACGIH Threshold Limit Value (TLV)	
As and soluble compounds	200 ug/m^3

Drinking Water:

USEPA Maximum Contaminant Level	0.05 mg/L

Water:

Ambient Water Quality Criteria:	
Ingesting water and organisms	2.2×10^{-6} mg/L
Ingesting organisms only	1.75×10^{-5} mg/L

G. Toxicity Factors

Non-carcinogen Toxicity Factors	
Oral (RfD)	3.0×10^{-4} mg/kg-day (IRIS, 1993)
Inhalation (RfC)	No data
Carcinogen Toxicity Factors	
Oral Carcinogenic Slope Factor	1.5 $(mg/kg\text{-}day)^{-1}$ (IRIS, 1995)
Inhalation Unit Risk	4.3×10^{-3} per (ug/m^3) (IRIS, 1995)

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological profile for arsenic. U.S. Public Health Service.

Eisler, Ronald. *Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD, 1988.

Integrated Risk Information System (IRIS) on-line database, accessed on 6/96.
Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

BARIUM

CAS No. 7440-39-3

A. Potential Sources and Exposure

Barium is a naturally-occurring, highly abundant metal that is used in various alloys, in paints, soap, paper, rubber, and in the manufacture of ceramics and glass. Barium sulfate, an insoluble compound, is used in diagnoses made via x-rays.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	137.3 g/mol
Water Solubility	decomposes at 25°C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

C. Toxicity

The toxicity of barium compounds depends on their solubility. Acute oral exposure to barium can cause gastroenteritis, muscle paralysis, and cardiovascular effects. Chronic inhalation of barium-containing dust can cause a reversible, benign pneumoconiosis (Klaassen, 1995). There is no evidence that barium is carcinogenic.

D. Toxicokinetics

The soluble compounds of barium are absorbed, and small amounts accumulate in the bone, and to a lesser extent in the kidney, spleen, muscle, heart, brain, and liver (Klaassen, 1995). Although some barium is excreted in the urine, it is reabsorbed by the renal tubules. The major route of excretion is the feces.

E. Ecological Effects

Bioaccumulation

Barium is accumulated by freshwater and marine organisms but is not concentrated when passed through the food chain. Therefore, residue in invertebrates and higher species rarely exceed 10 mg/kg while residues in plants and algae can greatly exceed this value.

Toxic Effects to Aquatic Organisms

Little information has been determined about the toxicity of barium in marine and freshwater

organisms. Barium is believed to be moderately acutely toxic and preliminary guidelines employ a level of 5 mg Ba/L for the protection of aquatic life.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	
soluble Ba compounds	0.5 mg/m^3
barium sulfate	
total dust	10 mg/m^3
respirable fraction	5 mg/m^3
ACGIH Threshold Limit Value (TLV)	
barium	0.5 mg/m^3
barium sulfate (total dust)	10 mg/m^3

Drinking Water:

USEPA Maximum Contaminant Level	2 mg/L

Water:

Ambient Water Quality Criteria:	
Ingesting water and organisms	1 x 10^{+3} ug/L

G. Toxicity Factors

Oral Reference Dose (mg/kg-day):	7 x 10^{-2}
Inhalation RfC	Not available

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological profile for barium. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

BERYLLIUM
CAS No. 7440-41-7

A. Potential Sources and Exposure

Beryllium is an uncommon metal that is obtained by extraction from mineral ores. Beryllium is incorporated into alloy metals that are used in jet engine parts and electrical components. Pure beryllium metal is used in parts for nuclear weapons, nuclear reactors, precision instruments, and aircraft brakes. Beryllium in the environment largely results from coal combustion.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	9.01 g/mol
Water Solubility	insoluble at 25°C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

C. Toxicity

Contact dermatitis is the most common beryllium-related effect. Exposure to soluble beryllium compounds can result in papulovesicular lesions on the skin. This is a cell-mediated delayed-type hypersensitivity reaction. Exposure to insoluble beryllium compounds results can result in a chronic granulomatous lesion which may be ulcerative.

Acute pulmonary disease from the inhalation of beryllium is an inflammatory reaction of the entire respiratory tract, and in the most severe cases results in acute pneumonitis, the symptoms of which include cough, shortness of breath, and fatigue. These symptoms can persist and even worsen after exposure to beryllium has been discontinued. Chronic inhalation exposures to low concentrations of beryllium can produce chronic granulomatous disease (berylliosis), which results in inhibited breathing efficiency (Klaassen, 1995).

The USEPA classifies beryllium as a Group B2 - Probable Human Carcinogen based on animal studies in which beryllium has been shown to induce lung cancer via inhalation.

D. Toxicokinetics

Gastrointestinal absorption of beryllium probably only occurs in the stomach due to acidic conditions. Beryllium dust is well absorbed in the lung. Distribution is rapid to all tissues, but the highest concentration is initially seen in the liver and then the bone (Klaassen, 1995). The half-life in tissues is relatively short, except in the lungs, and a variable fraction of the administered dose is excreted in the urine (ATSDR, 1991).

E. Ecological Effects

Bioaccumulation
Bioaccumulation occurs for beryllium but the concentration factors are low, usually < 1 mg/L.

Toxic Effects to Aquatic Organisms
Neither acute nor chronic toxicity of beryllium has been documented adequately. Acute toxicity is, however, strongly affected by water hardness. Recommended concentrations, based on available data, are 0.11 and 1.10 mg/L for soft water and hard water, respectively.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	
Be and Be compounds	0.002 mg/m^3
ACGIH Threshold Limit Value (TLV)	
Be and Be compounds	0.002 mg/m^3

Drinking Water:

USEPA Maximum Contaminant Level (proposed, 1991)	0.001 mg/L

Water:

Ambient Water Quality Criteria:	
Ingesting water and organisms	0.68-68 ng/L
Ingesting organisms only	11.7-1,170 ng/L

G. Toxicity Factors

Non-Carcinogen Toxcity Factors	
Oral RfD	5.0 x 10^{-3} mg/kg-day (IRIS, 1993)
Inhalation RfC	No data

Carcinogen Toxicity Factors

Oral Slope Factor	4.3 $(mg/kg\text{-}day)^{-1}$ (IRIS, 1992)
Inhalation Unit Risk	2.4×10^{-3} per (ug/m^3) (IRIS, 1992)

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological profile for beryllium. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

CADMIUM
Cas. No. 7440-43-9

A. Potential Exposure

Cadmium and cadmium compounds are typically used as a protective coating for other metals; in the production of metal alloys; fluorescent lamps, semiconductors, photocells, and jewelry; and in batteries, nuclear reactors, engraving and pesticides. Food and cigarette smoke are the largest potential sources of cadmium exposure for the general population. Ingestion and inhalation are primarily routes of exposure for cadmium. Average cadmium levels in foods within the United States range from 2 to 40 ug/kg. The average level of cadmium in cigarettes range from 1,000 to 3,000 ug/kg. Workers can be exposed to cadmium via inhalation or dermal contact while soldering or welding metal. Shellfish can be a major source of cadmium and can contain levels from 100 to 1,000 mg/kg.

Cadmium is also a concern in agricultural soils where sewage sludge is used as compost because it is more readily taken up by plants than other metals. The uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys).

B. Physical and Chemical Properties

Property	Value
Molecular Weight	112.4

C. Toxicity

Acute inhalation of cadmium fumes or dust can cause destruction of lung epithelial cells, resulting in pulmonary edema, tracheobronchitis and pneumonitis. As a result of breathing high cadmium levels, the acute toxicity can range from a slight irritation of the upper respiratory tract to death. High-level acute oral exposure to cadmium irritates the gastrointestinal epithleum causing nausea, vomiting, and abdominal pain. Breathing lower levels of cadmium for long period of time, can lead to accumulation of cadmium in the kidneys thus causing severe kidney damage. Heavy smoking has been reported to considerably increase tissue cadmium levels (ATSDR, 1992). Non-occupational inhalation exposure to cadmium is unlikely to be excessive enough to cause respiratory effects. However, chronic inhalation exposure at lower levels can lead to decreased pulmonary function and emphysema. Based on epidemiological and animal studies, it appears that cadmium-induced emphysema is related only to cadmium exposure via inhalation (USEPA, 1985a).

The lung and kidney are the main target organs for cadmium toxicity following intermediate or chronic duration exposure by the inhalation or oral routes. The earliest clinical signs of cadmium poisoning are proteinuria, glucosuria, and aminoaciduria (USEPA, 1985a). Cadmium damages the renal tubules resulting in an inhibition of tubular reabsorption, but rarely results in renal failure (ATSDR, 1992). Prolonged exposure to cadmium which causes renal dysfunction can lead to painful and debilitating bone disease after inhalation or oral exposure as a result of cadmium's effect calcium metabolism (ATSDR, 1992).

A toxicokinetic model is available to determine the level of chronic human oral exposure which results in 200 ug Cd/g wet human renal cortex (the highest renal level not associated with significant proteinuria, the NOAEL). The model assumes 2.5% absorption of Cd from food or 5% from water, and that 0.01% day of the Cd body burden is eliminated per day (USEPA, 1985b). The model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively. Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water, an oral RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent oral RfD for Cd in food is 0.001 mg Cd/kg/day. A risk assessment for an inhalation RfD for cadmium is under review by an EPA work group.

USEPA has classified cadmium as a Group B1 or probable human carcinogen. This classification is based on occupational epidemiology studies that have shown an increased risk of lung cancer in workers exposed to cadmium via inhalation. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers (Thun et. al., 1985 as cited in USEPA, 1985b). USEPA has estimated a cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ through inhalation route only. The CPF is based on several animal studies (Takenaka et al., 1983; Sanders and Mahaffey, 1984).

D. Toxicokinetics

Cadmium compounds are poorly absorbed from the skin and intestinal tract, but relatively well absorbed from the respiratory tract. Following ingestion or inhalation, cadmium is distributed to most tissues of the body. Initially, highest levels are found in the liver. Later, relocation occurs and highest concentrations appear in the renal cortex (ATSDR, 1992). In a study exposing rats daily to cadmium fumes, the distribution of Cd in the tissue was kidney > lung > liver > spleen > aorta > blood (ATSDR, 1992). Blood levels in the exposed animals were no different from those of unexposed animals. Similar distributions were found using guinea pigs and monkeys.

Following oral administration, 1-5% of the dose is absorbed. Variations in absorption are induced by many factors such as age, dietary calcium, and dietary protein levels. Excretion occurs primarily via the kidney at a very slow rate. The biological half-life of cadmium is estimated to be on the order of decades in humans (ATSDR, 1992).

E. Ecological Effects

Aquatic and terrestrial organisms bioaccumulate cadmium. Cadmium bioconcentrates in freshwater and marine animals to concentrations hundreds to thousands times higher than the cadmium concentrations in the water.

Levels of cadmium in plant tissue which are considered to be phytotoxic range from 5-700 ppm (Chaney, 1982), 5-30 ppm (Kabata-Pendias and Pendias, 1984) and 8-15 ppm (Davis et al., (1978). NAS (1980) has established a maximum dietary cadmium concentration chronically tolerated by livestock of 0.5 ppm (based upon cadmium residues in animal products used in human foods).

Aquatic and terrestrial organisms bioaccumulate cadmium. Cadmium bioconcentrates in freshwater and marine animals to concentrations hundreds to thousands times higher than the cadmium concentration in the water.

Of the 44 freshwater genera for which genus mean acute toxicity values are available (USEPA, 1984), the most sensitive genus, Salmo, trout is 3,400 times more sensitive than the most resistant genus, Carassius goldfish. Of the freshwater species, rainbow and brown trout appear to be extremely sensitive to cadmium when acutely exposed to concentrations ranging from 1 ug/l to 4 ug/l. The freshwater final acute value of 3.589 ug/l at hardness of 50 mg/l is used to protect against Salmo gairdneri, rainbow trout. However, brown trout is more sensitive than rainbow trout based on an EC50 of 1.63 ug/l from a static test. Chronic mean values derived from acute toxicity values representing 44 genus were used to calculate a final freshwater chronic value of 0.6582 ug/l at hardness of 50 mg/l. The genus mean chronic values for Moina and Daphnia, both cladocerans are below the final freshwater chronic value.

Growth reduction is a major factor toxic effect observed with freshwater aquatic plants and reported values are in the range of concentrations causing chronic effects on aquatic animals. In addition, the lowest toxicity values for freshwater fish and invertebrates species are lower thana the lowest values for aquatic plants.

The acute toxicity of cadmium generally increases as salinity increases. The acute values for saltwater invertebrates species range from 41.29 ug/l to 135,000 ug/l for an oligochaete worm. Saltwater mollusks have species Mean Acute Values from 227.9 ug/l for the Pacific oyster to 19,170 ug/l for the mud snail. Saltwater fish species were generally more resistant to cadmium than freshwater fish species with acute values ranging from 779.8 ug/l for the Atlantic silverside to 50,570 ug/l for the mummichog. Of the 33 saltwater genera for which acute values are available, the most sensitive, mysidoposis is 2,000 times more sensitive than the most resistant, Monopylephorus, oligoclaete worms. The saltwater final acute value is 85.09 ug/l and is slightly above the Species Mean Acute Value of 78 ug/l for the American Lobster. For the two saltwater species (mysids) for which both chronic and acute toxicity ratios exist, a final saltwater chronic value of 9.345 ug/l was obtained.

Concentrations causing 50% reductions in the growth rates of marine diatoms range from 60 ug/l to 175 ug/l. One of the most sensitive marine plants is a red algae, Champia parvula due to growth inhibition at cadmium concentration of 22.8 ug/l.

Bioconcentration factors (BCFs) determined with a variety of saltwater invertebrates range from 5 to 3,160. BCF for bivalve mollusks were above 1,000 in long exposures with no indication that a steady state had been reached.

F. Federal Regulations, Standards, Guidelines and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL)
  8-hour Time Weighted Average

cadmium fume	0.1 mg/m^3
cadmium dust	0.2 mg/m^3

  Ceiling:

cadmium fume	0.3 mg/m^3
cadmium dust	0.6 mg/m^3

- ACGIH Threshold Limit Value

cadmium dusts & salts	0.05 mg/m^3
cadmium oxide production	0.05 mg/m^3

- NIOSH Immediately Dangerous to Life and Health for dust & fume: 40 mg/m^3

Food:

Food Drug Administration Limit	15 ppm
in bottled water	0.01 mg/l

Drinking Water:

- USEPA Maximum Contaminant Level (MCL): 0.005 mg/l
- USEPA Maximum Contaminant Level Goal (MCLG): 0.005 mg/l
- Drinking Water Health Advisories:

One-day Health Advisory (child)	0.04 mg/l
Ten-day Health Advisory (child)	0.04 mg/l
Longer-term Health Advisory (child)	0.005 mg/l
Longer-term Health Advisory (adult)	0.02 mg/l
Lifetime Health Advisory	0.02 mg/l

Water:

- Ambient Water Quality Criteria, Freshwater:

Acute	3.9 ug/l (1-hour average)
Chronic	1.1 ug/l (4-day average)
at hardness of 50 mg/l as $CaCO_3$	

- Ambient Water Quality Criteria, Marine:

Acute	4.3 ug/l (1-hour average)
Chronic	9.3 ug/l (4-day average)

G. Toxicity Factors

Reference Doses:

	Chronic
Oral	Food 1.0×10^{-3} mg/kg/day
	Water 5×10^{-4} mg/kg/day

Cancer Potency Factor:
Inhalation 6.1 $(mg/kg/day)^{-1}$

Various state guidelines may differ federal regulations and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR), 1992. Draft Toxicological profile for Cadmium. U.S. Public Health Service.

Barlow, S.M. and F. M. Sullivan. 1982. Reproductive hazards of industrial chemicals: An evaluation of animal and human data. Academic Press, New York.

Chaney, R.L. 1982, Fate of toxic substances in sludge applied to cropland. In: Proceedings of an International Symposium on Land Application of Sewage Sludge. Tokyo, Japan.

Davis, R.D., Beckett, P.H.T. and Wollan, E., 1978. Critical levels of twenty potentially toxic elements in young spring barley. Plant and Soil 49:95.

Integrated Risk Information System (IRIS) on-line database, accessed on 6/4/92.

Kabata-Pendias, A. and Pendias, H., 1984. Trace Elements in Soils and Plants. CRC Press, Boca Raton, Florida.

National Academy of Sciences (NAS). 1980. Drinking water and health. Volume 3. Safe Drinking Water Committee, National Research Council, National Academy of Sciences.

National Academy Press. Washington, D.C.

Sanders, C.L. and J. A. Mahaffey. 1984. Carcinogenicity of single and multiple intratracheal installations of cadmium oxide in the rat. Environ. Res. 33: 227-233.

Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer and G. Oberdoerster. 1983. Carcinogenicity of cadmium aerosols in Wistar rats. J. Natl. Cancer Inst. 70: 367-373.

U.S. Environmental Protection Agency (USEPA). 1984. Ambient Water Quality Criteria for Cadmium. Office of Water Regulations and Standards, Criteria and Standards Division. EPA 440/5-84-32, PB 85-227031. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1985a. Updated mutagenicity and carcinogenicity assessment of cadmium. Final Report. Office of Health and Environmental Assessment. EPA-600/8-83/025F. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1985b. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1992. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms.

CHROMIUM (III)

(Chromic Chloride, Chromium Chloride Anhydrous, Chromium Chloride, chromic ion, trivalent chromium, Cr3+)
CAS No. 16065-83-1

A. Potential Sources and Exposure

In general, chromium (Cr) III is a naturally occurring compound. It tends to form stable complexes with negatively charged organic and inorganic species such as those found in soils. Chromium (III) is used as brick lining for high-temperature industrial furnaces. The chromium compounds, mostly chromium (III) and (VI) forms produced by the chemical industry are used for chrome plating, dye manufacturing, leather tanning, wood preservatives, and treatment of cooling water as a corrosive agent. The most common routes of exposure to chromium, (III) are ingestion of food, inhalation and dermal contact (ATSDR, 1990). Various methods of processing, storage, and preparation can alter the chromium content of food.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	51.99 g/mol

C. Toxicity

Chromium is a sensitizing agent that produces allergic skin reactions or asthma following subsequent exposures. As a very strong irritant, it can produce dermatitis, dermatosis, eczema, erythema, and skin ulceration. Nasal septum perforations are attributed to occupational chromium inhalation exposure. Chromic acid has a direct corrosive effect on the skin and mucous membranes of the upper respiratory tract. It is difficult to attribute these effects specifically to chromium (III) since chromium was in the form of dust which may also contain Cr (VI).

Chromium (III) compounds have been administered to animals by the oral route, with little or no toxicity observed. The oral RfD of 1 mg/kg/day is based on a subchronic study in which rats were fed chromic oxide baked in bread at dietary levels. No effects due to chronic oxide exposure were observed. This RfD is limited to metallic chromium (III) which form insoluble salts (IRIS, 1992). Very limited data suggest that chromium (III) may have respiratory effects on humans.

The inhalation RfC is under review by the USEPA work group and is based on a human study with LOAEL of 0.002 mg Cr (VI)/m^3 as chromic acid. The critical effect was nasal mucosa

atrophy (IRIS, 1992).

D. Toxicokinetics

Chromium (III) compounds are not readily absorbed either by inhalation or oral routes of exposure. In the gastrointestinal tract, about 0.5-20% chromium (III) is absorbed (Anderson, 1981). Absorption into the bloodstream into the lungs has been estimated to be approximately 5%-30% for chromium (III). Both chromium (III) and (VI) can penetrate human skin to some extent. Factors influencing dermal absorption include the chromium salt employed, the valence state (III or VI), anionic form, concentration, and pH. Systemic toxicity has been observed in humans following dermal exposure to chromium compounds.

Once absorbed, chromium (III) is transported by binding to proteins in the blood (Hopkins and Schwartz, 1964 as cited in USEPA, 1984a). There appears to be significant in vivo conversion of chromium (VI) to chromium (III). Chromium (III) compounds are essential to normal metabolism. Chromium (III) compounds are capable of forming complexes with nuclei acids and proteins and is cleared rapidly from the blood and slowly from tissues, while chromium (VI) is cleared slowly from blood and rapidly from tissues. Chromium is distributed primarily to the liver, spleen, bone marrow, lung, and kidney. Chromium (III) can be transferred to the fetus through the placenta and to infants via breast milk. Excretion primarily occurs through the urine (50 to 60%) with some fecal elimination (about 8%) (Onkelinx, 1977 as cited in USEPA, 1984a). The remainder is deposited in various tissue compartments and has a long biological half-life. Chromium (VI) is eliminated much faster than chromium (III).

E. Ecological Effects

Chromium exists in natural systems as either trivalent chromium (chromium III) or hexavalent chromium (VI). Hexavalent chromium is the more soluble form. Trivalent chromium tends to form stable complexes with negatively charged organic or inorganic species. Trivalent chromium is relatively insoluble in salt water since it tends to precipitate in a series of complex reactions with the various inorganic ions in seawater.

Acute toxicity values for chromium (III) for 20 fresh water animal species in 18 genera ranged from 2,221 for a mayfly to 71,060 for a caddis fly. Hardness has a significant influence on toxicity, with chromium (III) being more toxic in soft water. A life cycle test with Daphnia Magna in soft water gave a chronic value of 66 ug/l. In a life cycle test with fathead minnow, the chronic value was 1,025 ug/l. Toxicity data are available for two freshwater plant species. A concentration of 9,900 ug/l inhibited growth of Eurasion Water Millfoil roots, and a green alga was affected at 397 ug/l in soft water.

There are only two acute values for chromium (III) in salt water, 10,300 ug/l for the eastern oyster and 31,500 for the mummichog (USEPA, 1987).

F. Federal Regulations, Standards, Guidelines and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL) 0.5 mg/m^3
- ACGIH Threshold Limit Value 0.5 mg/m^3

Drinking Water:

- USEPA Maximum Contaminant Level (MCL) (total chromium) 0.1 mg/l
- USEPA Maximum Contaminant Level Goal (MCLG) 0.1 mg/l
- Drinking Water Health Advisories (total chromium):

One-day Health Advisory (child)	1.0 mg/l
Ten-day Health Advisory (child)	1.0 mg/l
Longer-term Health Advisory (child)	0.2 mg/l
Lifetime Health Advisory (adult)	0.1 mg/l
Longer-term Health Advisory (adult)	0.8 mg/l
Drinking Water Equivalent (DWEL)	0.2 mg/l

Water:

- Ambient Water Quality Criteria, Freshwater:

Acute (hardness dependent)	1700 ug/l
Chronic (hardness dependent)	210 ug/l

- Ambient Water Quality Criteria, Marine:

Acute	10,300 ug/L

G. Toxicity Factors

Reference Doses:

	Subchronic
oral	1.0 mg/kg/day

State guidance may differ from federal guidance and should be consulted.

H. References

Agency of Toxic Substances and Disease Registry (ATSDR), 1992. Toxicological Profile for Chromium III

Chromium. U.S. Department of Health and Human Services.

Integrated Risk Information System (IRIS), 1992. On-line database accessed on 1/22/92.

Olsen, K.R. and R.C. Harrel. 1973. Effects of salinity on the toxicity of mercury, copper, and chromium for Rangia cuneata. Contrib. Mar. Science, 17:9.

Reish, D.J. and R.S. Carr. 1978. The effect of heavy metals on the survival, reproduction, development and life cycles of two species of polychaetous annelids. Mar. Pollut. Bull. 9:24.

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for Chromium. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1984. Health Effects Assessment for Trivalent Chromium. PB86-134467. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1984a. Health Assessment document for chromium, EPA-600/8-83-014F.

U.S. Environmental Protection Agency (USEPA). 1987. Quality Criteria for Water. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). April, 1992. Office of Water. Drinking Water Regulations and Health Advisories.

COPPER
CAS No. 7440-50-8

A. Potential Sources and Exposure

Metallic copper (Cu) is used for wires due to its conductive properties and copper compounds are used as insecticides, algicides and molluscicides as well as for electroplating reagents. Copper tends to form complexes with both organic and inorganic ligands, such as soils. Copper is used in water distribution piping, cooking utensils, coinage, and natural gas piping. Exposure to copper for the general population is typically via ingestion of drinking water which has passed through copper piping. Occupational exposure to copper occurs primarily through inhalation of fumes or dusts generated during welding.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	63.5 g/mol

C. Toxicity

Various effects from acute/subchronic exposures of humans to ingested copper/copper sulfate have been reported: nausea, vomiting, epigastric pain, headache, dizziness, and abdominal cramps. Dermal exposure to relatively high doses of copper salts may produce skin irritation and eczema. In eyes, copper salts may cause conjunctivitis, and even ulceration and turbidity of the cornea. Inhalation of copper fumes and dust may cause irritation of upper respiratory tract, metallic taste in the mouth, nausea, metal fume fever and in some instances, discoloration of skin and hair. The inhalation of dusts and mists of copper salts through occupational exposure may result in irritation of the nasal mucous membranes and the pharynx, and ulceration and perforation of the nasal septum. No adverse effects via the occupational exposure of copper welders to copper fumes were reported at concentrations up to 0.4 mg Cu/m^3.

Chronic copper toxicity occurs in humans with Wilson's disease, a genetic condition of copper metabolism. Patients with this condition are unable to adequately metabolize copper at normal exposure level, resulting in damage to erythrocytes, kidneys, corneas, and the central nervous system (Scheinberg and Sternlieb, 1969).

Chronic exposure (3 to 15 years) to copper sulfate by vineyard sprayers is reported to have resulted in copper-containing benign granulomas in the lungs (Pimental and Menezes, 1975).

D. Toxicokinetics

Copper may be absorbed by dermal, oral, or inhalation exposure routes. Copper absorption is influenced by climate, soil chemistry, diet, water softness, and pH. Bioaccumulation in biological

organisms does not tend to occur upon repeated exposure indicating fairly rapid excretion.

E. Ecological Effects

The toxicity of copper to aquatic life is related primarily to the presence of the free cupric ion, Cu^{2+} and possibly some of the hydroxy complexes (USEPA, 1984). The Cu^{2+} forms stable complexes and precipitates with many inorganic and organic constituents in natural waters. Generally, the concentration of free ion is low compared to total copper present in the water. Organic and inorganic copper complexes appear to be less toxic than the free cupric ion. Aquatic toxicity studies indicate that increasing alkalinity, hardness, and total organic carbon in natural waters decreases copper toxicity. Three major classes of compounds contribute to alkalinity in natural waters. These classes include hydroxide, carbonates and bicarbonates. More copper is complexed as carbonate species, resulting in a significant reduction of the free Cu^{2+}. A change in ionic strength of water alters sensitivity of some aquatic species to copper. The copper ion is significantly more toxic in lower ionic strength waters such as tap water (USEPA, 1984).

Acute toxicity data are available for species in 41 genera of freshwater animals. At a hardness of 50 mg/L the genera range in sensitivity from 16.74 ug/L for *Pytochocheilus* (northern squawfish) to 10,240 ug/L for *Acroneuria* (stonefly). The next most sensitive species after *Pytchocheilus* were the Cladoceran and amphipod species (USEPA, 1984). Data for eight species indicate that acute toxicity decreases as hardness increases. Additional data for several species indicate that toxicity also decreases with increases in alkalinity and total organic carbon.

Chronic values are available for fifteen freshwater species and range from 3.873 ug/L for brook trout to 60.36 ug/L for northern pike (USEPA, 1984). Fish and invertebrate species seem to be about equally sensitive to the chronic toxicity of copper.

The acute sensitivities of saltwater animals to copper range from 5.9 ug/L for the blue mussel to 600 ug/L for the green crab. Chronic tests in a mysid observed adverse effects at 77 ug/L but not at 38 ug/L, yielding an acute-chronic ratio of 3.346 (USEPA, 1984). Effects were observed in several saltwater algal species between 5 and 100 ug/L. Oysters can bioaccumulate copper up to 28,000 times, and become bluish-green, apparently without significant mortality. In long-term exposures, the bay scallop was killed at 5 ug/L.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL)
  8-hour Time Weighted Average

Copper fume	0.1 mg/m^3
Copper dusts and mists	1 mg/m^3

Drinking Water:

- USEPA Maximum Contaminant Level (MCL) 1.3 mg/L
- USEPA Secondary Maximum Contaminant Level (SMCL) 1.0 mg/L

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute 9.2 ug/L
  - Chronic 6.5 ug/L
  - at hardness of 50 mg/L as $CaCO_3$
- Ambient Water Quality Criteria, Marine:
  - Acute 2.9 ug/L
  - Chronic not available

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Subchronic	Chronic
Oral	1.3 mg/L	1.3 mg/L

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry. (ATSDR).1992. *Toxicological Profile for Copper*. U.S. Department of Health and Human Services.

Integrated Risk Information System (IRIS). 1992. On-line database.

U.S. Environmental Protection Agency 1984. *Ambient Water Quality Criteria for Copper*, Office of Water Regulations and Standards, EPA 440/5-84-031, January 1985.

U.S. Environmental Protection Agency. April, 1992. *Drinking Water Regulations and Health Advisories*. Office of Water.

CYANIDE
CAS No. 57-12-5

A. Potential Sources and Exposure

At low levels, cyanides occur naturally in the fruits, roots and leaves of numerous plants. Cyanides are used or produced in various occupational settings where activities include but are not limited to electroplating, metallurgy, metal cleaning, tanning, blacksmithing, photography, fire fighting, and photoengraving. Automobile exhaust is the major source of cyanide released into the air. Exposure occurs primarily through inhalation and less frequently by skin absorption. Cigarette smokers and nonsmokers who inhale secondary smoke are potentially exposed to higher levels of cyanide than the general population.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	27.04 g/mol
pKa	9.21
Water Solubility	miscible
Boiling Point	25.7°C
Vapor Pressure	807.23 mm Hg (27.22°C)

C. Toxicity

Low levels of exposure can produce symptoms such as headache, vertigo, nausea and giddiness (Windholz, 1983; Rumack and Peterson, 1980). More serious effects of cyanide poisoning include convulsions, paralysis, and coma. Death is usually the result of respiratory arrest (Smith, 1980).

Hydrogen cyanide and its simple salts, such as sodium cyanide, are highly toxic by all routes of exposure. Chronic exposure to low levels of cyanide salts has been reported to cause enlargement of the thyroid gland in humans, apparently due to inefficient elimination of the cyanide metabolite thiocyanate. Human data are insufficient to derive an RfD because effective dose levels of chronically ingested uncomplexed cyanide are not documented. The RfD for an oral exposure to free cyanide of 0.02 mg/kg-day is based on observation of weight loss, thyroid effects, and nerve degeneration in a rat chronic dietary study (IRIS, 1992). In this study (Howard and Hanzel, 1955), rats were administered food fumigated with hydrogen cyanide.

There are very little data available for complex cyanides such as ferri and ferro-cyanide compounds. In general, the toxicity of these chemical complexes is expected to be lower.

The literature appears to indicate that the toxicity of cyanide complexes is related to the degree to which they disassociate to form free cyanide.

With regard to the chronic and subchronic effects of complexed cyanides, an RfD of 3.8 mg/kg-day for potassium cyanide, based on cyanide content, has been established by USEPA. Studies by Philbrick et al. (1979) showed decreased weight gain and thyroxin levels and myelin degeneration in rats at 30 mg/kg/day. CN.

D. Toxicokinetics

Cyanide is rapidly absorbed by all routes of administration, including ingestion, inhalation, and dermal contact (Rumack and Peterson, 1980; USEPA, 1980). Although there appears to be no information available on the rate of cyanide absorption in vivo, the very rapid appearance of cyanide toxicity is consistent with rapid absorption (Rumack and Peterson, 1980; USEPA, 1980). Cyanide is rapidly detoxified by enzymatic conversion to thiocyanate (SCN), which is then excreted in the urine (Williams, 1959; Rumack and Peterson, 1980; USEPA, 1980). Because of the rapid detoxification and elimination of cyanide, the effects of a given dose of cyanide will be strongly influenced by the period of time over which the dose is administered. The accumulation of a toxic dose will appear when the rate of dosing exceeds the rate of detoxification plus excretion (Scofield et. al., 1985). There is no evidence that cyanide accumulates in mammals.

E. Ecological Effects

The fate and transport of cyanide in the environment is dependent on the cyanide compound. Most free cyanide will be HCN in aquatic environments. From surface waters, HCN will probably evaporate, although biodegradation is a possible fate process. Metal cyanides are generally insoluble and for that reason may accumulate in sediments. In general, the fate of cyanides in soil has been studied inadequately. By drawing an analogy from its expected fate in water, it can be predicted that the fate of cyanides depends on the pH of the soil. In acid soils, the loss of hydrogen cyanide through volatilization may be the dominant mechanism for loss from surface soils. In subsurface soils, hydrogen cyanide may undergo some microbial degradation, but some may leach through the soil given its low sorption characteristics and high water solubility. Sorption occurs but is not considered an important transport or fate process. In the absence of such destabilizing factors in water as high temperature and extreme pH conditions, complex metal cyanides are expected to have long lifetimes and may undergo substantial transport in aquatic media.

Cyanide at former manufactured-gas plant sites is thought to exist largely in the form of the iron-cyanide complexes ferrocyanide (hexacyanoferrate II, [Fe(CN)6]-4) and ferricyanide (hexacyanoferrate III, [Fe(CN)6]-3). These and other cyanide complexes are measured in the determination of "total cyanide" (NRCC, 1982; APHA-AWWA-WPCF, 1981). As discussed below, the iron-cyanide complexes exhibit very low toxicity. Thus, determination of "free cyanide," "dissociable cyanide" or "cyanide amenable to chlorination," provides the best

indication of any cyanide hazard that may be present (APHA-AWWA-WPCF, 1981; USEPA, 1985a).

The ferro- and ferri-cyanide complexes are extremely stable, normally releasing negligible amounts of cyanide ion (CN) (NRCC 1982). However, in the presence of visible or UV light, iron-cyanide complexes can release cyanide ions (NRCC, 1982, APHA, AWWA-WPCF, 1981). In deep, turbid, or shaded waters the rate of cyanide release can be assumed to be negligible (Broderius and Smith, 1980). Burdick and Lipschuetz (1948), however, showed that non-toxic levels of ferro- and ferri-cyanide could release sufficient amounts of cyanide ion on bright, sunny days to cause toxic effects in fish. Thus, local factors such as depth and turbidity of water and light levels must be considered when evaluating the significance of measured levels of iron-cyanide complexes in water.

Iron cyanide complexes themselves are considered to be "essentially nontoxic" to aquatic organisms (NCRR, 1982).

Data on the toxicity of free cyanide are available for a wide variety of fresh water species. The acute sensitivities ranged from 44.73 ug/L to 2,490 ug/L, but all of the species with acute sensitivities above 400 ug/L were invertebrates. A long term survival test, and a partial and life-cycle test with fish yielded chronic values of 13.57, 7.849, and 16.39 ug/L respectively. Chronic values for two fresh water invertebrates were 18.33 and 34.06 ug/L. Fresh water plants were affected at 30 ug/L to 26,000 ug/L.

Acute toxicities for salt water species ranged from 4.893 ug/L to > 10,000 ug/L. Long term survival tests with a sheepshead minnow gave a chronic value of 36.12 ug/L. Long-term survival in a mypid life-cycle test resulted in a chronic value of 69.71 ug/L. Tests with red algae showed cyanide toxicity at 11 to 25 ug/L, but other species were affected at concentrations up to 3,000 ug/L.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL) — 5 mg/m^3

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute — 5.2 ug/L
  - Chronic — 22 ug/L

- Ambient Water Quality Criteria, Marine:
  - Acute — 1 ug/L

Drinking Water:

- Drinking Water Health Advisories:

lifetime	0.154 mg/L	

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Subchronic	Chronic
Oral	0.02	0.02

State guidance may differ from federal guidance and should be consulted.

H. References

American Public Health Association, American Water Works Association, Water Pollution Control Federation (APHA-AWWA-WPCF). 1981. Cyanides in Standard Methods for the Examination of Water and Waste Water. 15th Edition. Washington, D.C.

Agency of Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Cyanide. U.S. Department of Health and Human Services. Public Health Service.

Broderius, S.J. and L.L. Smith. 1980. Direct photolysis of hexacyanoferrate complexes, Proposed application to the aquatic environment. EPA-600/3-80-003. Duluth, MN.

Burdick, G.E. and M. Lipschuetz. 1948. Toxicity of ferro- and ferri-cyanide solutions to fish, and determination of the causes of mortality. *Trans. Amer. Fish Soc.* 78:192-202.

Howard, J.W. and R.F. Hanzal. 1955. Chronic toxicity for rats of food treated with hydrogen cyanide. *Agric. Food Chem.* 3:325-329.

Integrated Risk Information System (IRIS), on-line database, accessed 1/92.

National Research Council Canada (NRCC). 1982. NRCC Associate Committee on Scientific Criteria for Environmental Quality. The effects of cyanide on aquatic organisms with emphasis upon fresh water fishes. Ottawa, Canada: NRCC/CNRC.

Philbrick, D.J., J.B. Hopkins, D.C. Hill, J.C. Alexander and R.G. Thomson, 1979. Effects of prolonged cyanide and thiocyanate feeding in rats. *J. Toxicol. Environ. Health.* 5:579-592.

Rumack, B.H. and R.G.Peterson. 1980. Clinical toxicology. In: Casarett and Doull's Toxicology, The basic science of poisons. Editors, J. Doull, C.D. Klaassen and M.O. Amdur. Second Edition. p. 677-698. Macmillan Publishing Co., Inc. NY.

Scofield, R., D.N. Layton, G. DeNike, K. Heckman and D. Hsieh. 1985. Criteria and recommendations for standards for cyanide in military drinking water supplies. UCRL-53481 (Draft).

U.S. Environmental Protection Agency (USEPA). 1980. Office of Water Regulations and Standards, Ambient Water Quality Criteria for Cyanides. PB 81-117483. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1984. Health Effects Document for Cyanide. Draft Report. EPA ECAO-CIN-H011. Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. Environmental Protection Agency (USEPA). 1985a. Office of Research and Development, Environmental Research Laboratories, Ambient Aquatic Life Water Quality Criteria for Cyanide. PB 85-227460. Duluth, MN and Narragansett, RI.

LEAD
CAS No. 7439-92-1

A. Potential Sources and Exposure

For the general population, exposure to lead(Pb) occurs by eating foods that contain lead, inhalation of outdoor/household dust, incidental ingestion of soil and lead paint, and through the consumption of lead in drinking water. Through atmospheric deposition, lead enters the environment. Lead can be translocated from the soil into plants. Lead may enter prepared foods when food is prepared in improper glazed pottery and ceramic dishes. Drinking water from acidic water supplies may contain lead which enters through the distribution system (lead pipes, solder and brass faucets). Household dust may contain lead which is attributed to the weathering of lead-based paints. Children, especially those of preschool age, may be exposed to lead due to their hand-to-mouth behavior. Preschool children typically swallow non-food items such as paint chips and dirt which may contain lead.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	207.2 g/mol
Vapor Pressure	1.77 mm Hg at 1000°C

C. Toxicity

Toxic effects resulting from chronic lead exposure are well documented and many have been associated with accompanying blood-lead (PbB) levels. Children have been found to develop symptoms at lower PbB levels than do adults. The most serious effects associated with lead intoxication are the neurotoxic effects. Lead encephalopathy can result from blood lead levels greater than 100 ug/100 ml and is characterized by irritability, loss of memory and ability to concentrate, delirium, hallucinations, cerebral edema, and coma (USEPA, 1984). Less severe neurotoxic effects have been observed at lower blood lead levels. For example, lowered nerve conduction velocities, indicative of peripheral nerve dysfunction, have been noted in adults at blood levels of 30 to 40 ug/100 ml (USEPA, 1986).

Hematologic effects appear to be among the most sensitive indicators of lead absorption. Lead interference with heme synthesis has been noted in humans and other mammalian species at levels below 10-15 ug/100 ml. Lead can also lead to the accumulation of porphyrin in erythrocytes with elevated levels of erythrocyte protoporphyrin (EP) associated with blood lead levels of 25-30 ug/100 ml in adults and 15 ug/100 ml in children (U.S. EPA, 1986). Anemia is characteristic of more severe cases of lead poisoning, resulting from erythrocyte destruction and reduced hemoglobin synthesis (USEPA, 1987).

Renal toxicity has also been observed in victims of lead intoxication. Reversible proximal tubule damage has been observed primarily in cases of short-term exposure with reduced glomerular function associated with more chronic exposures (US EPA, 1984). The gastrointestinal system is one of the earliest to show symptoms of lead intoxication with colic (acute abdominal pain) considered a consistent early symptom of lead poisoning. Recently, environmental epidemiological studies have produced results suggestive of possible lead-induced reproductive effects.

USEPA classifies inorganic lead as a category B2, probable human carcinogen. There is inadequate evidence of carcinogenicity based on human studies, but several animal bioassays have shown statistically significant increases in renal tumors following dietary and drinking water exposure to lead acetate or lead subacetate, two soluble lead salts (IRIS, 1992). USEPA has not calculated a cancer slope factor for inorganic lead because of the large uncertainties involved, including the effect of age, health, nutritional status, and body burden (IRIS, 1992).

The USEPA has not established a risk reference dose (RfD) for lead because it appears that some of the observed effects occur at such low doses as to be essentially without a threshold (IRIS, 1992). Because an USEPA derived reference dose is not available, an alternative approach called USEPA's uptake/biokinetic model is used to evaluate the potential for adverse health effects due to lead. The uptake/biokinetic model was originally developed by USEPA to aid in setting the National Ambient Air Quality Standard for lead. This is a validated model that calculates blood lead levels based on estimated exposure doses of lead to children in to various media such as food and water. The USEPA's uptake/biokinetic model estimates blood lead levels from lead exposures. Once blood lead levels are estimated, adverse effects can be predicted. To determine an estimation of the health risk due to exposure to lead at the site of interest, a threshold based on blood lead has been defined, at 10-15 ug/dl (CDC, 1991).

D. Toxicokinetics

Lead can be absorbed through the gastrointestinal tract, the lungs, and the skin. Absorption of lead within the respiratory tract depends on the solubility of the inhaled particles, particle size and ventilation rate of the individual. The rate of deposition of particulate airborne lead in adult humans is approximately 30%-50%. Once deposited in the lower respiratory tract, any form of lead is completely absorbed (USEPA, 1984). The gastrointestinal absorption of lead in young children is considerably greater than in adults, with about 50 percent of dietary lead absorbed. Numerous factors, including diet and the chemical nature of the lead, influence absorption of lead from the gastrointestinal tract.

Upon entering the body, most lead compounds dissociate; in these cases, metabolism of lead is not an issue. Inorganic lead ion is not known to be biotransformed (Phase I metabolism), but it does undergo conjugation (Phase II metabolism) before excretion. Conversely, the family of alkyl lead compounds (principally, tetramethyl lead and tetraethyl lead) are metabolized in the liver to form the more toxic tri- and dialkyl metabolites (USEPA, 1984).

In the body, about 94% of the adult body burden of lead is localized in the skeleton, about 4% is in the blood, and 2% is in soft tissue. In children, only about 73% of lead in the body is in the bone. Lead is known to cross the placenta, and thus concern exists over distribution during pregnancy and possible toxic effects on the fetus (USEPA, 1984).

The two primary routes of excretion of lead from the body appear to be via the urine and the feces (USEPA, 1984).

E. Ecological Effects

The effects of metals in soils are very much dependent upon the availability of the metal from the soil matrix. Lead seems to be tightly bound by most soils, and substantial amounts must accumulate before it affects the growth of higher plants (Eisler, 1988). Plants readily accumulate lead in soils with low pH or low organic content. Lead has very high residence time in forest litter. Estimates range from 220 years to 500 years (as summarized in Eisler, 1988). Lead toxicosis has been observed in plants from lead concentrations ranging from 0.005 to 33,000 mg/L. Effects include growth stimulation (at low levels), growth inhibition, leaf yellowing, abscission, inhibition of mitosis and chlorophyll synthesis, loss of turgor pressure and death.

Eisler (1988) reviewed the potential effects of lead contamination to wildlife for the US Fish and Wildlife Service. Lead toxicity in water fowl through the ingestion of lead pellets is well documented. Several accidental lead poisoning cases have been reported in livestock. Cattle and horses in the vicinity of a lead smelter died due to lead exposure. A sharp decrease in total milk yield and a significant increase in stillbirths and abortions were reported in dairy cattle that ingested lead-contaminated hay. Eisler also notes that there is no evidence for biomagnification of lead in the food chain of vegetation, to cattle, to the dung beetle, nor is there convincing evidence that any terrestrial vegetation is important in food chain biomagnification of lead.

At a water hardness of 50 mg/L, the acute sensitivies of ten freshwater species range from 142.5 ug/L for an amphipod to 235,000 ug/L for a midge (USEPA, 1984). The lowest and highest available chronic values (12.26 and 128.1 ug/L) are both for a cladoceran. Freshwater algae are affected by concentrations of lead above 500 ug/L, based on data for four species. Acute values are available for 13 marine fauna and range from 315 ug/L for the mummichog to 27,000 ug/L for the soft-shell clam. A chronic toxicity test was conducted with a mysid; unacceptable effects were observed at 37 ug/L. USEPA believes that a measurement such as "acid soluble" would provide a more scientifically correct basis upon which to establish criteria for lead, but USEPA does not yet have an approved method for this analysis (USEPA, 1984).

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

• OSHA Permissible Exposure Limit(PEL) 8 hour Time Weighted Average	50 ug/m^3
Action Level	130 ug/m^3
• National Ambient Air Quality Standard	1.5 ug/m^3
• ACGIH Threshold Limit Value (TLV) for inorganic lead	0.15 mg/m^3
lead chromate	0.05 mg/m^3
lead arsenate	0.15 mg/m^3
• NIOSH Recommended Exposure Level(REL)	<0.1 mg/m^3

Drinking Water:

• USEPA action level	0.015 mg/L
• USEPA Maximum Contaminant Level Goal (MCLG)	0 mg/L

Water:

• Ambient Water Quality Criteria, Freshwater:	
Acute LEC (1-hr average)	82 ug/L
Chronic LEC (4-hr average)	3.2 ug/L
Ambient Water Quality Criteria, Marine:	
Acute LEC (1 hr-average)	140 ug/L
Chronic LEC (4-hr average)	5.6 ug/L

Food:

• FDA action levels in leaching solutions:	
for pottery flatware	7 ug/mL
for small hollowware	5 ug/mL
for large hollowware	2.5 ug/mL
for silver-plated hollowware	7 ug/mL
for silver-plated hollowware	0.5 ug/mL

G. Toxicity Factors

Reference Doses (mg/kg-day):

for alkyl lead

	Subchronic	Chronic
oral	1×10^{-6}	1×10^{-7}

Various state guidelines may differ from federal regulations and should be consulted.

H. References

Agency of Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Lead. U.S. Department of Health and Human Services. Public Health Service.

ACGIH, 1990-91. Threshold Limit Values and Biological Exposure Indices for 1990-1991. American Conference of Governmental Industrial Hygienists,Cincinnati, OH

Eisler, R., 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Biological Report 85(1.14). Contaminant Hazard Reviews Report No. 14., US Fish and Wildlife Service Patuxent Wildlife Research Center, Laurel, MD.

Integrated Risk Information System (IRIS), on-line database, accessed 1/92.

U.S. Environmental Protection Agency (USEPA). 1984. Health Effects Assessment for Lead. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/055. NTIS PB85-16996/AS.

U.S. Environmental Protection Agency (USEPA). 1984a. Ambient Water Quality Criteria for Lead. Office of Water.

U.S. Environmental Protection Agency (USEPA). 1986. Air Quality Criteria Document for Lead. Volumes III, IV. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028d.

U.S. Environmental Protection Agency (USEPA). 1987. Preliminary review of the carcinogenic potential of lead associated with oral exposure. Prepared by the Office of Health and Environmental Assessment, Carcinogenic Assessment Group, Washington, DC, for the Office of Drinking Water, Office of Solid Waste and the Office of Emergency and Remedial Response (Superfund). OHEA-C-267. Internal Review Draft.

MANGANESE
CAS No. 7439-96-5

A. Potential Sources and Exposure

Manganese is a naturally-occurring metal found in many types of rock. It does not usually exist in the environment as a pure metal, but is found combined with sulfur, oxygen, and chlorine. Manganese is a component of some ceramics, pesticides, fertilizers, and nutritional supplements. It is an essential element, small amounts are required for human health.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	54.94 g/mol
Water Solubility	decomposes at 25°C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

C. Toxicity

The systemic toxicity of manganese is low, due in part to its toxicokinetics, although large oral doses of manganese salts causes gastrointestinal irritation. Target organs are the lung and CNS. Industrial toxicity from inhalation exposure, generally to manganese dioxide in mining or manufacturing is of two types: manganese pneumonitis as a result of acute exposure, and manganese poisoning (manganism) as a result of long-term (greater than two years) exposure. Manganism is a neuropsychiatric disorder that is evidenced by mental and emotional disturbances, and slow and clumsy body movements. There is no evidence to support the carcinogenicity of manganese (Klaassen, 1995).

D. Toxicokinetics

Gastrointestinal absorption of manganese is less than five percent (Klaassen, 1995). Manganese is widely distributed throughout the body and concentrates in the mitochondria of cells. It readily crosses the blood-brain barrier, and its half-life in the brain is longer than in the rest of the body. Manganese is eliminated in the bile and is resorbed in the intestine, but is excreted in the feces.

E. Ecological Effects

Bioaccumulation
Manganese is found in high concentrations in marine and freshwater plants and in low concentrations in both invertebrates and fish.

Toxic Effects to Aquatic Organisms
Manganese has been shown to be only slightly to moderately toxic to most aquatic plant species. For the majority of invertebrates, manganese is moderately toxic typically with a lethal concentration of 300 mg/L (for Mn^{2+}). However, it has also been shown that Mn^{7+} can be much more toxic with a lethal concentration < 1mg/L. Manganese (Mn^{2+}) is not acutely toxic in fish but has caused chronic effects at a wide range of concentrations. Manganese, in general, demonstrates a protective mechanism against more toxic heavy metals. By saturating metal-binding sites, the moderate effects of manganese guard against harsher effects of other metals. For this reason, governments have tended to not impose a guideline concentration level.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	
Mn fumes, as Mn	1.0 mg/m^3
ACGIH Threshold Limit Value (TLV)	
Mn dust and compounds	5.0 mg/m^3

Drinking Water:

USEPA Secondary MCL for aesthetics	0.05 mg/L

G. Toxicity Factors

Non-carcinogen Toxicity Factors

Oral Reference Dose (RfD)	1.4×10^{-1} mg/kg-day (IRIS, 1995)
Inhalation RfC	5×10^{-5} mg/m^3 (IRIS, 1993)

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological profile for manganese. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

NICKEL
CAS No. 744-02-0

A. Potential Sources and Exposure

Nickel is a naturally-occurring metal that is mined and is combined with other metals to form alloys. Nickel is emitted into the air through fossil fuel combustion, incinerators, chemical and cement manufacturing, coke ovens, and nickel recovery operations. Evidence has accumulated indicating that nickel may be a trace metal essential for human health.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	58.69 g/mol
Water Solubility	insoluble at 25°C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

C. Toxicity

The target organs of nickel toxicity are skin and lungs. Allergic contact dermatitis to nickel-containing metals is common in the general public. The major adverse effects seen as a result of high exposure levels to nickel, likely found only in the workplace, include dermatitis, chemical pneumonitis, and lung and nasal cancers. Nickel carbonyl is extremely toxic, resulting in chest pain, dry coughing, cyanosis, gastrointestinal symptoms, sweating, visual impairment, and weakness. This is often followed by pulmonary hemorrhage and edema. Survivors may be left with pulmonary fibrosis.

The USEPA classifies nickel as a Group A - Human Carcinogen based on epidemiological studies in which a causal association exists between exposure to nickel refinery dust and lung and nasal tumors.

D. Toxicokinetics

Nickel is poorly absorbed from the gastrointestinal tract. Absorption from the respiratory tract is dependent on the solubility of the nickel compounds, with higher urinary nickel observed in workers exposed to soluble nickel compounds (Ni chloride, Ni sulfate) than those

exposed to insoluble nickel compounds (Ni oxide, Ni subsulfide). Nickel applied directly to the skin can be absorbed into the skin where it may remain rather than entering the systemic circulation. Following inhalation exposure, nickel tends to accumulate in the lungs. Nickel can cross the placenta and it can accumulate in breastmilk. Regardless of the exposure route, absorbed nickel is excreted in the urine.

E. Ecological Effects

Bioaccumulation

Nickel concentrations in plants are generally low, < 150 mg/kg dry weight, but occasional reports will show much higher concentrations of 150-700 mg/kg. Likewise, invertebrate concentrations are low, usually < 5 mg/kg. Nickel cannot be considered a significant, widespread contaminant except at certain site-specific points. Uptake in invertebrates occurred principally through the water and ingested particulate nickel was excreted. In fish, concentrations again are generally low, < 0.5 mg/kg wet weight, but instances of higher concentrations do exist near polluted areas (1-2 mg/kg wet weight).

Toxic Effects to Aquatic Organisms

Nickel (Ni^{2+}) is considered moderately to highly toxic to most aquatic plant species. To invertebrates, Ni^{2+} is one of the least toxic inorganic agents. To both marine and freshwater fish, Ni^{2+} is relatively nontoxic but when exposed to low levels over extended periods effects include reduced skeletal calcification and reduced diffusion capacity of gills. Both acute and chronic toxicity of Ni^{2+} is strongly related to water hardness.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	
Ni metal and insoluble compounds	1 mg/m^3
Ni and soluble compounds	0.1 mg/m^3
ACGIH Threshold Limit Value (TLV)	
Ni metal and insoluble compounds	1 mg/m^3
Ni and soluble compounds	0.1 mg/m^3

Drinking Water:

USEPA Maximum Contaminant Level(MCL)	0.1 mg/L

Water:

Ambient Water Quality Criteria:	
Ingesting water and organisms	0.61 mg/L
Ingesting organisms only	4.6 mg/L

G. Toxicity Factors

Non-carcinogenic Factors

Oral (RfD) (nickel, soluble compounds)	0.02 mg/kg-day (IRIS, 1995)
Inhalation (RfC)	No data

Carcinogenic Factors

Inhalation Unit Risk (Ni refinery dust & Ni subsulfide)	2.4×10^{-4} per (ug/m^3) (IRIS, 1995)

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for nickel. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

THALLIUM
CAS No. 7440-28-0

A. Potential Sources and Exposure

Thallium is a naturally-occurring metal that is used in the manufacture of electronic devices, low temperature thermometers, semiconductors, some optical lenses, and is present in many alloys. It has been used medically as a depilatory agent. Thallium has been used as a rat poison and insecticide until it was banned in 1972. Most exposures occur via ingestion or inhalation of thallium-contaminated soils.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	204.38 g/mol
Water Solubility	insoluble at 20°C
K_{oc}	no data
log K_{ow}	no data
Henry's Law Constant	no data

C. Toxicity

Thallium is one of the most toxic of the metals. Acute toxicity results in gastrointestinal irritation, shock, ascending paralysis, seizures, and psychic disturbances. Thallium compounds can affect the respiratory, cardiovascular, gastrointestinal systems, the liver, kidneys and the male reproductive system. It appears as though thallium can compete with potassium as well as with sulfhydral groups on proteins (Klaassen, 1995). Alopecia, nail changes, peripheral neuropathy, and kidney damage are signs of chronic thallium poisoning. Loss of vision and hearing impairment have been related to industrial thallium exposures. There are no data available on the carcinogenic effects of thallium exposures, although there are some data to suggest some thallium salts may be genotoxic.

D. Toxicokinetics

Thallium is efficiently absorbed from the gastrointestinal tract and to a lesser extent from the skin. Data from intratracheal administration studies in animals suggest that uptake through the respiratory epithelium was rapid and complete. Following absorption, distribution of thallium occurs to the greatest extent to the kidney, followed by lesser amounts to the heart, bone, brain, skin, and other organs. Thallium accumulates in tissues where there is a high concentration of potassium as it appears to substitute for potassium in many enzymes

requiring potassium (Klaassen, 1995). Excretion of thallium occurs via urinary and fecal routes (ATSDR, 1991).

E. Ecological Effects

Bioaccumulation
Little is known about the accumulation of total thallium in the aquatic food chain.

Toxic Effects to Aquatic Organisms
For plants, Tl^{+} is generally more toxic than Tl^{3+}. Thallium compounds are only moderately toxic to most aquatic invertebrate and fish species. Little data are available regarding the effects of low levels of thallium from chronic exposure.

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

OSHA Permissible Exposure Limit (PEL)	0.1 mg/m^3
ACGIH Threshold Limit Value (TLV)	0.1 mg/m^3

Drinking Water:

USEPA Maximum Contaminant Level	not available

Water

Ambient Water Quality Criteria:	
Ingesting water and organisms	13 ug/L
Ingesting organisms only	48 ug/L

G. Toxicity Factors

Reference Doses(mg/kg-dy):

Oral	8×10^{-5}

State guidance may differ from federal guidance and should be consulted.

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological profile for thallium. U.S. Public Health Service.

Integrated Risk Information System (IRIS) on-line database.

Klaassen, Curtis, D., Mary, Amdur, John Doull, 1995. Toxicology: The Basic Science of Poisons, 5th edition; McGraw-Hill, New York.

Moore, James W. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities*, Springer-Verlag, New York, 1991.

ZINC
CAS No. 7440-66-6

A. Potential Exposure

Zinc occurs in nature in the 0 and +2 valence states, although it is also found in four other stable valences. Metallic zinc is insoluble in water, although some zinc salts are soluble and found naturally in drinking water. Exposure to zinc in very low concentrations occurs daily through the diet. Average zinc intake through the diet ranges from 7 to 16.3 mg/day. Zinc is an essential trace element. Zinc is used in the manufacture of galvanized iron, bronze, white paint, rubber, glazes, enamel, glass, paper and as a wood preservative. Exposure to zinc at higher levels can occur from drinking water or other liquids stored in galvanized metal containers.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	65.4 mg/l

C. Toxicity

Ingestion of excessive amounts of zinc above the recommended daily allowance for zinc of 15 mg may cause fever and gastrointestinal distress. Following acute, intermediate, or chronic ingestion of zinc, the primary effects in humans are pancreatic abnormalities, and gastrointestinal irritation. Ingestion of zinc has resulted in the reduction of HDL-cholesterol levels in humans. Oral exposure has been reported to impair immune and inflammatory responses. Anemia may occur after high level acute, intermediate, or chronic oral exposure to zinc.

Inhalation exposure to zinc dust or fumes has been associated with pulmonary fibrosis and metal fumer fever. Acute high level exposure to zinc oxide causes metal fume fever. Zinc oxide penetrates the alveoli, damages the lung tissue, and transiently impairs respiratory function. Metal fume fever is believed to be the result of an immune reaction to inhaled oxide particles. Chronic exposure to zinc has produced anemia. Zinc needs to be present at certain levels to predict fetal/developmental abnormalities or effects.

There is no evidence to indicate zinc and its compounds are associated with carcinogenicity in humans (IRIS, 1992).

D. Pharmacokinetics

It appears that zinc is absorbed via ingestion and inhalation. Zinc is widely distributed throughout the body, and is found in high concentrations in male reproductive organs, pancreatic isle
ts, muscle, kidney, liver, and bone. Excretion of zinc is m ainly through the gastrointestinal tract, though some of the zinc is reabsorbed. it is also excreted via urine, sweat, hair, and mild. Placental transfer of zinc may also occur. The half-life of zinc in humans is 200 to 400 days (ATSDR, 1992).

E. Environmental Effects

Zinc is an essential micronutrient for all living organisms. Because zinc is essential, zinc is bioaccumulated by all organisms. The toxicity of zinc is dependent upon its chemical form and degree of interconversion among the various forms. Zinc will not be sorbed or bound unless it is dissolved, but bound zinc will dissolve in the digestive tract following the ingestion of particulates. The toxicity of undissolved zinc to a particular species depends on the feeding habits. Aquatic plants and most fish are relatively unaffected by suspended zinc in the water column. Both terrestrial and aquatic invertebrates and filter feeder fish might be adversely affected by ingestion of sufficient quantities of particulates containing zinc. The acute toxicity of zinc to aquatic animals is influenced by several parameters including increasing hardness, abundant dissolved oxygen and low temperatures which lower the potential toxicity of zinc.

Reported acute toxicity testing for freshwater organisms indicates that insects are most resistant whereas cladocerans and the striped bass are the most sensitive to zinc. The reported mean genus acute value for cladoceran is 50.56 ug/l at a hardness of 50 mg/l. The final acute value representing zinc toxicity to freshwater species is 108.4 ug/l at a hardness of 50 mg/l.

The range of species mean acute values for saltwater invertebrates extends from 166 ug/l for embryos of the quahog clams, Mercenaria mercenaria, to 320,400 ug/l for adults of the clam, Macoma balthica. In general, early life stages of saltwater invertebrates and fish are more sensitive to zinc than juveniles and adults. The salt water final acute value for zinc is 174.5 ug/l which is higher than the acute value of 166 ug/l for the quahog clam. Chronic toxicity values range from 47 to 852 ug/l and appear to be relatively unaffected by hardness.

Zinc was found to accumulate in freshwater animal tissues from 51 to 1,130 times the concentration present in the water (USEPA, 1980). Steady-state zinc bioconcentration factors for 12 aquatic species range form approximately 4 to 24,000 (USEPA, 1980).

Zinc bioconcentration from soil by terrestrial plants, invertebrates, and mammals, values of 0.4, 8 and 0.6 have been reported by Fishbein (1981). Davis et al. (1978) reported phytotoxic tissue zinc levels ranging from 200 to 400 ppm. Studies have reported that 60 to 81 ppm of zinc in wheat and corn tissue is phytoxic (Collins, 1981; Monenco, 1984).

The tolerance of domestic livestock to zinc in animal feed ranges form 300 to 1000 ppm (NAS, 1980). Zinc poisoning has occured in cattle. In one outbreak, poisoning was caused by food accidentally contminated with zinc at a concentration of 20g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited server enteritis, and some died or had to be slaughtered. Some researchers have speculated that exposure to excessive amounts of zinc may constitute a hazard to horses. Findings in foals living near lead-zinc smelters suggest that excessive exposure to zinc may produce bone changes, joint afflictions, and lameness. In swine given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints.

F. Federal Regulations, Standards, and Guidelines

Air:

- OSHA Permissible Exposure Limit (PEL)
  - for zinc chloride 1 mg/m³
  - for zinc oxide 5 mg/m³

- ACGIH Threshold Limit Value-Time Weighted Average
  - for zinc chloride 1 mg/m³
  - STEL 2 mg/m³

- ACGIH Threshold Limit Value-Time Weighted Average
  - for zinc oxide fumes 5 mg/m³

Drinking Water:

- Drinking Water Health Advisories:
  - Drinking Water Equilvalent Level (DWEL) 10.5 mg/l
  - Lifetime Health Advisory 2.1 mg/l

- USEPA Secondary Maximum Contaminant Level 5 mg/l

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute 120 ug/l (1 hr average)
  - Chronic 110 ug/l (4 hr average)

- Ambient Water Quality Criteria, Marine:
  - Acute 95 ug/l (1 hr average)
  - Chronic 86 ug/l (4 hr average)

G. Toxicity Factors

Reference Doses (mg/kg/day):

	Subchronic	Chronic
Oral	0.2	0.2

H. References

Integrated Risk Information Service (IRIS). On-line data base.

National Academy of Sciences (NAS). 1980. Drinking water and health. Volume 3. Safe Drinking Water Committee, National Research Council, National Academy of Sciences. National Academy Press. Washington, D.C.

USEPA (Environmental Criteria and Assessment Office). 1980. Ambient Water Quality Criteria for Zinc. Cincinnati, OH. NTIS PB 81-117897.

USEPA (Environmental Criteria and Assessment Office). 1984. Health Effects Assessment for Zinc. Cincinnati, OH. Final Draft.

Vanadium
Cas No. 7440-62-2

A. Potential Exposure

Vanadium is a ubiquitous metal found in rocks, minerals, and soils. It co-occurs with uranium in uranium ores and is also found in coal and in crude oils. Vanadium is a transition metal with possible valence states ranging from -3 to +5. Of these, the +3, +4, and +5 valence states can exist in natural aquatic systems such as groundwater or surface water. Ionic forms of the +3, +4, and +5 valence states may be present in solution under environmental conditions as oxygen or hydroxide anions . Vanadate and vanadyl ions are most likely to be found under environmental conditions, based on the pH of the water and its oxidation-reduction potential (Eh). Vanadium pentoxide (V_2O_5) and vanadyl sulfate ($VOSO_4$? $3H_2O$) are the forms of vanadium found in oil-fired fly ash (Henry and Knapp, 1980). Humans may be exposed to vanadium from soils and groundwater.

B. Toxicity

Vanadium is a trace element that is considered an essential in chicks and rats, although it has not been established whether it is an essential element for humans. Its deficiency (in chicks and rats) can cause reduced growth, impairment of reproduction and disturbances in lipid metabolism. There are no known vanadium deficiency symptoms in humans.

The molecular mechanisms of the toxicity of vanadium have not been well established. It is believed that vanadium salts are not very toxic, although vanadate has been shown to be a potent inhibitor of many key cellular enzymes, including skeletal muscle and heart enzymes.

Several animal studies clearly demonstrate that target organs for soluble vanadium toxicity include the kidney and nervous system, and the respiratory tract for inhaled vanadium metal. Vanadate may alter blood flow and the removal of metabolic waste in the kidney (Dafnis and Sabatini, 1994) and impair lipid metabolism (Sasi et al., 1994).

Hematological (blood-related) effects of vanadium have been studied by several researchers, however, no agreement has been reached as to the significance of the results. Administration of ammonium metavanadate to male and female Wistar rats in drinking water at a dose of 1.5 or 5-6 mg/kg/day for four weeks resulted in hematological effects, including a decrease in the white blood cell count, hemoglobin levels (the oxygen carrier in the blood), and hematocrit index at the high dose (Zaporowska, et al., 1993). A twelve-week oral drinking water study in which Wistar rats were treated with 7.6 to 10.2 mg vanadium/kg/day, concluded that there was no significant hematological toxicity demonstrated under the study conditions (Dai et al., 1995).

The developmental toxicity of vanadium has recently been assessed (and reviewed by Domingo,

1994) in several animal systems. The oral administration of vanadyl sulfate pentahydrate (37.5, 75, 150 mg/kg/day) to pregnant mice during development of organs caused toxicity to the mother and developing embryos, and birth defects (cleft palate) at all dose levels tested (Paternain et al., 1990). A significant increase in the number of resorptions and dead fetuses was also observed when sodium metavanadate was administered orally to pregnant rats at 20 mg/kg/day. Vanadate treatment of diabetic pregnant rats was found to be toxic; causing death in 45% of the animals, and markedly reducing the number of live fetuses per pregnancy (Ganguli et al., 1994).

Llobet et al. (1993) evaluated the effect of sodium metavanadate in drinking water for 64 days on male reproduction in mice. Although fertility was not decreased in male mice at 20 and 40 mg/kg/day, it was significantly decreased at 80 mg/kg/day.

There are no studies published relating human cancers to vanadium exposures. Also, there is no evidence that vanadium and vanadium salts are carcinogenic to intact animals. There are some *in vitro* studies that suggest that vanadate may promote transformation in cells (Klarlund et al., 1985). Although vanadium compounds are not clastogenic (causing large alterations in DNA structure), they can be weakly mutagenic in certain systems (Leonard and Gerber, 1994).

C. Toxicokinetics

As with many other trace metals, it is believed that vanadium is poorly absorbed from the gastrointestinal tract, but that vanadium dusts are efficiently absorbed through the lungs. Once absorbed in the tissues, two vanadium species (V^{5+} and V^{4+}) may be present under physiological conditions. In general, pentavalent vanadium is the more toxic form because it enters the cell more readily. However, once vanadium of either form enters cells it is reduced by intracellular glutathione and other agents to the trivalent species (V^{3+}), which binds readily to cellular macromolecules. Vanadate is mainly excreted by the kidneys (Dafnis and Sabatini, 1994). Accumulation of the element occurs primarily in the kidney and to some extent in fat tissue and bone. Its distribution appears to vary according to the route and duration of administration.

D. Environmental Effects

The existing marine toxicity data for vanadium represent four species of phytoplankton, three invertebrates species (a worm, a mollusc, and a crustacean), and one fish species. These data represent acute toxicity only. There are insufficient data available for use in developing marine ambient water quality criteria for vanadium due to the number of species tested and the lack of chronic toxicity data (USEPA, 1985). From these limited data it appears that the marine toxicity of vanadium is low and that it is more toxic to marine phytoplankton than to invertebrates or fish.

The freshwater toxicity data for vanadium include results of acute and chronic tests. The acute

data represent one invertebrate species (a crustacean) and ten fish species. In addition, several studies discuss chronic effects to freshwater phytoplankton and fish.

The freshwater toxicity of many metals, including vanadium depends on the hardness of the water. The toxicity data indicate that freshwater organisms may be slightly more sensitive to vanadium than marine organisms. Unlike other metals, vanadium appears to be more toxic to older life-stages of fish than to fry or juveniles (Hamilton, 1995; Stendahl and Sprague, 1982; and Ernst and Garside, 1987).

Jagadeesh et al. (1989) tested the effects of increasing concentrations of vanadium on two species of catfish (*Clarias batrachus* (Linn.) and *Channa punctatus* (Bloch)). They subjected the fish to 5,000, 10,000, and 15,000 ug/l of vanadium (vanadium compound unspecified) and found decreasing glycogen content in the tissues of both species with increasing vanadium exposure. The effect of these vanadium concentrations on the survival or reproductive ability of the fish is not reported. Chakraborty et al. (1995) investigated the effects of vanadium on enzyme activity in catfish (*Clarias batrachus* (Linn.)) demonstrating a dose-dependent increase in metabolizing enzyme activity. There is no indication, however, as to whether these concentrations of vanadium effected the survival or reproductive ability of the fish.

Savouré (1984) demonstrated that a 1,000 ug/l solution of vanadium in the form NH_4VO_3 (a pentavalent form) effects the metabolism of four freshwater algal species by increasing the soluble sugar content of the cells and nitrate-reductase activity.

With a few notable exceptions, vanadium does not appear to bioconcentrate in aquatic organisms (Moore, 1991). There are no available bioaccumulation factors for vanadium. However, studies exist on vanadium uptake from food and concentrations of vanadium in marine organisms indicating that there is little likelihood for vanadium to bioconcentrate in a marine food chain since the vanadium content of the organisms decreased from a lower trophic level to a higher one (Fowler, 1986). Certain marine invertebrates, in particular ascidians or sea squirts, can bioconcentrate vanadium to elevated levels. *Phallusia mammilata*, a seaworm, can have up to 1,900,000 ug/l vanadium in its blood. A sea squirt, *Ascidia nigra*, can accumulate up to 1.45% vanadium in its blood cells. These organisms are highly acidic (Greenwood and Earnshaw, 1984).

E. References

Chakraborty, A., S. Bhattacharjee, and M. Chatterjee. 1995. Alterations in enzymes in an Indian catfish, *Clarias batrachus* (Linn.), exposed to vanadium. Bull. Environ. Contam. Toxicol. 54: 281-288.

Dafinis E., Sabatini, S. 1994. Biochemistry and pathophysiology of Vanadium. Texas Tech University Health Sciences Center. Nephron 67:133-143.

Dai, S., Vera, E., McNeill, J.H. 1995. Lack of haematological effects of oral Vanadium treatment in rats. Pharmacology & Toxicology, 76:263-268.

Domingo, J. L. 1994. Metal-induced developmental toxicity in mammals: A review. J. Toxicol. and Environ. Health. 42:123-141.

Domingo, J.L., Llobet, J.M. Tomas, J.M. 1985. Short-term toxicity studies of vanadium in rats. Journal of Applied Toxicology, 5(6)418-421.

Ernst, W.R. and E.T Garside. 1987. Lethal effects of vanadium to two life stages of brook trout Salvelinus fontinalis (Mitchill). Can. J. Zool. 65, 628-634. Cited in: Hamilton, 1995.

Fowler, S.W. 1986. Trace metal monitoring of pelagic organisms from the open Mediterranean Sea. Environmental Monitoring and Assessment. 7:59-78.

Ganguli, S., Reuland, D.J., LeRoy, A.F., Deakins, D.D., Johnston, W.J., Pasha, A.. 1994. Effects of maternal vanadate treatment on fetal development. Life Sciences, 55(16)1267-1276.

Greenwood, N.N. and A. Earnshaw. 1984. *Chemistry of the Elements*. Pergamon Press, New York.

Hamilton, S.J. 1995. Hazard assessment of inorganics to three endangered fish in the Green River, Utah. Ecotoxicology and Environmental Safety, 30, 134-142.

Henry, W.M and K.T. Knapp. 1980. Compound forms of fossil fuel fly ash emissions. Environmental Science and Technology. 14:4, 450-456.

Jagadeesh, K.B., S.A. Shaffi, and S. Jeelani. 1989. Vanadium induced changes in glycogen content of *Clarias batrachus* (Linn) and *Channa punctatus* (Bloch). Acta Physiologica Hungarica, 74 (1), 43-48.

Leonard, A. and Gerber, G.B. 1994. Mutagenicity, carcinogenicity and teratogenicity of vanadium compounds. Mutation Res. 317:81-88.

Llobet, J.M., Colomina, M.T., Sirvent, J.J., Domingo, J.L., Corbella, J. 1993. Reproductive toxicity evaluation of vanadium in male mice. toxicology, 80:199-206.

Moore, J.W. 1991. *Inorganic Contaminants in Surface Water, Research and Monitoring Priorities*. Springer-Verlag, New York.

Paternain, J.L., Domingo, J.L., Gomez, M., Ortega, A., and Corbella, J. 1990. Developmental toxicity of vanadium in mice after oral administration. J. Appl. Toxicol. 10:181-186.

Sasi, M.M., Haider, S.S., El-Fakhri, M., Ghwarsha, K.M. 1994. Microchromatographic analysis of lipids, protein, and occurrence of lipid peroxidation in various brain areas of vanadium exposed rats: a possible mechanism of vanadium neurotoxicity. NeuroToxicology 15(2): 413-420.

Savouré, B. 1984. Effets toxiques du vanadium sur le métabolisme de quelques algues d'eau douce cultivées *in vitro*. Hydrobiologia, 118, 147-151.

Stendahl, D.W. and J.B. Sprague. 1982. Effects of water hardness and pH on vanadium lethality to rainbow trout. Water Res. 16, 1479-1488. Cited in: Hamilton, 1995.

USEPA. 1985. Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and their Uses. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. PB85227049.

Zaporowska, H., Wasilewski, W., Stotwinska, M. 1993. Effect of chronic vanadium administration in drinking water to rats. Bio Metals. 6:3-10.

PHENANTHRENE
CAS No. 85-01-8

A. Potential Sources and Exposures

Phenanthrene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the genral profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	178.2 g/mol
Water Solubility	1.00 mg/L at 21°C
Vapor Pressure	6.8×10^{-4} atm at 25°C
K_{oc}	14000 mL/g
log K_{ow}	4.46
Henry's Law Constant	1.59×10^{-4} atm-m^3/mol at 25°C

C. Toxicity

There are no data on the toxicity of phenanthrene to humans (IARC, 1983). Phenanthrene has been tested for carcinogenicity in laboratory animals by the oral, dermal, and subcutaneous routes of administration (as cited in IARC, 1983); however, IARC (1983) and USEPA (IRIS, 1992) concluded that data from available studies were inadequate to permit an evaluation of its carcinogenicity of phenanthrene. In addition, the results of short-term mutagenicity tests are equivocal. Nonetheless, current theories regarding the mechanisms of metabolic activation of PAHs predict that phenanthrene may have carcinogenic potential (Jerina et al. 1978, as cited in IRIS, 1992).

D. Toxicokinetics

In general, many polycyclic aromatic hydrocarbon can produce toxicity after inhalation, oral, or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed function oxidase system and eliminated via the hepatobiliary tract.

Several metabolites of phenanthrene have been identified. They include the 1,2- 3,4- and 9,10-dihydrodiols, and the 1,2-diol-3,4-epoxide. The dihydrodiols displayed little or no

tumor-initiating activity on mouse skin (Wood et. al., 1979 as cited in IARC, 1983). The epoxide was found to be mutagenic in bacterial and mammalian cells (Wood et. al., 1979 as cited in IARC, 1983). Buening et. al. 1979 (as cited in USEPA, 1982) reported significant tumorigenic activity with the expoxide but not with phenanthrene itself in newborn mice.

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects. Acute toxicity of phenanthrene to fish has been reported at levels of 4,500 mg/L and would probably be lower for sensitive species or for chronic effects.

F. Federal Regulations, Standards, Guidelines, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Subchronic	Chronic
Oral	3.0	0.3

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

PYRENE
(Benzo(def)phenanthrene)
CAS No. 129-00-0

A. Potential Sources and Exposure

Pyrene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	202.2 g/mol
Vapor Pressure	2.5×10^{-6} at 25°C
Water Solubility	0.135 mg/L at 25°C
K_{oc}	38000 mL/g
log K_{ow}	4.88
Henry's Law Constant	5.1×10^{-6} atm-m^3/mol at 25°C

C. Toxicity

Pyrene is considered to be a skin irritant in humans (as cited in IRIS, 1992). Pyrene has produced negative results in most mutagenicity assays (USEPA, 1982). IARC (1983) concluded that there is limited evidence that pyrene is active in short-term mutagenicity assays. Pyrene is classified as a Group D carcinogen by the USEPA based on the lack of human carcinogenicity data and inadequate data from animal bioassays.

The RfD for oral exposure to pyrene is 0.03 mg/kg-day, based on the observation of kidney toxicity in mice that received subchronic dosing with pyrene by gavage (USEPA, 1989 as cited in IRIS, 1992). Confidence in the data base is low due to the lack of supporting evidence from other subchronic, chronic or developmental/reproductive studies.

D. Toxicokinetics

Human exposure to pyrene is almost exclusively through ingestion and inhalation although it can be absorbed through the skin. There are no pharmacokinetic data for pyrene in humans (USEPA, 1980). Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty

tissues.

Pyrene, like other PAHs, is apparently metabolized via the microsomal mixed function oxidase system in mammals.
Elimination of pyrene from rats exposed to a pyrene aerosol (500 mg/L, 0.3 - 0.5 mm particles) for 60 minutes was reported (Mitchell and Tu, 1979 as cited in USEPA, 1982) to rapidly occur primarily via the liver and biliary system. When 50 ug of pyrene was administered in a gelatin-saline suspension to two rats by stomach tube, approximately half of the administered pyrene was still present in the gastrointestinal tract after 24 hours (Mitchell and Tu, 1979 as cited in USEPA, 1982).

E. Ecological Effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects. A no effect level of 5 mg/L was observed for trout in an acute (24 hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

F. Federal Regulations, Guidelines, Standards, and Criteria

The reader is requested to review the toxicity profile for PAHs for information regarding federal regulations, standards, guidelines, and criteria.

G. Toxicity Factors

Reference Doses(mg/kg-day):

	Subchronic	Chronic
Oral	3.0	0.3

State guidance may differ from federal guidance and should be consulted.

H. References

For references, see the PAH toxicity profile.

BIS(2-ETHYLHEXYL)PHTHALATE

(DEHP, BEHP, di(2-ethylhexyl)phthalate)
CAS No. 117-81-7

A. Potential Sources and Exposure

Bis(2-ethylhexyl)phthalate (BEHP), a phthalic acid ester and is widely distributed at low levels in the environment. Phthalic acid esters are primarily used as plasticizers for specific plastics such as polyvinyl chloride. BEHP is also used in paper production, perfumes, lubricating oils, and in other industrial and consumer applications (Sittig, 1980). Human exposure occurs most commonly by oral and inhalation routes, although intravenous exposure can occur to patients receiving blood transfusions or dialysis. There are no reports of BEHP-induced health effects among exposed populations.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	391 g/mol
Vapor Pressure	2×10^{-7} mm Hg
Water Solubility	0.285 mg/L at 24°C
log K_{ow}	4.88
Henry's Law Constant	1×10^{-4} atm-m^3/mol

C. Toxicity

No human data on adverse effects associated with BEHP exposure are available. Several studies on the acute toxic effects of BEHP have been conducted in animals. When administered by oral, intraperitoneal, intravenous, and inhalation routes, BEHP has a low acute toxicity. Exposures to high acute levels can result in morphological and biochemical changes in the liver and testes (ATSDR, 1988).

USEPA has derived an oral reference dose (RfD) of 0.02 mg/kg-day for BEHP. This value is based on a one year feeding study of BEHP in guinea pigs. A no observed adverse effect level (NOAEL) was not observed in this study. The lowest observed adverse effect level (LOAEL), 19 mg/kg/day, resulted in an increase in the liver weight. An uncertainty factor of 1000 was applied to the LOAEL to obtain the RfD (IRIS, 1992).

BEHP is classified as a probable human carcinogen, Group B2 because of its ability to induce hepatic peroxisomal enzyme activity (Ganning et al., 1984), which often results in

hepatocarcinogenicity. The oral cancer potency factor for BEHP is 1.4×10^{-2} $(mg/kg/day)^{-1}$. This cancer potency factor is based on a two-year feeding study in both rats and mice where a statistically significant, dose-related increase in hepatocellular carcinomas and neoplastic nodules were observed in both male and female rats.

BEHP has been shown to have adverse effects on fertility and reproduction in both mice and rats. In male mice, the effect is linked to degeneration of seminiferous tubules. BEHP has also been found to be fetotoxic and teratogenic in experimental animals (IRIS, 1992). Studies indicate that BEHP is neither a direct nor indirect acting mutagen.

D. Toxicokinetics

BEHP is well absorbed from the gastrointestinal tract and may also be absorbed through the skin and lungs. Hydrolysis to the corresponding monoester metabolite, MEHP, with release of 2-ethylhexanol, largely occurs prior to intestinal absorption. Once absorbed, wide distribution in the body occurs with the liver being the primary storage site. BEHP is primarily converted to polar derivatives of the monoesters by oxidative metabolism prior to elimination via urinary excretion. Clearance for the body is rapid, and there is only a slight cumulative potential (ATSDR, 1988).

E. Ecological Effects

In a chronic assay with rainbow trout, BEHP gave a chronic value of 8.4 ug/L. No acute-chronic ratio could be calculated because of the absence of a 96 hour LC50 value. In a chronic test with *Daphnia magna,* significant reproductive impairment was found at 3 ug/L (USEPA, 1980). The bioconcentration factors for BEHP with fish and invertebrate species ranged from 54 to 2680 (USEPA, 1980).

F. Federal Regulations, Standards, Guidelines, and Criteria

Air:

- OSHA Permissible Exposure Limit (PEL) — 5 mg/m^3
- OSHA Short Term Exposure Limit (STEL) — 10 mg/m_3

Water:

- Ambient Water Quality Criteria, Freshwater:
  - Acute — 400 ug/L
  - Chronic — 360 ug/L

Drinking Water:

- Maximum Contaminant Level (MCL) proposed — 0.004 mg/L
- Maximum Contaminant Level Goal (MCLG) proposed — 0 mg/L

G. Toxicity Factors

Cancer Potency Factors $(mg/kg/day)^{-1}$: $1.4x10^{-2}$

Reference Doses (mg/kg-day):

	Subchronic	Chronic
Oral	0.02	0.02

H. References

Agency for Toxic Substances and Disease Registry (ATSDR). 1988. Toxicological Profile for Di(2-ethylhexyl)phthalate. Draft for Public Comment. U.S. Department of Health and Human Services.

Integrated Risk Information System (IRIS). 1992.

Sittig, M. (ed.) 1980. Priority Toxic Pollutants Health Impacts and Allowable Limits. New Jersey: Noyes Data Corporation, pp. 305-310.

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria Document for Phthalate Esters.

DIBENZOFURAN
CAS No. 132-64-9

A. Potential Sources and Exposure

Dibenzofuran is released into the environment in atmospheric emissions involved with the combustion of coal, biomass, refuse, and diesel fuel. The general population is primarily exposed to dibenzofuran through inhalation of air which has been contaminated by a variety of combustion sources.

B. Physical and Chemical Properties

Property	Value
Molecular Weight	168.19 g/mol
Vapor Pressure	0.0044 mm Hg at 25°C
Water Solubility	10 mg/L at 25°C.
log K_{ow}	4.12

C. Toxicity

There is minimal information available on the toxicity of dibenzofuran in the literature. There are no data on human exposures to the compound. It is believed that because of its structural relationship with dioxins, some of the toxicities might be similar. Acute early symptoms may include irritation and burning of the mucous membranes, skin and eyes, nausea and vomiting. Biochemical changes in liver metabolizing enzymes might occur as a result of acute and chronic exposures to dibenzofuran. Chloracne is considered an indicator of dioxin exposure, however it may not occur as frequently in exposures resulting from inhalation of dibenzofuran.

There are no data from which to derive a reference dose. There are no data on the possible carcinogenicity of dibenzofuran alone in humans. Studies have evaluated exposure to a mixture of polychlorinated biphenyls, polychlorinated dibenzofurans and polychlorinated quinones by consumptions of contaminated rice oil. There are no animal carcinogenicity data on dibenzofuran currently available. There is one study in which dibenzofuran was shown to be mutagenic.

D. Toxicokinetics

There are no data available on the toxicokinetics of dibenzofuran.

E. Ecological Effects

Dibenzofuran is biodegraded readily by soil microbes in the presence of sufficient oxygen. Bioconcentration studies have shown that dibenzofuran can bioaccumulate in aquatic organisms.

F. Federal Regulations, Standards, Guidelines, and Criteria

There are no current federal regulations, standards, guidelines, or criteria for dibenzofuran.

G. Toxicity Factors

There are no available toxicity factors for dibenzofuran.

H. References

Hazardous Substances Data Base (HSDB) on-line database.

Integrated Risk Information System (IRIS) on-line database.

APPENDIX E

Calculation of Risk Estimates

CALCULATION OF RISK ESTIMATES - TRESPASSER SCENARIO (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake					Chronic Hazard Index Estimates (Non-Carcinogenic Risks)				
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Sediment (mg/kg)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Sediment Ingestion (mg/kg/day)	Sediment Dermal (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Sediment Oral	Sediment Dermal
	Carcinogenic PAHs															
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	1.0E+00	5.0E-07	---	---	1.1E-07	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	---	6.0E-09	7.4E-01	6.8E-07	---	6.5E-10	8.1E-08	---	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.3E+00	7.3E-07	---	---	1.4E-07	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---		6.5E-07	---	---	---	---	---	---	---	---	---
	Inorganic Compounds															
7429-90-5	Aluminum	8.0E+03	7.6E-06	---	7.6E-06	9.2E+03	8.7E-04	9.1E-05	8.4E-07	1.0E-03	1.1E-04	8.7E-04	3.4E-04	2.4E-04	1.0E-03	4.1E-04
7440-38-2	Arsenic	9.0E+00	8.6E-09	---	8.6E-09	1.6E+01	9.9E-07	3.3E-07	9.5E-10	1.8E-06	5.9E-07	3.3E-03	1.2E-03	---	6.0E-03	2.1E-03
7440-41-7	Beryllium	3.6E-01	3.6E-10	---	3.6E-10		4.2E-08	4.4E-09	4.0E-11	---	---	8.3E-06	8.7E-05	---	---	---
7440-43-9	Cadmium (Food)	3.4E+00	3.3E-09	---	3.3E-09		3.8E-07	3.9E-08	3.6E-10	---	---	3.8E-04	1.6E-03	1.8E-06	---	---
7440-50-8	Copper		---	---	---	3.9E+02	---	---	---	4.3E-05	4.5E-06	---	---	---	3.3E-05	5.8E-06
7439-89-6	Iron	3.1E+04	3.0E-05	---	3.0E-05	2.7E+04	3.4E-03	3.5E-04	---	3.0E-03	3.1E-04	1.1E-02	1.2E-03	---	1.0E-02	1.0E-03
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07		6.9E-05	7.3E-06	6.7E-08	---	---	---	---	---	---	---
7439-96-5	Manganese	1.0E+03	9.9E-07	---	9.9E-07	1.3E+03	1.1E-04	1.2E-05	1.1E-07	1.4E-04	1.5E-05	4.9E-03	1.0E-02	2.2E-03	6.1E-03	1.3E-02

Pathway Risks				
2.1E-02	1.5E-02	2.4E-03	2.3E-02	1.7E-02

Total Hazard Index	7.7E-02
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - TRESPASSER SCENARIO (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates					Carcinogenic Risk Estimates				
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Sediment (mg/kg)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Sediment Ingestion (mg/kg/day)	Sediment Dermal (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Sediment Oral	Sediment Dermal
	Carcinogenic PAHs															
56-55-3	Benzo(a)anthracene	4.6E+00	—	—	—	1.0E+00	5.0E-08	—	—	1.1E-08	—	3.7E-08	—	—	8.0E-09	—
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	—	6.0E-09	7.4E-01	6.8E-08	—	6.5E-08	8.1E-09	—	5.0E-07	—	5.8E-11	5.9E-08	—
205-99-2	Benzo(b)fluoranthene	6.7E+00	—	—	—	1.3E+00	7.3E-08	—	—	1.4E-08	—	5.4E-08	—	—	1.0E-08	—
193-39-5	Indeno(123-cd)pyrene	5.9E+00	—	—	—		6.5E-08	—	—	—	—	4.7E-08	—	—	—	—
	Inorganic Compounds															
7429-90-5	Aluminum	8.0E+03	7.6E-06	—	7.6E-06	9.2E+03	8.7E-05	9.1E-06	8.4E-05	1.0E-04	1.1E-05	—	—	—	—	—
7440-38-2	Arsenic	9.0E+00	8.6E-09	—	8.6E-09	1.6E+01	9.9E-08	3.3E-08	9.5E-08	1.8E-07	5.9E-08	1.5E-07	5.2E-08	4.1E-10	2.7E-07	9.3E-08
7440-41-7	Beryllium	3.8E-01	3.6E-10	—	3.6E-10		4.2E-09	4.4E-10	4.0E-09	—	—	1.8E-08	1.9E-07	9.6E-12	—	—
7440-43-9	Cadmium (Food)	3.4E+00	3.3E-09	—	3.3E-09		3.8E-08	3.9E-09	3.6E-08	—	—	—	—	6.5E-11	—	—
7440-50-8	Copper		—	—	—	3.9E+02	—	—	—	4.3E-06	4.5E-07	—	—	—	—	—
7439-89-6	Iron	3.1E+04	3.0E-05	—	3.0E-05	2.7E+04	3.4E-04	3.5E-05	3.2E-04	3.0E-04	3.1E-05	—	—	—	—	—
7439-92-1	Lead	6.3E+02	6.1E-07	—	6.1E-07		6.9E-06	7.3E-07	6.7E-06	—	—	—	—	—	—	—
7439-96-5	Manganese	1.0E+03	9.9E-07	—	9.9E-07	1.3E+03	1.1E-05	1.2E-06	1.1E-05	1.4E-05	1.5E-06	—	—	—	—	—

Pathway Risks				
8.0E-07	2.4E-07	5.4E-10	3.5E-07	9.3E-08

Total Cancer Risk	1.5E-06
Reference Cancer Risk	1.0E-06

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - TRESPASSER SCENARIO (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake					Chronic Hazard Index Estimates (Non-Carcinogenic Risks)				
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Sediment (mg/kg)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Sediment Ingestion (mg/kg/day)	Sediment Dermal (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Sediment Oral	Sediment Dermal
	Carcinogenic PAHs															
56-55-3	Benzo(a)anthracene	2.0E+00	---	---	---	9.6E-01	2.2E-07	---	---	1.1E-07	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	2.7E+00	2.6E-09	---	2.6E-09	7.1E-01	3.0E-07	---	2.8E-10	7.8E-08	---	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	1.9E+00	---	---	---	1.2E+00	2.1E-07	---	---	1.3E-07	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	2.0E+00	---	---	---		2.2E-07	---	---	---	---	---	---	---	---	---
															---	---
	Inorganic Compounds														---	---
7429-90-5	Aluminum	5.5E+03	5.3E-06	---	5.3E-06	8.1E+03	6.0E-04	6.3E-05	5.8E-07	1.0E-03	1.1E-04	6.0E-04	2.3E-04	1.7E-04	1.0E-03	4.1E-04
7440-38-2	Arsenic	5.9E+00	5.7E-09	---	5.7E-09	1.3E+01	6.5E-07	2.2E-07	6.2E-10	1.8E-08	5.9E-07	2.2E-03	7.6E-04	---	6.0E-03	2.1E-03
7440-41-7	Beryllium	2.3E-01	2.2E-10	---	2.2E-10		2.5E-08	2.6E-09	2.4E-11	---	---	5.0E-06	5.3E-05	---	---	---
7440-43-9	Cadmium (Food)	1.2E+00	1.2E-09	---	1.2E-09		1.3E-07	1.4E-08	1.3E-10	---	---	1.3E-04	5.5E-04	6.3E-07	---	---
7440-50-8	Copper		---	---	---	2.9E+02	---	---	---	3.2E-05	3.4E-06	---	---	---	2.5E-05	4.3E-06
7439-89-6	Iron	1.8E+04	1.7E-05	---	1.7E-05	2.6E+04	2.0E-03	2.1E-04	---	3.0E-03	3.1E-04	6.5E-03	6.8E-04	---	1.0E-02	1.0E-03
7439-92-1	Lead	1.3E+02	1.2E-07	---	1.2E-07		1.4E-05	1.4E-06	1.3E-08	---	---	---	---	---	---	---
7439-96-5	Manganese	4.3E+02	4.1E-07	---	4.1E-07	9.9E+02	4.7E-05	4.9E-06	4.5E-08	1.4E-04	1.5E-05	2.1E-03	4.3E-03	9.0E-04	6.1E-03	1.3E-02

Pathway Risks				
1.1E-02	6.6E-03	1.1E-03	2.3E-02	1.7E-02

Total Hazard Index	5.9E-02
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - TRESPASSER SCENARIO (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates					Carcinogenic Risk Estimates				
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Sediment (mg/kg)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Sediment Ingestion (mg/kg/day)	Sediment Dermal (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Sediment Oral	Sediment Dermal
	Carcinogenic PAHs															
56-55-3	Benzo(a)anthracene	2.0E+00	---	---	---	9.6E-01	2.2E-08	---	---	1.1E-08	---	1.6E-08	---	---	7.7E-09	---
50-32-8	Benzo(a)pyrene	2.7E+00	2.6E-09	---	2.6E-09	7.1E-01	3.0E-08	---	2.8E-08	7.8E-09	---	2.2E-07	---	2.5E-11	5.7E-08	---
205-99-2	Benzo(b)fluoranthene	1.9E+00	---	---	---	1.2E+00	2.1E-08	---	---	1.3E-08	---	1.5E-08	---	---	9.2E-09	---
193-39-5	Indeno(123-cd)pyrene	2.0E+00	---	---	---		2.2E-08	---	---	---	---	1.6E-08	---	---	---	---
															---	---
	Inorganic Compounds														---	---
7429-90-5	Aluminum	5.5E+03	5.3E-06	---	5.3E-06	8.1E+03	6.0E-05	6.3E-06	5.8E-05	1.0E-04	1.1E-05	---	---	---	---	0.0E+00
7440-38-2	Arsenic	5.9E+00	5.7E-09	---	5.7E-09	1.3E+01	6.5E-08	2.2E-08	6.2E-08	1.8E-07	5.9E-08	9.7E-08	3.4E-08	2.7E-10	2.7E-07	9.3E-08
7440-41-7	Beryllium	2.3E-01	2.2E-10	---	2.2E-10		2.5E-09	2.6E-10	2.4E-09	---	---	1.1E-08	1.1E-07	5.8E-12	---	---
7440-43-9	Cadmium (Food)	1.2E+00	1.2E-09	---	1.2E-09		1.3E-08	1.4E-09	1.3E-08	---	---	---	---	2.3E-11	---	---
7440-50-8	Copper		---	---	---	2.9E+02	---	---	---	3.2E-06	3.4E-07	---	---	---	---	---
7439-89-6	Iron	1.8E+04	1.7E-05	---	1.7E-05	2.6E+04	2.0E-04	2.1E-05	1.9E-04	3.0E-04	3.1E-05	---	---	---	---	0.0E+00
7439-92-1	Lead	1.3E+02	1.2E-07	---	1.2E-07		1.4E-06	1.4E-07	1.3E-06	---	---	---	---	---	---	---
7439-96-5	Manganese	4.3E+02	4.1E-07	---	4.1E-07	9.9E+02	4.7E-06	4.9E-07	4.5E-06	1.4E-05	1.5E-06	---	---	---	---	---

Pathway Risks				
3.8E-07	1.5E-07	3.2E-10	3.4E-07	9.3E-08

Total Cancer Risk	9.6E-07
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - INDUSTRIAL WORKER SCENARIO (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

CAS #	COMPOUND	Exposure Point Concentrations				Chronic Average Daily Intake			Chronic Hazard Index Estimates (Non-Carcinogenic Risks)		
		Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	2.3E-06	---	---	---	---	---
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	3.0E-06	---	9.7E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	3.3E-06	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---	2.9E-06	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	7.9E+03	7.6E-06	---	7.6E-06	3.9E-03	1.3E-04	5.2E-06	3.9E-03	4.9E-04	1.5E-03
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09	5.0E-06	5.5E-07	6.8E-09	1.7E-02	1.9E-03	---
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10	2.6E-07	9.0E-09	3.6E-10	5.3E-05	1.8E-04	---
7440-43-9	Cadmium (Food)	5.8E+00	5.6E-09	---	5.6E-09	2.8E-06	9.6E-08	3.8E-09	2.8E-03	3.9E-03	1.9E-05
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	2.0E-02	7.0E-04	---	6.8E-02	2.3E-03	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	3.1E-04	1.1E-05	4.2E-07	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	3.9E-04	1.3E-05	5.3E-07	1.7E-02	1.2E-02	1.1E-02

Pathway Risks		
1.1E-01	2.0E-02	1.2E-02

Total Hazard Index	1.4E-01
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - INDUSTRIAL WORKER SCENARIO (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

CAS #	COMPOUND	Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
		Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	8.0E-07	---	---	5.9E-07	---	---
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	1.1E-06	---	3.5E-04	7.9E-06	---	3.1E-07
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.2E-06	---	---	8.5E-07	---	---
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---	1.0E-06	---	---	7.5E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	7.9E+03	7.6E-06	---	7.6E-06	1.4E-03	4.7E-05	1.9E-03	---	---	---
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09	1.8E-06	2.0E-07	2.4E-06	2.7E-06	3.1E-07	1.0E-08
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10	9.4E-08	3.2E-09	1.3E-07	4.1E-07	1.4E-06	3.0E-10
7440-43-9	Cadmium (Food)	5.8E+00	5.6E-09	---	5.6E-09	1.0E-06	3.4E-08	1.4E-06	---	---	2.5E-09
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	7.3E-03	2.5E-04	9.8E-03	---	---	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	1.1E-04	3.8E-06	1.5E-04	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	1.4E-04	4.8E-06	1.9E-04	---	---	---

Pathway Risks			
1.3E-05	1.7E-06	3.2E-07	

Total Cancer Risk	1.5E-05
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - INDUSTRIAL WORKER SCENARIO (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	Exposure Point Concentrations							(Non-Carcinogenic Risks)		
COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation
Volatile Organic Compounds										
Carcinogenic PAHs										
Benzo(a)anthracene	2.4E+00	—	—	—	1.2E-06	—	7.5E-07	—	—	—
Benzo(a)pyrene	3.2E+00	3.1E-09	4.2E-07	3.1E-09	1.6E-06	—	2.1E-09	—	—	—
Benzo(b)fluoranthene	2.5E+00	—	—	—	1.2E-06	—	—	—	—	—
Dibenzo(ah)anthracene	1.3E-01	—	—	—	6.4E-08	—	—	—	—	—
Indeno(123-cd)pyrene	2.5E+00	—	—	—	1.2E-06	—	—	—	—	—
Phthalates										
Aluminum	5.2E+03	5.0E-06	—	5.0E-06	2.5E-03	8.6E-05	3.4E-06	2.5E-03	3.2E-04	9.8E-04
Arsenic	5.6E+00	5.3E-09	—	5.3E-09	2.7E-06	3.0E-07	3.7E-09	9.1E-03	1.0E-03	—
Beryllium	2.7E-01	2.5E-10	—	2.5E-10	1.3E-07	4.4E-09	1.7E-10	2.6E-05	8.8E-05	—
Cadmium (Food)	1.8E+00	1.7E-09	—	1.7E-09	8.8E-07	3.0E-08	1.2E-09	8.8E-04	1.2E-03	5.9E-06
Iron	1.9E+04	1.8E-05	—	1.8E-05	9.1E-03	3.1E-04	—	3.0E-02	1.0E-03	—
Lead	1.4E+02	1.4E-07	—	1.4E-07	6.9E-05	2.3E-06	9.3E-08	—	—	—
Manganese	3.3E+02	3.2E-07	—	3.2E-07	1.6E-04	5.5E-06	2.2E-07	7.1E-03	4.8E-03	4.4E-03

Pathway Risks			
5.0E-02	8.5E-03	5.4E-03	

Total Hazard Index	6.4E-02
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - INDUSTRIAL WORKER SCENARIO (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	2.4E+00	---	---	---	4.1E-07	---	---	3.0E-07	---	---
50-32-8	Benzo(a)pyrene	3.2E+00	3.1E-09	4.2E-07	4.3E-07	5.7E-07	---	1.0E-04	4.1E-06	---	9.2E-08
205-99-2	Benzo(b)fluoranthene	2.5E+00	---	---	---	4.4E-07	---	---	3.2E-07	---	---
53-70-3	Dibenzo(ah)anthracene	1.3E-01	---	---	---	2.3E-08	---	---	1.7E-07	---	---
193-39-5	Indeno(123-cd)pyrene	2.5E+00	---	---	---	4.4E-07	---	---	3.2E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	5.2E+03	5.0E-06	---	5.0E-06	9.1E-04	3.1E-05	1.2E-03	---	---	---
7440-38-2	Arsenic	5.6E+00	5.3E-09	---	5.3E-09	9.7E-07	1.1E-07	1.3E-06	1.5E-06	1.7E-07	5.6E-09
7440-41-7	Beryllium	2.7E-01	2.5E-10	---	2.5E-10	4.6E-08	1.6E-09	6.2E-08	2.0E-07	6.8E-07	1.5E-10
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09	3.2E-07	1.1E-08	4.2E-07	---	---	7.6E-10
7439-89-6	Iron	1.9E+04	1.8E-05	---	1.8E-05	3.2E-03	1.1E-04	4.3E-03	---	---	---
7439-92-1	Lead	1.4E+02	1.4E-07	---	1.4E-07	2.5E-05	8.4E-07	3.3E-05	---	---	---
7439-96-5	Manganese	3.3E+02	3.2E-07	---	3.2E-07	5.8E-05	2.0E-06	7.8E-05	---	---	---

Pathway Risks		
6.9E-06	8.4E-07	9.9E-08

Total Cancer Risk	7.9E-06
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - ON-SITE CONSTRUCTION WORKER (RME Case)
SUBCHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
CAS #	COMPOUND	Surface and Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface and Subsurface Soil Ingestion (mg/kg/day)	Surface and Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface and Subsurface Soil Oral	Surface and Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.7E+01	---	---	---	2.2E-04	---	---	---	---	---
50-32-8	Benzo(a)pyrene	3.5E+01	6.0E-09	1.4E-06	1.4E-06	1.6E-04	---	9.7E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	3.9E+01	---	---	---	1.8E-04	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	1.8E+01	---	---	---	8.5E-05	---	---	---		---
53-70-3	Dibenzo(ah)anthracene	1.4E-01	---	---	---	6.4E-07	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	1.5E+01	---	---	---	7.0E-05	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.4E+04	7.6E-06	---	7.6E-06	6.7E-02	6.3E-04	5.2E-06	6.7E-02	2.3E-03	1.5E-03
7440-38-2	Arsenic	1.1E+01	9.9E-09	---	9.9E-09	5.0E-05	1.5E-06	6.8E-09	1.7E-01	5.2E-03	---
7440-41-7	Beryllium	8.1E-01	5.2E-10	---	5.2E-10	3.8E-06	3.6E-08	3.6E-10	7.6E-04	7.1E-04	---
7440-43-9	Cadmium (Food)	5.8E+00	5.6E-09	---	5.6E-09	2.7E-05	2.6E-07	3.8E-09	2.7E-02	1.0E-02	1.9E-05
7439-89-6	Iron	3.3E+04	4.0E-05	---	4.0E-05	1.5E-01	1.5E-03	2.7E-05	5.2E-01	4.9E-03	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	3.0E-03	2.8E-05	4.2E-07	---	---	---
7439-96-5	Manganese	9.4E+02	7.7E-07	---	7.7E-07	4.4E-03	4.2E-05	5.3E-07	1.9E-01	3.6E-02	1.1E-02
7440-28-0	Thallium	3.0E+00	---	---	---	1.4E-05	1.3E-07	---	1.8E-01	1.7E-03	---

Pathway Risks		
1.1E+00	6.1E-02	1.2E-02

Total Hazard Index	1.2E+00
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	9.5E-01	2.5E-02	1.5E-03
CNS Hazard Index	1.9E-01	3.6E-02	1.1E-02
Cardiovascular Hazard Index	---	---	0.0E+00
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00

Total Systemic	9.8E-01
Total CNS	2.4E-01
Total Cardiovascular	0.0E+00
Total Reproductive	0.0E+00

CALCULATION OF RISK ESTIMATES - ON-SITE CONSTRUCTION WORKER (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Surface and Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface and Subsurface Soil Ingestion (mg/kg/day)	Surface and Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface and Subsurface Soil Oral	Surface and Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.7E+01	---	---	---	3.2E-06	---	---	2.3E-06	---	---
50-32-8	Benzo(a)pyrene	3.5E+01	6.0E-09	1.4E-06	1.4E-06	2.3E-06	---	1.4E-05	1.7E-05	---	1.2E-08
205-99-2	Benzo(b)fluoranthene	3.9E+01	---	---	---	2.6E-06	---	---	1.9E-06	---	---
207-08-9	Benzo(k)fluoranthene	1.8E+01	---	---	---	1.2E-06	---	---	8.8E-08	---	---
53-70-3	Dibenzo(ah)anthracene	1.4E-01	---	---	---	9.1E-09	---	---	6.6E-08	---	---
193-39-5	Indeno(123-cd)pyrene	1.5E+01	---	---	---	1.0E-06	---	---	7.3E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.4E+04	7.6E-06	---	7.6E-06	9.6E-04	9.0E-06	7.4E-05	---	---	---
7440-38-2	Arsenic	1.1E+01	9.9E-09	---	9.9E-09	7.1E-07	2.1E-08	9.7E-08	1.1E-06	3.4E-08	4.2E-10
7440-41-7	Beryllium	8.1E-01	5.2E-10	---	5.2E-10	5.4E-08	5.1E-10	5.1E-09	2.3E-07	2.2E-07	1.2E-11
7440-43-9	Cadmium (Food)	5.8E+00	5.6E-09	---	5.6E-09	3.9E-07	3.7E-09	5.4E-08	---	---	9.8E-11
7439-89-6	Iron	3.3E+04	4.0E-05	---	4.0E-05	2.2E-03	2.1E-05	3.9E-04	---	---	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	4.3E-05	4.0E-07	6.0E-06	---	---	---
7439-96-5	Manganese	9.4E+02	7.7E-07	---	7.7E-07	6.3E-05	5.9E-07	7.6E-06	---	---	---
7440-28-0	Thallium	3.0E+00	---	---	---	2.0E-07	1.9E-09	---	---	---	---

Pathway Risks		
2.4E-05	2.5E-07	1.3E-08

Total Cancer Risk	2.4E-05
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - ON-SITE CONSTRUCTION WORKER (CTE Case)
SUBCHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
CAS #	COMPOUND	Surface and Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface and Subsurface Soil Ingestion (mg/kg/day)	Surface and Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface and Subsurface Soil Oral	Surface and Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	9.2E+00	---	---	---	4.3E-05	---	---	---	---	---
50-32-8	Benzo(a)pyrene	7.2E+00	3.1E-09	4.1E-07	4.1E-07	3.4E-05	---	2.8E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	7.0E+00	---	---	---	3.3E-05	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	2.2E+00	---	---	---	1.1E-05	---	---	---		---
53-70-3	Dibenzo(ah)anthracene	1.1E-01	---	---	---	5.2E-07	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.0E+00	---	---	---	1.4E-05	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	9.4E+03	5.0E-06	---	5.0E-06	4.4E-02	4.2E-04	3.4E-06	4.4E-02	1.5E-03	9.8E-04
7440-38-2	Arsenic	5.7E+00	5.3E-09	---	5.3E-09	2.7E-05	8.1E-07	3.7E-09	8.9E-02	2.8E-03	---
7440-41-7	Beryllium	5.3E-01	2.5E-10	---	2.5E-10	2.5E-06	2.4E-08	1.7E-10	5.0E-04	4.7E-04	---
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09	8.5E-06	8.0E-08	1.2E-09	8.5E-03	3.2E-03	5.9E-06
7439-89-6	Iron	2.2E+04	1.8E-05	---	1.8E-05	1.0E-01	9.7E-04	1.2E-05	3.4E-01	3.2E-03	---
7439-92-1	Lead	7.5E+01	1.4E-07	---	1.4E-07	3.5E-04	3.3E-06	9.3E-08	---	---	---
7439-96-5	Manganese	4.8E+02	3.2E-07	---	3.2E-07	2.3E-03	2.1E-05	2.2E-07	9.9E-02	1.9E-02	4.4E-03
7440-28-0	Thallium	1.2E+00	---	---	---	5.5E-06	5.2E-08	---	6.9E-02	6.5E-04	---

Pathway Risks		
6.5E-01	3.0E-02	5.4E-03

Total Hazard Index	6.88E-01
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	5.5E-01	1.2E-02	9.8E-04
CNS Hazard Index	9.9E-02	1.9E-02	4.4E-03
Cardiovascular Hazard Index	---	---	0.0E+00
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00

Total Systemic	5.7E-01
Total CNS	1.2E-01
Total Cardiovascular	0.0E+00
Total Reproductive	0.0E+00

CALCULATION OF RISK ESTIMATES - ON-SITE CONSTRUCTION WORKER (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Surface and Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Surface and Subsurface Soil Ingestion (mg/kg/day)	Surface and Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Surface and Subsurface Soil Oral	Surface and Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	9.2E+00	---	---	---	6.2E-07	---	---	4.5E-07	---	---
50-32-8	Benzo(a)pyrene	7.2E+00	3.1E-09	4.1E-07	4.1E-07	4.8E-07	---	4.1E-06	3.5E-06	---	3.6E-09
205-99-2	Benzo(b)fluoranthene	7.0E+00	---	---	---	4.7E-07	---	---	3.4E-07	---	---
207-08-9	Benzo(k)fluoranthene	2.2E+00	---	---	---	1.5E-07	---	---	1.1E-08	---	---
53-70-3	Dibenzo(ah)anthracene	1.1E-01	---	---	---	7.4E-09	---	---	5.4E-08	---	---
193-39-5	Indeno(123-cd)pyrene	3.0E+00	---	---	---	2.0E-07	---	---	1.5E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	9.4E+03	5.0E-06	---	5.0E-06	6.3E-04	6.0E-06	4.9E-05	---	---	---
7440-38-2	Arsenic	5.7E+00	5.3E-09	---	5.3E-09	3.8E-07	1.2E-08	5.2E-08	5.7E-07	1.8E-08	2.3E-10
7440-41-7	Beryllium	5.3E-01	2.5E-10	---	2.5E-10	3.6E-08	3.4E-10	2.5E-09	1.5E-07	1.4E-07	6.0E-12
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09	1.2E-07	1.1E-09	1.7E-08	---	---	3.0E-11
7439-89-6	Iron	2.2E+04	1.8E-05	---	1.8E-05	1.5E-03	1.4E-05	1.7E-04	---	---	---
7439-92-1	Lead	7.5E+01	1.4E-07	---	1.4E-07	5.0E-06	4.7E-08	1.3E-06	---	---	---
7439-96-5	Manganese	4.8E+02	3.2E-07	---	3.2E-07	3.2E-05	3.0E-07	3.1E-06	---	---	---
7440-28-0	Thallium	1.2E+00	---	---	---	7.9E-08	7.4E-10	---	---	---	---

Pathway Risks		
5.2E-06	1.6E-07	3.9E-09

Total Cancer Risk	5.4E-06
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (RME Case)
SUBCHRONIC RISK
Betweeen the Susquehanna River and the waste water treatment plant
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
Carcinogenic PAHs										
Benzo(a)anthracene	1.1E+01	---	---	---	5.2E-05	---	---	---	---	---
Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	4.4E-05	---	2.6E-07	---	---	---
Benzo(b)fluoranthene	7.6E+00	---	---	---	3.6E-05	---	---	---	---	---
Benzo(k)fluoranthene	6.6E+00	---	---	---	3.1E-05	---	---	---		---
Dibenzo(ah)anthracene	3.6E-01	---	---	---	1.7E-06	---	---	---	---	---
Indeno(123-cd)pyrene	6.0E+00	---	---	---	2.8E-05	---	---	---	---	---
Inorganic Compounds										
Aluminum	1.6E+04	---	---	---	7.7E-02	7.2E-04	---	7.7E-02	2.7E-03	---
Arsenic	6.9E+00	---	---	---	3.2E-05	9.8E-07	---	1.1E-01	3.4E-03	---
Beryllium	1.6E+00	---	---	---	7.5E-06	7.1E-08	---	1.5E-03	1.4E-03	---
Iron	2.6E+04	---	---	---	1.2E-01	1.1E-03	---	4.0E-01	3.8E-03	---
Manganese	8.5E+02	---	---	---	4.0E-03	3.7E-05	---	1.7E-01	3.3E-02	---

Pathway Risks		
7.7E-01	4.5E-02	0.0E+00

Total Hazard Index	8.1E-01
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (RME Case)
CARCINOGENIC RISK
Betweeen the Susquehanna River and the waste water treatment plant
UGI Columbia *Former* MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	1.1E+01	---	---	---	7.4E-07	---	---	5.4E-07	---	---
50-32-8	Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	6.2E-07	---	3.7E-06	4.6E-06	---	3.2E-09
205-99-2	Benzo(b)fluoranthene	7.6E+00	---	---	---	5.1E-07	---	---	3.7E-07	---	---
207-08-9	Benzo(k)fluoranthene	6.6E+00	---	---	---	4.4E-07	---	---	3.2E-08	---	---
53-70-3	Dibenzo(ah)anthracene	3.6E-01	---	---	---	2.4E-08	---	---	1.8E-07	---	---
193-39-5	Indeno(123-cd)pyrene	6.0E+00	---	---	---	4.0E-07	---	---	2.9E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.6E+04	---	---	---	1.1E-03	1.0E-05	---	---	---	---
7440-38-2	Arsenic	6.9E+00	---	---	---	4.6E-07	1.4E-08	---	6.9E-07	2.2E-08	---
7440-41-7	Beryllium	1.6E+00	---	---	---	1.1E-07	1.0E-09	---	4.6E-07	4.3E-07	---
7439-89-6	Iron	2.6E+04	---	---	---	1.7E-03	1.6E-05	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	5.7E-05	5.4E-07	---	---	---	---

Pathway Risks		
7.1E-06	4.6E-07	3.2E-09

Total Cancer Risk	7.6E-06
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (CTE Case)
SUBCHRONIC RISK
Betweeen the Susquehanna River and the waste water treatment plant
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	2.8E-05	---	---	---	---	---
50-32-8	Benzo(a)pyrene	5.6E+00	---	2.3E-07	2.3E-07	2.6E-05	---	1.5E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	1.9E-05	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---	1.1E-05	---	---	---		---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---	8.9E-07	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---	1.5E-05	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.4E+04	---	---	---	6.7E-02	6.3E-04	---	6.7E-02	2.3E-03	---
7440-38-2	Arsenic	6.8E+00	---	---	---	3.2E-05	9.6E-07	---	1.1E-01	3.4E-03	---
7440-41-7	Beryllium	1.2E+00	---	---	---	5.7E-06	5.4E-08	---	1.1E-03	1.1E-03	---
7439-89-6	Iron	2.4E+04	---	---	---	1.1E-01	1.1E-03	---	3.7E-01	3.5E-03	---
7439-96-5	Manganese	6.1E+02	---	---	---	2.9E-03	2.7E-05	---	1.2E-01	2.4E-02	---
7440-28-0	Thallium	8.8E-01	---	---	---	4.1E-06	3.9E-08	---	5.2E-02	4.9E-04	---

Pathway Risks		
7.2E-01	3.4E-02	0.0E+00

Total Hazard Index	7.6E-01
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (CTE Case)
CARCINOGENIC RISK
Betweeen the Susquehanna River and the waste water treatment plant
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	4.0E-07	---	---	2.9E-07	---	---
50-32-8	Benzo(a)pyrene	5.6E+00	---	2.3E-07	2.3E-07	3.8E-07	---	2.2E-06	2.7E-06	---	2.0E-09
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	2.8E-07	---	---	2.0E-07	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---	1.6E-07	---	---	1.2E-08	---	---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---	1.3E-08	---	---	9.3E-08	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---	2.1E-07	---	---	1.6E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.4E+04	---	---	---	9.5E-04	9.0E-06	---	---	---	---
7440-38-2	Arsenic	6.8E+00	---	---	---	4.6E-07	1.4E-08	---	6.8E-07	2.2E-08	---
7440-41-7	Beryllium	1.2E+00	---	---	---	8.1E-08	7.6E-10	---	3.5E-07	3.3E-07	---
7439-89-6	Iron	2.4E+04	---	---	---	1.6E-03	1.5E-05	---	---	---	---
7439-96-5	Manganese	6.1E+02	---	---	---	4.1E-05	3.9E-07	---	---	---	---
7440-28-0	Thallium	8.8E-01	---	---	---	5.9E-08	5.6E-10	---	---	---	---

Pathway Risks		
4.5E-06	3.5E-07	2.0E-09

Total Cancer Risk	4.9E-06
Reference Cancer Risk	1.0E-06

IR/GINAL (Req)

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (RME Case)
SUBCHRONIC RISK
Betweeen Front St. and the Railroad Tracks
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.7E+01	---	---	---	2.2E-04	---	---	---	---	---
50-32-8	Benzo(a)pyrene	2.4E+01	---	9.7E-07	9.7E-07	1.1E-04	---	6.6E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	2.1E+01	---	---	---	9.9E-05	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	3.2E+00	---	---	---	1.5E-05	---	---	---		---
53-70-3	*Dibenzo(ah)anthracene*	8.4E+00	---	---	---	3.9E-05	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.1E+00	---	---	---	1.5E-05	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.6E+04	---	---	---	7.7E-02	7.2E-04	---	7.7E-02	2.7E-03	---
7440-38-2	Arsenic	7.0E+00	---	---	---	3.3E-05	9.9E-07	---	1.1E-01	3.5E-03	---
7440-41-7	Beryllium	1.6E+00	---	---	---	7.5E-06	7.1E-08	---	1.5E-03	1.4E-03	---
7439-89-6	Iron	2.6E+04	---	---	---	1.2E-01	1.1E-03	---	4.0E-01	3.8E-03	---
7439-96-5	Manganese	8.5E+02	---	---	---	4.0E-03	3.7E-05	---	1.7E-01	3.3E-02	---

Pathway Risks		
7.7E-01	4.5E-02	0.0E+00

Total Hazard Index	8.1E-01
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (RME Case)
CARCINOGENIC RISK
Betweeen Front St. and the Railroad Tracks
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	4.7E+01	---	---	---	3.2E-06	---	---	2.3E-06	---	---
50-32-8	Benzo(a)pyrene	2.4E+01	---	9.7E-07	9.7E-07	1.6E-06	---	9.5E-06	1.2E-05	---	8.4E-09
205-99-2	Benzo(b)fluoranthene	2.1E+01	---	---	---	1.4E-06	---	---	1.0E-06	---	---
207-08-9	Benzo(k)fluoranthene	3.2E+00	---	---	---	2.1E-07	---	---	1.6E-08	---	---
53-70-3	Dibenzo(ah)anthracene	8.4E+00	---	---	---	5.6E-07	---	---	4.1E-06	---	---
193-39-5	Indeno(123-cd)pyrene	3.1E+00	---	---	---	2.1E-07	---	---	1.5E-07	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	1.6E+04	---	---	---	1.1E-03	1.0E-05	---	---	---	---
7440-38-2	Arsenic	7.0E+00	---	---	---	4.7E-07	1.4E-08	---	7.0E-07	2.2E-08	---
7440-41-7	Beryllium	1.6E+00	---	---	---	1.1E-07	1.0E-09	---	4.6E-07	4.3E-07	---
7439-89-6	Iron	2.6E+04	---	---	---	1.7E-03	1.6E-05	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	5.7E-05	5.4E-07	---	---	---	---

Pathway Risks		
2.1E-05	4.6E-07	8.4E-09

Total Cancer Risk	2.1E-05
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (CTE Case)
SUBCHRONIC RISK
Betweeen Front St. and the Railroad Tracks
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Subchronic Average Daily Intake			Subchronic Hazard Index Estimates (Non-Carcinogenic Risks)		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	1.3E+01	---	---	---	6.1E-05	---	---	---	---	---
50-32-8	Benzo(a)pyrene	6.9E+00	---	2.8E-07	2.8E-07	3.2E-05	---	1.9E-07	---	---	---
205-99-2	Benzo(b)fluoranthene	6.0E+00	---	---	---	2.8E-05	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	1.1E+00	---	---	---	5.2E-06	---	---	---		---
53-70-3	Dibenzo(ah)anthracene	2.2E+00	---	---	---	1.0E-05	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	1.1E+00	---	---	---	5.2E-06	---	---	---	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	6.6E+03	---	---	---	3.1E-02	2.9E-04	---	3.1E-02	1.1E-03	---
7440-38-2	Arsenic	6.9E+00	---	---	---	3.2E-05	9.8E-07	---	1.1E-01	3.4E-03	---
7440-41-7	Beryllium	8.3E-01	---	---	---	3.9E-06	3.7E-08	---	7.8E-04	7.3E-04	---
7439-89-6	Iron	1.9E+04	---	---	---	8.9E-02	8.4E-04	---	3.0E-01	2.8E-03	---
7439-96-5	Manganese	5.0E+02	---	---	---	2.3E-03	2.2E-05	---	1.0E-01	1.9E-02	---
7440-28-0	Thallium	8.0E-01	---	---	---	3.8E-06	3.5E-08	---	4.7E-02	4.4E-04	---

Pathway Risks		
5.8E-01	2.8E-02	0.0E+00

Total Hazard Index	6.1E-01
Reference Hazard Index	1.00 (Default)

CALCULATION OF RISK ESTIMATES - OFF-SITE CONSTRUCTION WORKER SCENARIO (CTE Case)
CARCINOGENIC RISK
Betweeen Front St. and the Railroad Tracks
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations				Average Daily Intake (Lifetime) Estimates			Carcinogenic Risk Estimates		
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	*Subsurface Soil* Ingestion (mg/kg/day)	*Subsurface Soil* Dermal (mg/kg/day)	*Air (Total)* Inhalation (mg/m3)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation
	Carcinogenic PAHs										
56-55-3	Benzo(a)anthracene	1.3E+01	---	---	---	8.7E-07	---	---	6.4E-07	---	---
50-32-8	Benzo(a)pyrene	6.9E+00	---	2.8E-07	2.8E-07	4.6E-07	---	2.7E-06	3.4E-06	---	2.4E-09
205-99-2	Benzo(b)fluoranthene	6.0E+00	---	---	---	4.0E-07	---	---	2.9E-07	---	---
207-08-9	Benzo(k)fluoranthene	1.1E+00	---	---	---	7.4E-08	---	---	5.4E-09	---	---
53-70-3	Dibenzo(ah)anthracene	2.2E+00	---	---	---	1.4E-07	---	---	1.1E-06	---	---
193-39-5	Indeno(123-cd)pyrene	1.1E+00	---	---	---	7.4E-08	---	---	5.4E-08	---	---
	Inorganic Compounds										
7429-90-5	Aluminum	6.6E+03	---	---	---	4.4E-04	4.2E-06	---	---	---	---
7440-38-2	Arsenic	6.9E+00	---	---	---	4.6E-07	1.4E-08	---	6.9E-07	2.2E-08	---
7440-41-7	Beryllium	8.3E-01	---	---	---	5.6E-08	5.2E-10	---	2.4E-07	2.3E-07	---
7439-89-6	Iron	1.9E+04	---	---	---	1.3E-03	1.2E-05	---	---	---	---
7439-96-5	Manganese	5.0E+02	---	---	---	3.3E-05	3.2E-07	---	---	---	---
7440-28-0	Thallium	8.0E-01	---	---	---	5.4E-08	5.1E-10	---	---	---	---

Pathway Risks		
6.4E-06	2.5E-07	2.4E-09

Total Cancer Risk	6.6E-06
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD OFF-SITE RESIDENT (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.9E+01	---	---	---	2.7E+00	---	---	---	8.9E+02
100-41-4	Ethylbenzene		---	---	---	2.0E+00	---	---	---	1.4E-01	---	---	---	1.4E+00
108-88-3	Toluene		---	---	---	2.6E+00	---	---	---	1.8E-01	---	---	---	8.9E-01
	1,2,4-Trimethylbenzene		---	---	---	1.1E-01	---	---	---	7.5E-03	---	---	---	1.5E-01
1330-20-7	Xylenes (Mixed)		---	---	---	6.0E-01	---	---	---	4.1E-02	---	---	---	2.1E-02
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	1.5E-01	---	---	---	1.0E-02	---	---	---	1.7E-01
	1-Methylnaphthalene		---	---	---	3.5E-01	---	---	---	2.4E-02	---	---	---	6.0E-01
91-57-6	2-Methylnaphthalene		---	---	---	3.9E-01	---	---	---	2.7E-02	---	---	---	6.7E-01
91-20-3	Naphthalene		---	---	---	6.3E+00	---	---	---	4.3E-01	---	---	---	1.1E+01
85-01-8	Phenanthrene		---	---	---	4.0E-02	---	---	---	2.7E-03	---	---	---	6.8E-02
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	1.1E+01	---	---	---	ND	1.5E-04	---	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	ND	1.3E-04	---	3.6E-07	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	7.6E+00	---	---	---	ND	1.0E-04	---	---	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	6.6E+00	---	---	---		9.0E-05	---	---	---	---	---	---	---
53-70-3	Dibenzo(ah)anthracene	3.6E-01	---	---	---	NA	4.9E-06	---	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	6.0E+00	---	---	---	NA	8.2E-05	---	---	---	---	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.4E-02	---	---	---	9.6E-04	---	---	---	2.4E-01
	Inorganic Compounds													
7429-90-5	Aluminum	1.6E+04	---	---	---	1.0E-01	2.2E-01	1.8E-03	---	6.8E-03	2.2E-01	6.8E-03	---	6.8E-03
7440-38-2	Arsenic	6.9E+00	---	---	---	ND	9.5E-05	2.5E-06	---	---	3.2E-01	8.7E-03	---	---
7440-39-3	Barium		---	---	---	1.2E-01	---	---	---	8.2E-03	---	---	---	1.2E-01
7440-41-7	Beryllium	1.6E+00	---	---	---	ND	2.2E-05	1.8E-07	---	---	4.4E-03	3.6E-03	---	---
57-12-5	Cyanide		---	---	---	2.2E-01	---	---	---	1.5E-02	---	---	---	7.5E-01
7439-89-6	Iron	2.6E+04	---	---	---	2.5E+01	3.5E-01	2.9E-03	---	1.7E+00	1.2E+00	9.6E-03	---	5.6E+00
7439-92-1	Lead		---	---	---	1.5E-03	---	---	---	1.0E-04	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	3.0E+00	1.2E-02	9.5E-05	---	2.1E-01	5.0E-01	8.3E-02	---	8.9E+00
7440-28-0	Thallium	1.1E+00	---	---	---	ND	1.4E-05	1.2E-07	---	---	1.8E-01	1.5E-03	---	---

Total Pathway Risks			
2.4E+00	1.1E-01	0.0E+00	9.2E+02

Total Hazard Index	9.2E+02
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.9E+00	3.0E-02	0.0E+00	9.1E+02
CNS Hazard Index	5.0E-01	8.3E-02	0.0E+00	8.9E+00
Cardiovascular Hazard Index	---	---	0.0E+00	1.2E-01
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	2.1E-02

Total Systemic	9.1E+02
Total CNS	9.5E+00
Total Cardiovascular	1.2E-01
Total Reproductive	2.1E-02

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD OFF-SITE RESIDENT (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.9E+01	---	---	---	2.3E-01	---	---	---	6.6E-03
100-41-4	Ethylbenzene		---	---	---	2.0E+00	---	---	---	1.2E-02	---	---	---	---
108-88-3	Toluene		---	---	---	2.6E+00	---	---	---	1.5E-02	---	---	---	---
	1,2,4-Trimethylbenzene		---	---	---	1.1E-01	---	---	---	6.5E-04	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	6.0E-01	---	---	---	3.5E-03	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	1.5E-01	---	---	---	8.8E-04	---	---	---	---
	1-Methylnaphthalene		---	---	---	3.5E-01	---	---	---	2.1E-03	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	3.9E-01	---	---	---	2.3E-03	---	---	---	---
91-20-3	Naphthalene		---	---	---	6.3E+00	---	---	---	3.7E-02	---	---	---	---
85-01-8	Phenanthrene		---	---	---	4.0E-02	---	---	---	2.3E-04	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	1.1E+01	---	---	---	ND	1.3E-05	---	---	---	9.4E-06	---	---	---
50-32-8	Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	ND	1.1E-05	---	3.1E-05	---	8.0E-05	---	2.7E-08	---
205-99-2	Benzo(b)fluoranthene	7.6E+00	---	---	---	ND	8.9E-06	---	---	---	6.5E-06	---	---	---
207-08-9	Benzo(k)fluoranthene	6.6E+00	---	---	---		7.7E-06	---	---	---	5.7E-07	---	---	---
53-70-3	Dibenzo(ah)anthracene	3.6E-01	---	---	---	NA	4.2E-07	---	---	---	3.1E-06	---	---	---
193-39-5	Indeno(123-cd)pyrene	6.0E+00	---	---	---	NA	7.0E-06	---	---	---	5.1E-06	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.4E-02	---	---	---	8.2E-05	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	1.6E+04	---	---	---	1.0E-01	1.9E-02	1.6E-04	---	5.9E-04	---	---	---	---
7440-38-2	Arsenic	6.9E+00	---	---	---	ND	8.1E-06	2.1E-07	---	---	1.2E-05	3.4E-07	---	---
7440-39-3	Barium		---	---	---	1.2E-01	---	---	---	7.0E-04	---	---	---	---
7440-41-7	Beryllium	1.6E+00	---	---	---	ND	1.9E-06	1.5E-08	---	---	8.1E-06	6.6E-06	---	---
57-12-5	Cyanide		---	---	---	2.2E-01	---	---	---	1.3E-03	---	---	---	---
7439-89-6	Iron	2.6E+04	---	---	---	2.5E+01	3.0E-02	2.5E-04	---	1.4E-01	---	---	---	---
7439-92-1	Lead		---	---	---	1.5E-03	---	---	---	8.8E-06	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	3.0E+00	9.9E-04	8.1E-06	---	1.8E-02	---	---	---	---
7440-28-0	Thallium	1.1E+00	---	---	---	ND	1.2E-06	1.0E-08	---	---	---	---	---	---

Pathway Risks			
1.2E-04	7.0E-06	2.7E-08	6.6E-03

Total Cancer Risk	6.7E-03
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD OFF-SITE RESIDENT (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.1E+00	---	---	---	2.1E-01	---	---	---	7.1E+01
100-41-4	Ethylbenzene		---	---	---	3.7E-01	---	---	---	2.5E-02	---	---	---	2.5E-01
108-88-3	Toluene		---	---	---	9.0E-01	---	---	---	6.2E-02	---	---	---	3.1E-01
	1,2,4-Trimethylbenzene		---	---	---	5.7E-02	---	---	---	3.9E-03	---	---	---	7.8E-02
1330-20-7	Xylenes (Mixed)		---	---	---	3.1E-01	---	---	---	2.1E-02	---	---	---	1.1E-02
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	4.8E-02	---	---	---	3.3E-03	---	---	---	5.5E-02
	1-Methylnaphthalene		---	---	---	1.1E-01	---	---	---	7.5E-03	---	---	---	1.9E-01
91-57-6	2-Methylnaphthalene		---	---	---	1.0E-01	---	---	---	6.8E-03	---	---	---	1.7E-01
91-20-3	Naphthalene		---	---	---	8.0E-01	---	---	---	5.5E-02	---	---	---	1.4E+00
85-01-8	Phenanthrene		---	---	---	3.0E-02	---	---	---	2.1E-03	---	---	---	5.1E-02
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	ND	8.1E-05	---	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	5.8E+00	---	2.2E-07	2.2E-07	ND	7.6E-05	---	2.2E-07	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	ND	5.6E-05	---	---	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---		3.2E-05	---	---	---	---	---	---	---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---		2.6E-06	---	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---		4.4E-05	---	---	---	---	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.2E-02	---	---	---	8.2E-04	---	---	---	2.1E-01
	Inorganic Compounds													
7429-90-5	Aluminum	1.4E+00	---	---	---	4.2E-02	1.9E-05	1.6E-07	---	2.9E-03	1.9E-05	5.8E-07	---	2.9E-03
7440-38-2	Arsenic	6.8E+00	---	---	---		9.3E-05	2.4E-06	---	---	3.1E-01	8.6E-03	---	---
7440-39-3	Barium		---	---	---	9.0E-02	---	---	---	6.2E-03	---	---	---	8.8E-02
7440-41-7	Beryllium	1.2E+00	---	---	---		1.7E-05	1.4E-07	---	---	3.3E-03	2.7E-03	---	---
57-12-5	Cyanide		---	---	---	6.2E-02	---	---	---	4.2E-03	---	---	---	2.1E-01
7439-89-6	Iron	2.4E+00	---	---	---	3.0E+00	3.3E-05	2.7E-07	---	2.1E-01	1.1E-04	8.9E-07	---	6.8E-01
7439-92-1	Lead		---	---	---	1.2E-03	---	---	---	8.2E-05	---	---	---	---
7439-96-5	Manganese	6.1E+00	---	---	---	6.9E-01	8.4E-05	6.9E-07	---	4.7E-02	3.6E-03	6.0E-04	---	2.1E+00
7440-28-0	Thallium	8.8E+00	---	---	---		1.2E-04	9.8E-07	---	---	1.5E+00	1.2E-02	---	---

Total Pathway Risks			
1.8E+00	2.4E-02	0.0E+00	7.7E+01

Total Hazard Index	7.8E+01
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.8E+00	2.4E-02	0.0E+00	7.4E+01
CNS Hazard Index	3.6E-03	6.0E-04	0.0E+00	2.1E+00
Cardiovascular Hazard Index	---	---	0.0E+00	8.8E-02
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	1.1E-02

Total Systemic	7.6E+01
Total CNS	2.1E+00
Total Cardiovascular	8.8E-02
Total Reproductive	1.1E-02

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD OFF-SITE RESIDENT (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.1E+00	---	---	---	1.8E-02	---	---	---	5.3E-04
100-41-4	Ethylbenzene		---	---	---	3.7E-01	---	---	---	2.2E-03	---	---	---	---
108-88-3	Toluene		---	---	---	9.0E-01	---	---	---	5.3E-03	---	---	---	---
	1,2,4-Trimethylbenzene		---	---	---	5.7E-02	---	---	---	3.3E-04	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	3.1E-01	---	---	---	1.8E-03	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	4.8E-02	---	---	---	2.8E-04	---	---	---	---
	1-Methylnaphthalene		---	---	---	1.1E-01	---	---	---	6.5E-04	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	1.0E-01	---	---	---	5.9E-04	---	---	---	---
91-20-3	Naphthalene		---	---	---	8.0E-01	---	---	---	4.7E-03	---	---	---	---
85-01-8	Phenanthrene		---	---	---	3.0E-02	---	---	---	1.8E-04	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	ND	7.0E-06	---	---	---	5.1E-06	---	---	---
50-32-8	Benzo(a)pyrene	5.6E+00	---	2.2E-07	2.2E-07	ND	6.6E-06	---	1.8E-05	---	4.8E-05	---	1.6E-08	---
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	ND	4.8E-06	---	---	---	3.5E-06	---	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---		2.8E-06	---	---	---	2.0E-07	---	---	---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---		2.2E-07	---	---	---	1.6E-06	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---		3.7E-06	---	---	---	2.7E-06	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.2E-02	---	---	---	7.0E-05	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	1.4E+00	---	---	---	4.2E-02	1.7E-06	1.4E-08	---	2.5E-04	---	---	---	---
7440-38-2	Arsenic	6.8E+00	---	---	---		8.0E-06	2.1E-07	---	---	1.2E-05	3.3E-07	---	---
7440-39-3	Barium		---	---	---	9.0E-02	---	---	---	5.3E-04	---	---	---	---
7440-41-7	Beryllium	1.2E+00	---	---	---		1.4E-06	1.2E-08	---	---	6.1E-06	5.0E-06	---	---
57-12-5	Cyanide		---	---	---	6.2E-02	---	---	---	3.6E-04	---	---	---	---
7439-89-6	Iron	2.4E+00	---	---	---	3.0E+00	2.8E-06	2.3E-08	---	1.6E-02	---	---	---	---
7439-92-1	Lead		---	---	---	1.2E-03	---	---	---	7.0E-06	---	---	---	---
7439-96-5	Manganese	6.1E+00	---	---	---	6.9E-01	7.2E-06	5.9E-08	---	4.1E-03	---	---	---	---
7440-28-0	Thallium	8.8E+00	---	---	---		1.0E-05	8.4E-08	---	---	---	---	---	---

Pathway Risks			
7.9E-05	5.3E-06	1.6E-08	5.3E-04

Total Cancer Risk	8.1E-04
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN OFF-SITE RESIDENT (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.9E+01	---	---	---	1.1E+00	---	---	---	3.6E+02
100-41-4	Ethylbenzene		---	---	---	2.0E+00	---	---	---	5.5E-02	---	---	---	5.5E-01
108-88-3	Toluene		---	---	---	2.6E+00	---	---	---	7.1E-02	---	---	---	3.6E-01
	1,2,4-Trimethylbenzene		---	---	---	1.1E-01	---	---	---	3.0E-03	---	---	---	6.0E-02
1330-20-7	Xylenes (Mixed)		---	---	---	6.0E-01	---	---	---	1.6E-02	---	---	---	8.2E-03
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	1.5E-01	---	---	---	4.1E-03	---	---	---	6.8E-02
	1-Methylnaphthalene		---	---	---	3.5E-01	---	---	---	9.6E-03	---	---	---	2.4E-01
91-57-6	2-Methylnaphthalene		---	---	---	3.9E-01	---	---	---	1.1E-02	---	---	---	2.7E-01
91-20-3	Naphthalene		---	---	---	6.3E+00	---	---	---	1.7E-01	---	---	---	4.3E+00
85-01-8	Phenanthrene		---	---	---	4.0E-02	---	---	---	1.1E-03	---	---	---	2.7E-02
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	1.1E+01	---	---	---	ND	1.5E-05	---	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	ND	1.3E-05	---	3.6E-07	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	7.6E+00	---	---	---	ND	1.0E-05	---	---	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	6.6E+00	---	---	---		9.0E-06	---	---	---	---	---	---	---
53-70-3	Dibenzo(ah)anthracene	3.6E-01	---	---	---		4.9E-07	---	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	6.0E+00	---	---	---		8.2E-06	2.6E-06	---	---	---	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.4E-02	---	---	---	3.8E-04	---	---	---	9.6E-02
	Inorganic Compounds													
7429-90-5	Aluminum	1.6E+04	---	---	---	1.0E-01	2.2E-02	7.0E-04	---	2.7E-03	2.2E-02	2.6E-03	---	2.7E-03
7440-38-2	Arsenic	6.9E+00	---	---	---		9.5E-06	9.7E-07	---	---	3.2E-02	3.4E-03	---	---
7440-39-3	Barium		---	---	---	1.2E-01	---	---	---	3.3E-03	---	---	---	4.7E-02
7440-41-7	Beryllium	1.6E+00	---	---	---		2.2E-06	7.0E-08	---	---	4.4E-04	1.4E-03	---	---
57-12-5	Cyanide		---	---	---	2.2E-01	---	---	---	6.0E-03	---	---	---	3.0E-01
7439-89-6	Iron	2.6E+04	---	---	---	2.5E+01	3.6E-02	1.1E-03	---	6.7E-01	1.2E-01	3.8E-03	---	2.2E+00
7439-92-1	Lead		---	---	---	1.5E-03	---	---	---	4.1E-05	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	3.0E+00	1.2E-03	3.7E-05	---	8.2E-02	5.1E-02	3.3E-02	---	3.6E+00
7440-28-0	Thallium	1.1E+00	---	---	---		1.5E-06	4.8E-08	---	---	1.9E-02	6.1E-04	---	---

Total Pathway Risks			
2.4E-01	4.4E-02	0.0E+00	3.7E+02

Total Hazard Index	3.7E+02
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.9E-01	1.2E-02	0.0E+00	3.6E+02
CNS Hazard Index	5.1E-02	3.3E-02	0.0E+00	3.6E+00
Cardiovascular Hazard Index	---	---	0.0E+00	4.7E-02
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	8.2E-03

Total Systemic	3.6E+02
Total CNS	3.7E+00
Total Cardiovascular	4.7E-02
Total Reproductive	8.2E-03

IR(GW)A1 (Req)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN OFF-SITE RESIDENT (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.9E+01	---	---	---	3.7E-01	---	---	---	1.1E-02
100-41-4	Ethylbenzene		---	---	---	2.0E+00	---	---	---	1.9E-02	---	---	---	---
108-88-3	Toluene		---	---	---	2.6E+00	---	---	---	2.4E-02	---	---	---	---
	1,2,4-Trimethylbenzene		---	---	---	1.1E-01	---	---	---	1.0E-03	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	6.0E-01	---	---	---	5.6E-03	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	1.5E-01	---	---	---	1.4E-03	---	---	---	---
	1-Methylnaphthalene		---	---	---	3.5E-01	---	---	---	3.3E-03	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	3.9E-01	---	---	---	3.7E-03	---	---	---	---
91-20-3	Naphthalene		---	---	---	6.3E+00	---	---	---	5.9E-02	---	---	---	---
85-01-8	Phenanthrene		---	---	---	4.0E-02	---	---	---	3.8E-04	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	1.1E+01	---	---	---	ND	5.2E-06	---	---	---	3.6E-06	---	---	---
50-32-8	Benzo(a)pyrene	9.3E+00	---	3.7E-07	3.7E-07	ND	4.4E-06	---	1.2E-04	---	3.2E-05	---	1.1E-07	---
205-99-2	Benzo(b)fluoranthene	7.6E+00	---	---	---	ND	3.6E-06	---	---	---	2.6E-06	---	---	---
207-08-9	Benzo(k)fluoranthene	6.6E+00	---	---	---		3.1E-06	---	---	---	2.3E-07	---	---	---
53-70-3	Dibenzo(ah)anthracene	3.6E-01	---	---	---		1.7E-07	---	---	---	1.2E-06	---	---	---
193-39-5	Indeno(123-cd)pyrene	6.0E+00	---	---	---		2.8E-06	---	---	---	2.1E-06	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.4E-02	---	---	---	1.3E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	1.6E+04	---	---	---	1.0E-01	7.5E-03	2.4E-04	---	9.4E-04	---	---	---	---
7440-38-2	Arsenic	6.9E+00	---	---	---		3.2E-06	3.3E-07	---	---	4.9E-06	5.3E-07	---	---
7440-39-3	Barium		---	---	---	1.2E-01	---	---	---	1.1E-03	---	---	---	---
7440-41-7	Beryllium	1.6E+00	---	---	---		7.5E-07	2.4E-08	---	---	3.2E-06	1.0E-05	---	---
57-12-5	Cyanide		---	---	---	2.2E-01	---	---	---	2.1E-03	---	---	---	---
7439-89-6	Iron	2.6E+04	---	---	---	2.5E+01	1.2E-02	3.9E-04	---	2.3E-01	---	---	---	---
7439-92-1	Lead		---	---	---	1.5E-03	---	---	---	1.4E-05	---	---	---	---
7439-96-5	Manganese	8.5E+02	---	---	---	3.0E+00	4.0E-04	1.3E-05	---	2.8E-02	---	---	---	---
7440-28-0	Thallium	1.1E+00	---	---	---		5.2E-07	1.7E-08	---	---	---	---	---	---

Pathway Risks			
5.0E-05	1.2E-05	1.1E-07	1.1E-02

Total Cancer Risk	1.1E-02
Reference Cancer Risk	1.0E-06

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN OFF-SITE RESIDENT (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

CAS #	COMPOUND	Exposure Point Concentrations: Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Chronic Average Daily Intake: Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Chronic Hazard Index Estimates (Non-Carcinogenic Risks): Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.1E+00	---	---	---	8.5E-02	---	---	---	2.8E+01
100-41-4	Ethylbenzene		---	---	---	3.7E-01	---	---	---	1.0E-02	---	---	---	1.0E-01
108-88-3	Toluene		---	---	---	9.0E-01	---	---	---	2.5E-02	---	---	---	1.2E-01
	1,2,4-Trimethylbenzene		---	---	---	5.7E-02	---	---	---	1.6E-03	---	---	---	3.1E-02
1330-20-7	Xylenes (Mixed)		---	---	---	3.1E-01	---	---	---	8.5E-03	---	---	---	4.2E-03
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	4.8E-02	---	---	---	1.3E-03	---	---	---	2.2E-02
	1-Methylnaphthalene		---	---	---	1.1E-01	---	---	---	3.0E-03	---	---	---	7.5E-02
91-57-6	2-Methylnaphthalene		---	---	---	1.0E-01	---	---	---	2.7E-03	---	---	---	6.8E-02
91-20-3	Naphthalene		---	---	---	8.0E-01	---	---	---	2.2E-02	---	---	---	5.5E-01
85-01-8	Phenanthrene		---	---	---	3.0E-02	---	---	---	8.2E-04	---	---	---	2.1E-02
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	ND	8.1E-06	---	---	---	---	---	---	---
50-32-8	Benzo(a)pyrene	5.6E+00	---	2.2E-07	2.2E-07	ND	7.6E-06	---	2.2E-07	---	---	---	---	---
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	ND	5.6E-06	---	---	---	---	---	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---		3.2E-06	---	---	---	---	---	---	---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---		2.6E-07	---	---	---	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---		4.4E-06	---	---	---	---	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.2E-02	---	---	---	3.3E-04	---	---	---	8.2E-02
	Inorganic Compounds													
7429-90-5	Aluminum	1.4E+04	---	---	---	4.2E-02	1.9E-02	6.3E-04	---	1.2E-03	1.9E-02	2.3E-03	---	1.2E-03
7440-38-2	Arsenic	6.8E+00	---	---	---		9.3E-06	9.6E-07	---	---	3.1E-02	3.4E-03	---	---
7440-39-3	Barium		---	---	---	9.0E-02	---	---	---	2.5E-03	---	---	---	3.5E-02
7440-41-7	Beryllium	1.2E+00	---	---	---		1.7E-06	5.3E-08	---	---	3.3E-04	1.1E-03	---	---
57-12-5	Cyanide		---	---	---	6.2E-02	---	---	---	1.7E-03	---	---	---	6.5E-02
7439-89-6	Iron	2.4E+04	---	---	---	3.0E+00	3.3E-02	1.0E-03	---	8.2E-02	1.1E-01	3.5E-03	---	2.7E-01
7439-92-1	Lead		---	---	---	1.2E-03	---	---	---	3.3E-05	---	---	---	---
7439-96-5	Manganese	6.1E+02	---	---	---	6.9E-01	8.4E-04	2.7E-05	---	1.9E-02	3.6E-02	2.3E-02	---	8.2E-01
7440-28-0	Thallium	8.8E-01	---	---	---		1.2E-06	3.9E-08	---	---	1.5E-02	4.8E-04	---	---

Total Pathway Risks			
2.1E-01	3.4E-02	0.0E+00	3.1E+01

Total Hazard Index	3.1E+01
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.7E-01	1.1E-02	0.0E+00	3.0E+01
CNS Hazard Index	3.6E-02	2.3E-02	0.0E+00	8.2E-01
Cardiovascular Hazard Index	---	---	0.0E+00	3.5E-02
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	4.2E-03

Total Systemic	3.0E+01
Total CNS	8.8E-01
Total Cardiovascular	3.5E-02
Total Reproductive	4.2E-03

IRIGINAL
IReg/

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN OFF-SITE RESIDENT (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Subsurface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Subsurface Soil Ingestion (mg/kg/day)	Subsurface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Subsurface Soil Oral	Subsurface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	3.1E+00	---	---	---	2.9E-02	---	---	---	8.4E-04
100-41-4	Ethylbenzene		---	---	---	3.7E-01	---	---	---	3.5E-03	---	---	---	---
108-88-3	Toluene		---	---	---	9.0E-01	---	---	---	8.5E-03	---	---	---	---
	1,2,4-Trimethylbenzene		---	---	---	5.7E-02	---	---	---	5.4E-04	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	3.1E-01	---	---	---	2.9E-03	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	4.8E-02	---	---	---	4.5E-04	---	---	---	---
	1-Methylnaphthalene		---	---	---	1.1E-01	---	---	---	1.0E-03	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	1.0E-01	---	---	---	9.4E-04	---	---	---	---
91-20-3	Naphthalene		---	---	---	8.0E-01	---	---	---	7.5E-03	---	---	---	---
85-01-8	Phenanthrene		---	---	---	3.0E-02	---	---	---	2.8E-04	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	5.9E+00	---	---	---	ND	2.8E-06	---	---	---	2.0E-06	---	---	---
50-32-8	Benzo(a)pyrene	5.6E+00	---	2.2E-07	2.2E-07	ND	2.6E-06	---	7.4E-05	---	1.9E-05	---	6.5E-08	---
205-99-2	Benzo(b)fluoranthene	4.1E+00	---	---	---	ND	1.9E-06	---	---	---	1.4E-06	---	---	---
207-08-9	Benzo(k)fluoranthene	2.4E+00	---	---	---		1.1E-06	---	---	---	8.1E-06	---	---	---
53-70-3	Dibenzo(ah)anthracene	1.9E-01	---	---	---		8.9E-08	---	---	---	6.5E-07	---	---	---
193-39-5	Indeno(123-cd)pyrene	3.2E+00	---	---	---		1.5E-06	---	---	---	1.1E-06	---	---	---
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	1.2E-02	---	---	---	1.1E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	1.4E+04	---	---	---	4.2E-02	6.7E-03	2.1E-04	---	3.9E-04	---	---	---	---
7440-38-2	Arsenic	6.8E+00	---	---	---		3.2E-06	3.3E-07	---	---	4.8E-06	5.2E-07	---	---
7440-39-3	Barium		---	---	---	9.0E-02	---	---	---	8.5E-04	---	---	---	---
7440-41-7	Beryllium	1.2E+00	---	---	---		5.7E-07	1.8E-08	---	---	2.4E-06	7.8E-06	---	---
57-12-5	Cyanide		---	---	---	6.2E-02	---	---	---	5.8E-04	---	---	---	---
7439-89-6	Iron	2.4E+04	---	---	---	3.0E+00	1.1E-02	3.6E-04	---	2.8E-02	---	---	---	---
7439-92-1	Lead		---	---	---	1.2E-03	---	---	---	1.1E-05	---	---	---	---
7439-96-5	Manganese	6.1E+02	---	---	---	6.9E-01	2.9E-04	9.2E-06	---	6.5E-03	---	---	---	---
7440-28-0	Thallium	8.8E-01	---	---	---		4.1E-07	1.3E-08	---	---	---	---	---	---

Pathway Risks			
3.2E-06	6.3E-06	6.5E-08	8.4E-04

Total Cancer Risk	8.8E-04
Reference Cancer Risk	1.0E-06

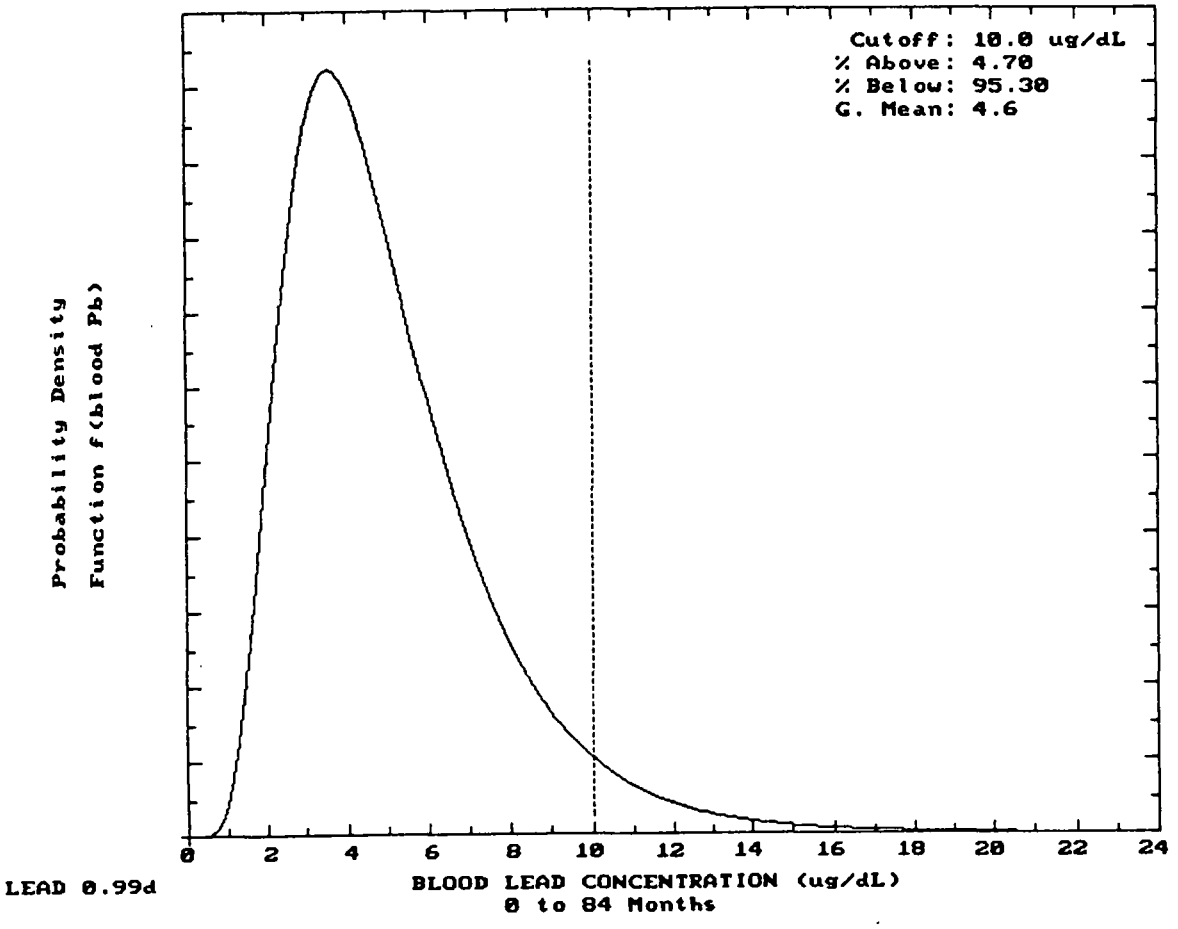
APPENDIX F

IEU/BK Modeling for Lead Exposure

ORIGINAL (Red)

IEUBK Lead Model Results

Future On-Site Resident Child
Average Exposure Point Concentration
UGI Columbia Former MGP Site
Columbia, Pennsylvania



Drinking Water Concentration = 4.3 µg/L
Drinking Water Ingestion Rate = 1 L/day
Soil/Dust Concentration = 141 ug/g
Soil Ingestion Rate = 200 ug/g
All other inputs are EPA Default Values

ORIGINAL (Red)

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.30 ug Pb/L
Other WATER Parameters (non-default):

Age	Water Consumption (L/day)
0-1	1.00
1-2	1.00
2-3	1.00
3-4	1.00
4-5	1.00
5-6	1.00
6-7	1.00

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	141.0	141.0
1-2	141.0	141.0
2-3	141.0	141.0
3-4	141.0	141.0
4-5	141.0	141.0
5-6	141.0	141.0
6-7	141.0	141.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	6.3	11.71	7.39
1-2:	5.3	12.16	7.60
2-3:	4.7	12.67	7.70
3-4:	4.5	12.73	7.80
4-5:	4.2	12.76	7.89
5-6:	4.0	13.03	7.94

6-7:	3.8	13.39	7.97	

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.42	1.88	0.00	0.02
1-2:	2.60	1.93	0.00	0.03
2-3:	2.95	1.96	0.00	0.06
3-4:	2.88	1.98	0.00	0.07
4-5:	2.80	2.00	0.00	0.07
5-6:	2.98	2.02	0.00	0.09
6-7:	3.30	2.03	0.00	0.09

ORIGINAL
(Red)

APPENDIX G

APPENDIX G

Hypothetical On-Site Resident Scenario

ORIGINAL (Red)

APPENDIX G-2
SUMMARY OF RISK ESTIMATES - HYPOTHETICAL ONSITE RESIDENT
UGI Columbia Former MGP Site
Columbia, Pennsylvania

Scenario	Chronic Total Hazard Index	Cancer Total Risk Estimates
Hypothetical On-Site Resident		
(soil and drinking water exposure)		
Child (Central Tendency)	2.30E+02	4.10E-03
Teen/Adult (Central Tendency)	9.30E+01	6.50E-03
Child (RME)	5.80E+02	1.20E-02
Teen/Adult (RME)	2.30E+02	1.90E-02
Risk Criteria	**1**	**1×10^{-6} to 1×10^{-4}**

NC = Not Calculated

APPENDIX G-1
HYPOTHETICAL ON-SITE RESIDENT SCENARIO
EXPOSURE POINT CONCENTRATIONS
UGI Columbia Former MGP Site
Columbia, Pennsylvania

	SURFACE SOIL		FUGITIVE DUST		VAPOR		GROUNDWATER	
COMPOUND	Average Concentration [a] (mg/kg)	Reasonable Maximum Concentration (mg/kg)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/m^3)	Reasonable Maximum Concentration (mg/m^3)	Average Concentration [a] (mg/L)	Reasonable Maximum Concentration (mg/L)
Volatile Organic Compounds								
Benzene	NC	NC	NC	NC	NC	NC	9.3E+00	2.3E+01
Ethylbenzene	NC	NC	NC	NC	NC	NC	2.2E+00	4.7E+00
Tetrachloroethene	NC	NC	NC	NC	NC	NC	5.0E-03	5.0E-03
Toluene	NC	NC	NC	NC	NC	NC	2.9E+00	7.0E+00
Trichloroethene	NC	NC	NC	NC	NC	NC	3.0E-03	3.0E-03
1,2,4-Trimethylbenzene	NC	NC	NC	NC	NC	NC	2.0E-01	4.4E-01
Xylenes (Mixed)	NC	NC	NC	NC	NC	NC	1.8E+00	3.7E+00
Semivolatile Organic Compounds								
Non-Carcinogenic PAHs								
Acenaphthene	NC	NC	NC	NC	NC	NC	2.1E-01	7.5E-01
Acenaphthylene	[c]	[c]	[c]	[c]	[c]	[c]	[c]	[c]
Benzo(ghi)perylene	[c]	[c]	[c]	[c]	[c]	[c]	[c]	[c]
Fluoranthene	NC	NC	NC	NC	NC	NC	7.3E-02	2.8E-01
1-Methylnaphthalene	NC	NC	NC	NC	NC	NC	2.2E-01	4.7E-01
2-Methylnaphthalene	NC	NC	NC	NC	NC	NC	7.7E-01	2.6E+00
Naphthalene	NC	NC	NC	NC	NC	NC	4.4E+00	8.1E+00
Phenanthrene	NC	NC	1.3E-09	3.3E-09	NC	NC	2.6E-01	1.2E+00
Pyrene	NC	NC	NC	NC	NC	NC	1.6E-01	7.2E-01
Carcinogenic PAHs [d]								
Benzo(a)anthracene	2.4E+00	4.6E+00	NC	NC	NC	NC	5.5E-02	1.9E-01
Benzo(a)pyrene	3.2E+00	6.2E+00	3.1E-09	6.0E-09	4.1E-07	1.4E-06	4.7E-02	1.5E-01
Benzo(b)fluoranthene	2.5E+00	6.7E+00	NC	NC	NC	NC	4.3E-02	1.3E-01
Benzo(k)fluoranthene	NC	NC	NC	NC	NC	NC	NC	NC
Chrysene	NC	NC	NC	NC	NC	NC	4.5E-02	1.4E-01
Dibenzo(ah)anthracene	1.3E-01	NC	NC	NC	NC	NC	NC	NC
Indeno(123-cd)pyrene	2.5E+00	5.9E+00	NC	NC	NC	NC	NC	NC
Phthalates								
bis(2-Ethylhexyl)phthalate	NC	NC	NC	NC	NC	NC	6.9E-02	6.9E-02
Other SVOCs								
Dibenzofuran	NC	NC	NC	NC	NC	NC	2.6E-02	8.1E-02
Inorganic Compounds								
Aluminum	5.2E+03	7.9E+03	5.0E-06	7.6E-06	NC	NC	1.9E+00	9.6E+00
Arsenic	5.6E+00	1.0E+01	5.3E-09	9.9E-09	NC	NC	NC	NC
Barium	NC	NC	NC	NC	NC	NC	1.7E-01	1.8E-01
Beryllium	2.7E-01	5.4E-01	2.5E-10	5.2E-10	NC	NC	NC	NC
Cadmium	1.8E+00	5.8E+00	1.7E-09	5.6E-09	NC	NC	NC	NC
Cyanide [b]	NC	NC	NC	NC	NC	NC	3.8E-02	1.4E-01
Iron	1.9E+04	4.2E+04	1.8E-05	4.0E-05	NC	NC	1.4E+01	4.2E+01
Lead	1.4E+02	6.3E+02	1.4E-07	6.1E-07	NC	NC	4.3E-03	1.9E-02
Manganese	3.3E+02	8.1E+02	3.2E-07	7.7E-07	NC	NC	4.2E-01	1.4E+00
Thallium	NC	NC	NC	NC	NC	NC	NC	NC

NC = Not a Contaminant of Potential Concern for exposure medium

[a] Where the reasonable maximum is "NC", the average concentration is one-half the method detection limit

[b] Free cyanide concentrations are estimated to be 15% of the measured total cyanide concentration

[c] Evaluated qualitatively

[d] EPA recommends that inhalation risk to carcinogenic PAHs be evaluated for benzo(a)pyrene only

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD ONSITE RESIDENT (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	2.3E+01	---	---	---	1.6E+00	---	---	---	5.3E+02
100-41-4	Ethylbenzene		---	---	---	4.7E+00	---	---	---	3.2E-01	---	---	---	3.2E+00
127-18-4	Tetrachloroethene		---	---	---	5.0E-03	---	---	---	3.4E-04	---	---	---	3.4E-02
108-88-3	Toluene		---	---	---	7.0E+00	---	---	---	4.8E-01	---	---	---	2.4E+00
79-01-6	Trichloroethene		---	---	---	3.0E-03	---	---	---	2.1E-04	---	---	---	3.4E-02
	1,2,4-Trimethylbenzene		---	---	---	4.4E-01	---	---	---	3.0E-02	---	---	---	6.0E-01
1330-20-7	Xylenes (Mixed)		---	---	---	3.7E+00	---	---	---	2.5E-01	---	---	---	1.3E-01
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	7.5E-01	---	---	---	5.1E-02	---	---	---	8.6E-01
206-44-0	Fluoranthene		---	---	---	2.8E-01	---	---	---	1.9E-02	---	---	---	4.6E-01
	1-Methylnaphthalene		---	---	---	4.7E-01	---	---	---	3.2E-02	---	---	---	8.0E-01
91-57-6	2-Methylnaphthalene		---	---	---	2.8E+00	---	---	---	1.8E-01	---	---	---	4.5E+00
91-20-3	Naphthalene		---	---	---	8.1E+00	---	---	---	5.5E-01	---	---	---	1.4E+01
85-01-8	Phenanthrene		---	---	---	1.2E+00	---	---	---	8.2E-02	---	---	---	2.1E+00
129-00-0	Pyrene		---	---	---	7.2E-01	---	---	---	4.9E-02	---	---	---	1.8E+00
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	1.9E-01	8.3E-05	---	---	1.3E-02	---	---	---	---
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	1.5E-01	8.5E-05	---	1.4E-06	1.0E-02	---	---	---	---
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.3E-01	9.2E-05	---	---	8.9E-03	---	---	---	---
218-01-9	Chrysene		---	---	---	1.4E-01	---	---	---	9.6E-03	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---		8.1E-05	---	---	---	---	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate		---	---	---	6.9E-02	---	---	---	4.7E-03	---	---	---	2.4E-01
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	8.1E-02	---	---	---	5.5E-03	---	---	---	1.4E+00
	Inorganic Compounds													
7429-90-5	Aluminum	7.8E+03	7.8E-06	---	7.8E-06	9.6E+00	1.1E-01	8.9E-04	7.3E-06	6.6E-01	1.1E-01	3.3E-03	2.1E-03	6.6E-01
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09		1.4E-04	3.7E-06	9.5E-09	---	4.7E-01	1.3E-02	---	---
7440-39-3	Barium		---	---	---	1.6E-01	---	---	---	1.2E-02	---	---	---	1.6E-01
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10		7.4E-06	6.1E-06	5.0E-10	---	1.5E-03	1.2E-03	---	---
7440-43-9	Cadmium (Food)	5.6E+00	5.6E-09	---	5.6E-09		7.9E-05	6.5E-07	5.3E-09	---	7.9E-02	2.6E-02	2.7E-05	---
57-12-5	Cyanide		---	---	---	1.4E-01	---	---	---	9.6E-03	---	---	---	4.6E-01
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	4.2E+01	5.7E-01	4.7E-03	---	2.9E+00	1.9E+00	1.6E-02	---	9.5E+00
7439-92-1	Lead	8.3E+02	8.1E-07	---	8.1E-07	1.9E-02	8.7E-03	7.1E-05	5.6E-07	1.3E-03	---	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	1.4E+00	1.1E-02	9.1E-05	7.4E-07	9.6E-02	4.8E-01	7.9E-02	1.5E-02	4.2E+00

Total Pathway Risks			
3.0E+00	1.4E-01	4.7E-02	6.7E+02

Total Hazard [illegible]	5.6E+02
[illegible] Hazard Index	1.00 (Default)

Systemic Hazard Index	2.6E+00	8.9E-02	2.1E-03	5.7E+02
CNS Hazard Index	4.8E-01	7.9E-02	1.5E-02	4.2E+00
Cardiovascular Hazard Index	0.0E+00	0.0E+00	0.0E+00	1.8E-01
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	3.6E-01

Total Systemic	5.7E+02
Total CNS	4.7E+00
Total Cardiovascular	1.8E-01
Total Reproductive	3.6E-01

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD ONSITE RESIDENT (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	2.3E+01	---	---	---	1.4E-01	---	---	---	3.9E-03
100-41-4	Ethylbenzene		---	---	---	4.7E+00	---	---	---	2.8E-02	---	---	---	---
127-18-4	Tetrachloroethene		---	---	---	5.0E-03	---	---	---	2.9E-05	---	---	---	1.5E-06
108-88-3	Toluene		---	---	---	7.0E+00	---	---	---	4.1E-02	---	---	---	---
79-01-6	Trichloroethene		---	---	---	3.0E-03	---	---	---	1.8E-05	---	---	---	1.9E-07
	1,2,4-Trimethylbenzene		---	---	---	4.4E-01	---	---	---	2.6E-03	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	3.7E+00	---	---	---	2.2E-02	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	7.5E-01	---	---	---	4.4E-03	---	---	---	---
206-44-0	Fluoranthene		---	---	---	2.8E-01	---	---	---	1.6E-03	---	---	---	---
	1-Methylnaphthalene		---	---	---	4.7E-01	---	---	---	2.8E-03	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	2.6E+00	---	---	---	1.5E-02	---	---	---	---
91-20-3	Naphthalene		---	---	---	8.1E+00	---	---	---	4.8E-02	---	---	---	---
85-01-8	Phenanthrene		---	---	---	1.2E+00	---	---	---	7.0E-03	---	---	---	---
119-00-0	Pyrene		---	---	---	7.2E-01	---	---	---	4.2E-03	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	1.9E-01	5.4E-06	---	---	1.1E-03	3.9E-06	---	---	8.1E-04
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	1.5E-01	7.3E-06	---	1.2E-04	8.8E-04	5.3E-05	---	1.0E-07	6.4E-03
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.3E-01	7.9E-06	---	---	7.6E-04	5.7E-06	---	---	5 6E-04
218-01-9	Chrysene		---	---	---	1.4E-01	---	---	---	8.2E-04	---	---	---	6.0E-06
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---		6.9E-06	---	---	---	5.1E-06	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate		---	---	---	6.9E-02	---	---	---	4.1E-04	---	---	---	5 7E-06
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	8.1E-02	---	---	---	4.8E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	7.9E+03	7.6E-06	---	7.6E-06	9.6E+00	9.3E-03	7.6E-05	6.2E-04	5.6E-02	---	---	---	---
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09		1.2E-05	3.2E-07	8.1E-07	---	1 8E-05	5.0E-07	3.5E-09	---
7440-39-3	Barium		---	---	---	1.8E-01	---	---	---	1.1E-03	---	---	---	---
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10		6.3E-07	5.2E-09	4.3E-08	---	2.7E-06	2.2E-06	1.0E-10	---
7440-43-9	Cadmium (Food)	5.8E+00	5.6E-09	---	5.6E-09		6.8E-06	5.6E-08	4.6E-07	---	---	---	8.2E-10	---
57-12-5	Cyanide		---	---	---	1.4E-01	---	---	---	8.2E-04	---	---	---	---
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	4.2E+01	4 9E-02	4.0E-04	3.3E-03	2.4E-01	---	---	---	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	1.9E-02	7.4E-04	6.1E-06	5.0E-05	1.1E-04	---	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	1.4E+00	9.5E-04	7.8E-06	6.4E-05	8.2E-03	---	---	---	---

Pathway Risks			
8.9E-05	2.7E-06	1.1E-07	1.2E-02

Total Cancer Risk	1.2E-02
Reference Cancer Risk	1.0E-06

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN ONSITE RESIDENT (RME Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene	---	---	---	---	2.3E+01	---	---	---	6.3E-01	---	---	---	2.1E+02
100-41-4	Ethylbenzene	---	---	---	---	4.7E+00	---	---	---	1.3E-01	---	---	---	1.3E+00
127-18-4	Tetrachloroethene	---	---	---	---	5.0E-03	---	---	---	1.4E-04	---	---	---	1.4E-02
108-88-3	Toluene	---	---	---	---	7.0E+00	---	---	---	1.9E-01	---	---	---	9.8E-01
79-01-6	Trichloroethene	---	---	---	---	3.0E-03	---	---	---	8.2E-05	---	---	---	1.4E-02
	1,2,4-Trimethylbenzene	---	---	---	---	4.4E-01	---	---	---	1.2E-02	---	---	---	2.4E-01
1330-20-7	Xylenes (Mixed)	---	---	---	---	3.7E+00	---	---	---	1.0E-01	---	---	---	5.1E-02
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene	---	---	---	---	7.5E-01	---	---	---	2.1E-02	---	---	---	3.4E-01
206-44-0	Fluoranthene	---	---	---	---	2.8E-01	---	---	---	7.7E-03	---	---	---	1.9E-01
	1-Methylnaphthalene	---	---	---	---	4.7E-01	---	---	---	1.3E-02	---	---	---	3.2E-01
91-57-6	2-Methylnaphthalene	---	---	---	---	2.6E+00	---	---	---	7.1E-02	---	---	---	1.8E+00
91-20-3	Naphthalene	---	---	---	---	8.1E+00	---	---	---	2.2E-01	---	---	---	5.5E+00
85-01-8	Phenanthrene	---	---	---	---	1.2E+00	---	---	---	3.3E-02	---	---	---	8.2E-01
129-00-0	Pyrene	---	---	---	---	7.2E-01	---	---	---	2.0E-02	---	---	---	6.6E-01
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	1.9E-01	6.3E-06	---	---	5.2E-03	---	---	---	---
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	1.5E-01	8.5E-06	---	1.4E-06	4.1E-03	---	---	---	---
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.3E-01	9.2E-06	---	---	3.6E-03	---	---	---	---
218-01-9	Chrysene	---	---	---	---	1.4E-01	---	---	---	3.8E-03	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---		8.1E-06	---	---	---	---	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate	0.0E+00	0.0E+00	0.0E+00	0.0E+00	6.9E-02	0.0E+00	0.0E+00	0.0E+00	1.9E-03	0.0E+00	0.0E+00	---	9.5E-02
	Other SVOCs													
132-64-9	Dibenzofuran	---	---	---	---	8.1E-02	---	---	---	2.2E-03	---	---	---	5.5E-01
	Inorganic Compounds													
7429-90-5	Aluminum	7.9E+03	7.6E-06	---	7.6E-06	9.8E+00	1.1E-02	3.5E-04	7.3E-06	2.6E-01	1.1E-02	1.3E-03	2.1E-03	2.6E-01
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09		1.4E-05	1.5E-06	9.5E-09	---	4.7E-02	5.1E-03	---	---
7440-39-3	Barium	---	---	---	---	1.8E-01	---	---	---	4.9E-03	---	---	---	7.0E-02
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10		7.4E-07	2.4E-08	5.0E-10	---	1.5E-04	4.6E-04	---	---
7440-43-9	Cadmium (Food)	5.6E+00	5.6E-09	---	5.6E-09		7.9E-06	2.6E-07	5.3E-09	---	7.9E-03	1.0E-02	2.7E-05	---
57-12-5	Cyanide	---	---	---	---	1.4E-01	---	---	---	3.8E-03	---	---	---	1.9E-01
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	4.2E+01	5.7E-02	1.8E-03	---	1.1E+00	1.9E-01	6.1E-03	---	3.8E+00
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	1.9E-02	8.7E-04	2.8E-05	5.8E-07	5.2E-04	---	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	1.4E+00	1.1E-03	3.6E-05	7.4E-07	3.8E-02	4.6E-02	3.1E-02	1.5E-02	1.7E+00

Total Pathway Risks			
3.0E-01	6.4E-02	1.7E-02	2.3E+02

Total Hazard Index	2.3E+02
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	2.8E-01	2.3E-02	2.1E-03	2.3E+02
CNS Hazard Index	4.6E-02	3.1E-02	1.5E-02	1.7E+00
Cardiovascular Hazard Index	0.0E+00	0.0E+00	0.0E+00	7.0E-02
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	1.5E-01

Total Systemic	2.3E+02
Total CNS	1.8E+00
Total Cardiovascular	7.0E-02
Total Reproductive	1.5E-01

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN ONSITE RESIDENT (RME Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene	---	---	---	---	2.3E+01	---	---	---	2.2E-01	---	---	---	6.2E-03
100-41-4	Ethylbenzene	---	---	---	---	4.7E+00	---	---	---	4.4E-02	---	---	---	---
127-18-4	Tetrachloroethene	---	---	---	---	5.0E-03	---	---	---	4.7E-05	---	---	---	2.4E-06
108-88-3	Toluene	---	---	---	---	7.0E+00	---	---	---	6.6E-02	---	---	---	---
79-01-6	Trichloroethene	---	---	---	---	3.0E-03	---	---	---	2.8E-05	---	---	---	3.1E-07
	1,2,4-Trimethylbenzene	---	---	---	---	4.4E-01	---	---	---	4.1E-03	---	---	---	---
1330-20-7	Xylenes (Mixed)	---	---	---	---	3.7E+00	---	---	---	3.5E-02	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene	---	---	---	---	7.5E-01	---	---	---	7.0E-03	---	---	---	---
206-44-0	Fluoranthene	---	---	---	---	2.8E-01	---	---	---	2.6E-03	---	---	---	---
	1-Methylnaphthalene	---	---	---	---	4.7E-01	---	---	---	4.4E-03	---	---	---	---
91-57-6	2-Methylnaphthalene	---	---	---	---	2.6E+00	---	---	---	2.4E-02	---	---	---	---
91-20-3	Naphthalene	---	---	---	---	8.1E+00	---	---	---	7.6E-02	---	---	---	---
85-01-8	Phenanthrene	---	---	---	---	1.2E+00	---	---	---	1.1E-02	---	---	---	---
119-00-0	Pyrene	---	---	---	---	7.2E-01	---	---	---	6.8E-03	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	4.6E+00	---	---	---	1.9E-01	2.2E-06	---	---	1.8E-03	1.6E-06	---	---	1.3E-03
50-32-8	Benzo(a)pyrene	6.2E+00	6.0E-09	1.4E-06	1.4E-06	1.5E-01	2.9E-06	---	4.7E-04	1.4E-03	2.1E-05	---	4.1E-07	1.0E-02
205-99-2	Benzo(b)fluoranthene	6.7E+00	---	---	---	1.3E-01	3.1E-06	---	---	1.2E-03	2.3E-06	---	---	8.9E-04
218-01-9	Chrysene	---	---	---	---	1.4E-01	---	---	---	1.3E-03	---	---	---	9.6E-06
193-39-5	Indeno(123-cd)pyrene	5.9E+00	---	---	---		2.8E-06	---	---	---	2.0E-06	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate	0.0E+00	0.0E+00	0.0E+00	0.0E+00	6.9E-02	0.0E+00	0.0E+00	0.0E+00	6.5E-04	0.0E+00	0.0E+00	0.0E+00	9.1E-06
	Other SVOCs													
132-64-9	Dibenzofuran	---	---	---	---	8.1E-02	---	---	---	7.6E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	7.9E+03	7.6E-06	---	7.6E-06	9.6E+00	3.7E-03	1.2E-04	2.5E-03	9.0E-02	---	---	---	---
7440-38-2	Arsenic	1.0E+01	9.9E-09	---	9.9E-09		4.8E-06	5.0E-07	3.3E-06	---	7.3E-06	7.9E-07	1.4E-08	---
7440-39-3	Barium	---	---	---	---	1.8E-01	---	---	---	1.7E-03	---	---	---	---
7440-41-7	Beryllium	5.4E-01	5.2E-10	---	5.2E-10		2.5E-07	8.2E-09	1.7E-07	---	1.1E-06	3.5E-06	4.1E-10	---
7440-43-9	Cadmium (Food)	5.6E+00	5.6E-09	---	5.6E-09		2.7E-06	8.8E-08	1.8E-06	---	---	---	3.3E-09	---
57-12-5	Cyanide	---	---	---	---	1.4E-01	---	---	---	1.3E-03	---	---	---	---
7439-89-6	Iron	4.2E+04	4.0E-05	---	4.0E-05	4.2E+01	2.0E-02	6.3E-04	1.3E-02	3.9E-01	---	---	---	---
7439-92-1	Lead	6.3E+02	6.1E-07	---	6.1E-07	1.9E-02	3.0E-04	9.6E-06	2.0E-04	1.8E-04	---	---	---	---
7439-96-5	Manganese	8.1E+02	7.7E-07	---	7.7E-07	1.4E+00	3.8E-04	1.2E-05	2.5E-04	1.3E-02	---	---	---	---

Pathway Risks			
3.6E-05	4.3E-06	4.3E-07	1.9E-02

Total Cancer Risk	1.9E-02
Reference Cancer Risk	1.0E-06

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD ONSITE RESIDENT (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	9.3E+00	---	---	---	6.4E-01	---	---	---	2.1E+02
100-41-4	Ethylbenzene		---	---	---	2.2E+00	---	---	---	1.5E-01	---	---	---	1.5E+00
127-18-4	Tetrachloroethene		---	---	---	5.0E-03	---	---	---	3.4E-04	---	---	---	3.4E-02
108-88-3	Toluene		---	---	---	2.9E+00	---	---	---	2.0E-01	---	---	---	9.9E-01
79-01-6	Trichloroethene		---	---	---	3.0E-03	---	---	---	2.1E-04	---	---	---	3.4E-02
	1,2,4-Trimethylbenzene		---	---	---	2.0E-01	---	---	---	1.4E-02	---	---	---	2.8E-01
1330-20-7	Xylenes (Mixed)		---	---	---	1.8E+00	---	---	---	1.2E-01	---	---	---	6.0E-02
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	2.1E-01	---	---	---	1.4E-02	---	---	---	2.4E-01
206-44-0	Fluoranthene		---	---	---	7.3E-02	---	---	---	5.0E-03	---	---	---	1.3E-01
	1-Methylnaphthalene		---	---	---	2.2E-01	---	---	---	1.5E-02	---	---	---	3.8E-01
91-57-6	2-Methylnaphthalene		---	---	---	7.7E-01	---	---	---	5.3E-02	---	---	---	1.3E+00
91-20-3	Naphthalene		---	---	---	4.4E+00	---	---	---	3.0E-01	---	---	---	7.5E+00
85-01-8	Phenanthrene		---	---	---	2.6E-01	---	---	---	1.8E-02	---	---	---	4.5E-01
129-00-0	Pyrene		---	---	---	1.6E-01	---	---	---	1.1E-02	---	---	---	3.7E-01
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	2.4E+00	---	---	---	5.5E-02	3.2E-05	---	---	3.8E-03	---	---	---	---
50-32-8	Benzo(a)pyrene	3.3E+00	3.1E-09	4.1E-07	4.1E-07	4.7E-02	4.5E-05	---	4.0E-07	3.2E-03	---	---	---	---
205-99-2	Benzo(b)fluoranthene	2.5E+00	---	---	---	4.3E-02	3.4E-05	---	---	2.9E-03	---	---	---	---
218-01-9	Chrysene		---	---	---	4.5E-02	---	---	---	3.1E-03	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	2.5E+00	---	---	---		3.5E-05	---	---	---	---	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate		---	---	---	6.9E-02	---	---	---	4.7E-03	---	---	---	2.4E-01
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	2.6E-02	---	---	---	1.8E-03	---	---	---	4.5E-01
	Inorganic Compounds													
7429-90-5	Aluminum	5.2E+03	5.0E-06	---	5.0E-06	1.9E+00	7.1E-02	5.6E-04	4.8E-06	1.3E-01	7.1E-02	2.2E-03	1.4E-03	1.3E-01
7440-38-2	Arsenic	5.6E+00	5.3E-09	---	5.3E-09		7.6E-05	2.0E-06	5.1E-09	---	2.5E-01	7.0E-03	---	---
7440-39-3	Barium		---	---	---	1.7E-01	---	---	---	1.2E-02	---	---	---	1.7E-01
7440-41-7	Beryllium	2.7E-01	2.5E-10	---	2.5E-10		3.6E-06	3.0E-08	2.4E-10	---	7.3E-04	5.9E-04	---	---
7440-43-9	Cadmium (Food)	1.6E+00	1.7E-09	---	1.7E-09		2.5E-05	2.0E-07	1.7E-09	---	2.5E-02	8.1E-03	8.3E-06	---
57-12-5	Cyanide		---	---	---	3.8E-02	---	---	---	2.6E-03	---	---	---	1.3E-01
7439-89-6	Iron	1.9E+04	1.8E-05	---	1.8E-05	1.4E+01	2.5E-01	2.1E-03	---	9.6E-01	8.5E-01	6.9E-03	---	3.2E+00
7439-92-1	Lead	1.4E+02	1.4E-07	---	1.4E-07	4.3E-03	1.9E-03	1.6E-06	1.3E-07	2.9E-04	---	---	---	---
7439-96-5	Manganese	3.3E+02	3.2E-07	---	3.2E-07	4.2E-01	4.6E-03	3.7E-06	3.1E-07	2.9E-02	2.0E-01	3.3E-02	6.1E-03	1.3E+00

Total Pathway Risks			
1.4E+00	5.7E-02	7.5E-03	2.3E+02

Total Hazard Index	2.3E+02
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.2E+00	2.5E-02	1.4E-03	2.3E+02
CNS Hazard Index	2.0E-01	3.3E-02	6.1E-03	1.3E+00
Cardiovascular Hazard Index	0.0E+00	0.0E+00	0.0E+00	1.7E-01
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	3.0E-01

Total Systemic	2.3E+02
Total CNS	1.5E+00
Total Cardiovascular	1.7E-01
Total Reproductive	3.0E-01

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL CHILD ONSITE RESIDENT (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	9.3E+00	---	---	---	5.5E-02	---	---	---	1.6E-03
100-41-4	Ethylbenzene		---	---	---	2.2E+00	---	---	---	1.3E-02	---	---	---	---
127-18-4	Tetrachloroethene		---	---	---	5.0E-03	---	---	---	2.9E-05	---	---	---	1.5E-06
108-88-3	Toluene		---	---	---	2.9E+00	---	---	---	1.7E-02	---	---	---	---
79-01-6	Trichloroethene		---	---	---	3.0E-03	---	---	---	1.8E-05	---	---	---	1.9E-07
	1,2,4-Trimethylbenzene		---	---	---	2.0E-01	---	---	---	1.2E-03	---	---	---	---
1330-20-7	Xylenes (Mixed)		---	---	---	1.8E+00	---	---	---	1.0E-02	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	2.1E-01	---	---	---	1.2E-03	---	---	---	---
206-44-0	Fluoranthene		---	---	---	7.3E-02	---	---	---	4.3E-04	---	---	---	---
	1-Methylnaphthalene		---	---	---	2.2E-01	---	---	---	1.3E-03	---	---	---	---
91-57-6	2-Methylnaphthalene		---	---	---	7.7E-01	---	---	---	4.5E-03	---	---	---	---
91-20-3	Naphthalene		---	---	---	4.4E+00	---	---	---	2.6E-02	---	---	---	---
85-01-8	Phenanthrene		---	---	---	2.6E-01	---	---	---	1.5E-03	---	---	---	---
119-00-0	Pyrene		---	---	---	1.6E-01	---	---	---	9.5E-04	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	2.4E+00	---	---	---	5.5E-02	2.8E-06	---	---	3.2E-04	2.0E-06	---	---	2.4E-04
50-32-8	Benzo(a)pyrene	3.3E+00	3.1E-09	4.1E-07	4.1E-07	4.7E-02	3.8E-06	---	3.4E-05	2.8E-04	2.8E-05	---	3.0E-08	2.0E-03
205-99-2	Benzo(b)fluoranthene	2.5E+00	---	---	---	4.3E-02	2.9E-06	---	---	2.5E-04	2.1E-06	---	---	1.8E-04
218-01-9	Chrysene		---	---	---	4.5E-02	---	---	---	2.6E-04	---	---	---	1.9E-06
193-39-5	Indeno(123-cd)pyrene	2.5E+00	---	---	---		3.0E-06	---	---	---	2.2E-06	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate		---	---	---	6.9E-02	---	---	---	4.1E-04	---	---	---	5.7E-06
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	2.6E-02	---	---	---	1.5E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	5.2E+03	5.0E-06	---	5.0E-06	1.9E+00	6.1E-03	5.0E-05	4.1E-04	1.1E-02	---	---	---	---
7440-38-2	Arsenic	5.6E+00	5.3E-09	---	5.3E-09		6.5E-06	1.7E-07	4.4E-07	---	9.8E-06	2.7E-07	1.9E-09	---
7440-39-3	Barium		---	---	---	1.7E-01	---	---	---	1.0E-03	---	---	---	---
7440-41-7	Beryllium	2.7E-01	2.5E-10	---	2.5E-10		3.1E-07	2.5E-09	2.1E-08	---	1.3E-06	1.1E-06	5.0E-11	---
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09		2.1E-06	1.7E-08	1.4E-07	---	---	---	2.6E-10	---
57-12-5	Cyanide		---	---	---	3.8E-02	---	---	---	2.2E-04	---	---	---	---
7439-89-6	Iron	1.9E+04	1.8E-05	---	1.8E-05	1.4E+01	2.2E-02	1.8E-04	1.5E-03	8.2E-02	---	---	---	---
7439-92-1	Lead	1.4E+02	1.4E-07	---	1.4E-07	4.3E-03	1.7E-04	1.4E-06	1.1E-05	2.5E-05	---	---	---	---
7439-96-5	Manganese	3.3E+02	3.2E-07	---	3.2E-07	4.2E-01	3.9E-04	3.2E-06	2.6E-05	2.5E-03	---	---	---	---

Pathway Risks			
4.5E-05	1.4E-06	3.2E-08	4.0E-03

Total Cancer Risk	4.1E-03
Reference Cancer Risk	1.0E-06

ORIGINAL Read

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN ONSITE RESIDENT (CTE Case)
CHRONIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Chronic Average Daily Intake				Chronic Hazard Index Estimates (Non-Carcinogenic Risks)			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/l)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene		---	---	---	9.3E+00	---	---	---	2.5E-01	---	---	---	8.5E+01
100-41-4	Ethylbenzene		---	---	---	2.2E+00	---	---	---	6.0E-02	---	---	---	6.0E-01
127-18-4	Tetrachloroethene		---	---	---	5.0E-03	---	---	---	1.4E-04	---	---	---	1.4E-02
108-88-3	Toluene		---	---	---	2.9E+00	---	---	---	7.9E-02	---	---	---	4.0E-01
79-01-6	Trichloroethene		---	---	---	3.0E-03	---	---	---	8.2E-05	---	---	---	1.4E-02
	1,2,4-Trimethylbenzene		---	---	---	2.0E-01	---	---	---	5.5E-03	---	---	---	1.1E-01
1330-20-7	Xylenes (Mixed)		---	---	---	1.6E+00	---	---	---	4.6E-02	---	---	---	2.4E-02
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene		---	---	---	2.1E-01	---	---	---	5.8E-03	---	---	---	9.6E-02
208-44-0	Fluoranthene		---	---	---	7.3E-02	---	---	---	2.0E-03	---	---	---	5.0E-02
	1-Methylnaphthalene		---	---	---	2.2E-01	---	---	---	6.0E-03	---	---	---	1.5E-01
91-57-6	2-Methylnaphthalene		---	---	---	7.7E-01	---	---	---	2.1E-02	---	---	---	5.3E-01
91-20-3	Naphthalene		---	---	---	4.4E+00	---	---	---	1.2E-01	---	---	---	3.0E+00
85-01-8	Phenanthrene		---	---	---	2.6E-01	---	---	---	7.2E-03	---	---	---	1.8E-01
129-00-0	Pyrene		---	---	---	1.6E-01	---	---	---	4.4E-03	---	---	---	1.5E-01
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	2.4E+00	---	---	---	5.5E-02	3.2E-06	---	---	1.5E-03	---	---	---	---
50-32-8	Benzo(a)pyrene	3.3E+00	3.1E-09	4.1E-07	4.1E-07	4.7E-02	4.5E-06	---	4.0E-07	1.3E-03	---	---	---	---
205-99-2	Benzo(b)fluoranthene	2.5E+00	---	---	---	4.3E-02	3.4E-06	---	---	1.2E-03	---	---	---	---
218-01-9	Chrysene		---	---	---	4.5E-02	---	---	---	1.2E-03	---	---	---	---
193-39-5	Indeno(123-cd)pyrene	2.5E+00	---	---	---		3.5E-06	---	---	---	---	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate		---	NA	NA	6.9E-02	0.0E+00	0.0E+00	---	1.9E-03	0.0E+00	0.0E+00	---	9.5E-02
	Other SVOCs													
132-64-9	Dibenzofuran		---	---	---	2.6E-02	---	---	---	7.1E-04	---	---	---	1.8E-01
	Inorganic Compounds													
7429-90-5	Aluminum	5.2E+03	5.0E-06	---	5.0E-06	1.9E+00	7.1E-03	2.3E-04	4.8E-06	5.2E-02	7.1E-03	8.5E-04	1.4E-03	5.2E-02
7440-38-2	Arsenic	5.6E+00	5.3E-09	---	5.3E-09		7.6E-06	7.9E-07	5.1E-09	---	2.5E-02	2.8E-03	---	---
7440-39-3	Barium		---	---	---	1.7E-01	---	---	---	4.7E-03	---	---	---	6.7E-02
7440-41-7	Beryllium	2.7E-01	2.5E-10	---	2.5E-10		3.6E-07	1.2E-06	2.4E-10	---	7.3E-05	2.3E-04	---	---
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09		2.5E-06	7.9E-06	1.7E-09	---	2.5E-03	3.2E-03	8.3E-06	---
57-12-5	Cyanide		---	---	---	3.8E-02	---	---	---	1.0E-03	---	---	---	5.2E-02
7439-89-6	Iron	1.9E+04	1.8E-05	---	1.8E-05	1.4E+01	2.5E-02	8.2E-04	---	3.8E-01	8.5E-02	2.7E-03	---	1.3E+00
7439-92-1	Lead	1.4E+02	1.4E-07	---	1.4E-07	4.3E-03	1.9E-04	6.2E-06	1.3E-07	1.2E-04	---	---	---	---
7439-96-5	Manganese	3.3E+02	3.2E-07	---	3.2E-07	4.2E-01	4.6E-04	1.5E-05	3.1E-07	1.2E-02	2.0E-02	1.3E-02	6.1E-03	5.0E-01

	Total Pathway Risks		
1.4E-01	2.3E-02	7.5E-03	9.2E+01

Total Hazard Index	9.3E+01
Reference Hazard Index	1.00 (Default)

Systemic Hazard Index	1.2E-01	9.7E-03	1.4E-03	9.2E+01
CNS Hazard Index	2.0E-02	1.3E-02	6.1E-03	5.0E-01
Cardiovascular Hazard Index	0.0E+00	0.0E+00	0.0E+00	6.7E-02
Reproductive Hazard Index	0.0E+00	0.0E+00	0.0E+00	1.2E-01

Total Systemic	9.2E+01
Total CNS	5.4E-01
Total Cardiovascular	6.7E-02
Total Reproductive	1.2E-01

ORIGINAL (Red)

CALCULATION OF RISK ESTIMATES - HYPOTHETICAL TEEN ONSITE RESIDENT (CTE Case)
CARCINOGENIC RISK
UGI Columbia Former MGP Site
Columbia, Pennsylvania

		Exposure Point Concentrations					Average Daily Intake (Lifetime) Estimates				Carcinogenic Risk Estimates			
CAS #	COMPOUND	Surface Soil (mg/kg)	Air (Dust) (mg/m3)	Air (Vapor) (mg/m3)	Air (Total) (mg/m3)	Drinking Water (mg/L)	Surface Soil Ingestion (mg/kg/day)	Surface Soil Dermal (mg/kg/day)	Air (Total) Inhalation (mg/m3)	Drinking Water Ingestion (mg/kg/day)	Surface Soil Oral	Surface Soil Dermal	Air Inhalation	Drinking Water Oral
	Volatile Organic Compounds													
71-43-2	Benzene	---	---	---	---	9.3E+00	---	---	---	8.7E-02	---	---	---	2.5E-03
100-41-4	Ethylbenzene	---	---	---	---	2.2E+00	---	---	---	2.1E-02	---	---	---	---
127-18-4	Tetrachloroethene	---	---	---	---	5.0E-03	---	---	---	4.7E-05	---	---	---	2.4E-06
108-88-3	Toluene	---	---	---	---	2.9E+00	---	---	---	2.7E-02	---	---	---	---
79-01-6	Trichloroethene	---	---	---	---	3.0E-03	---	---	---	2.8E-05	---	---	---	3.1E-07
	1,2,4-Trimethylbenzene	---	---	---	---	2.0E-01	---	---	---	1.9E-03	---	---	---	---
1330-20-7	Xylenes (Mixed)	---	---	---	---	1.8E+00	---	---	---	1.6E-02	---	---	---	---
	Semivolatile Organic Compounds													
	Non-Carcinogenic PAHs													
83-32-9	Acenaphthene	---	---	---	---	2.1E-01	---	---	---	2.0E-03	---	---	---	---
206-44-0	Fluoranthene	---	---	---	---	7.3E-02	---	---	---	6.9E-04	---	---	---	---
	1-Methylnaphthalene	---	---	---	---	2.2E-01	---	---	---	2.1E-03	---	---	---	---
91-57-6	2-Methylnaphthalene	---	---	---	---	7.7E-01	---	---	---	7.2E-03	---	---	---	---
91-20-3	Naphthalene	---	---	---	---	4.4E+00	---	---	---	4.1E-02	---	---	---	---
85-01-8	Phenanthrene	---	---	---	---	2.6E-01	---	---	---	2.5E-03	---	---	---	---
119-00-0	Pyrene	---	---	---	---	1.6E-01	---	---	---	1.5E-03	---	---	---	---
	Carcinogenic PAHs													
56-55-3	Benzo(a)anthracene	2.4E+00	---	---	---	5.5E-02	1.1E-06	---	---	5.2E-04	8.1E-07	---	---	3.8E-04
50-32-8	Benzo(a)pyrene	3.3E+00	3.1E-09	4.1E-07	4.1E-07	4.7E-02	1.5E-06	---	1.4E-04	4.4E-04	1.1E-05	---	1.2E-07	3.2E-03
205-99-2	Benzo(b)fluoranthene	2.5E+00	---	---	---	4.3E-02	1.2E-06	---	---	4.0E-04	8.6E-07	---	---	2.9E-04
218-01-9	Chrysene	---	---	---	---	4.5E-02	---	---	---	4.2E-04	---	---	---	3.1E-06
193-39-5	Indeno(123-cd)pyrene	2.5E+00	---	---	---		1.2E-06	---	---	---	8.7E-07	---	---	---
	Phthalates													
117-81-7	bis(2-Ethylhexyl)phthalate	0.0E+00	0.0E+00	NA	NA	6.9E-02	0.0E+00	0.0E+00	---	6.5E-04	0.0E+00	0.0E+00	---	9.1E-06
	Other SVOCs													
132-64-9	Dibenzofuran	---	---	---	---	2.6E-02	---	---	---	2.4E-04	---	---	---	---
	Inorganic Compounds													
7429-90-5	Aluminum	5.2E+03	5.0E-06	---	5.0E-06	1.9E+00	2.4E-03	7.8E-05	1.6E-03	1.8E-02	---	---	---	---
7440-38-2	Arsenic	5.6E+00	5.3E-09	---	5.3E-09		2.6E-06	2.7E-07	1.8E-06	---	3.9E-06	4.3E-07	7.6E-09	---
7440-39-3	Barium	---	---	---	---	1.7E-01	---	---	---	1.6E-03	---	---	---	---
7440-41-7	Beryllium	2.7E-01	2.5E-10	---	2.5E-10		1.2E-07	4.0E-09	8.4E-08	---	5.4E-07	1.7E-06	2.0E-10	---
7440-43-9	Cadmium (Food)	1.8E+00	1.7E-09	---	1.7E-09		8.5E-07	2.7E-08	5.7E-07	---	---	---	1.0E-09	---
57-12-5	Cyanide	---	---	---	---	3.8E-02	---	---	---	3.6E-04	---	---	---	---
7439-89-6	Iron	1.9E+04	1.8E-05	---	1.8E-05	1.4E+01	8.7E-03	2.8E-04	5.8E-03	1.3E-01	---	---	---	---
7439-92-1	Lead	1.4E+02	1.4E-07	---	1.4E-07	4.3E-03	6.6E-05	2.1E-06	4.5E-05	4.0E-05	---	---	---	---
7439-96-5	Manganese	3.3E+02	3.2E-07	---	3.2E-07	4.2E-01	1.6E-04	5.0E-06	1.1E-04	3.9E-03	---	---	---	---

Pathway Risks			
1.8E-05	2.1E-06	1.3E-07	6.4E-03

Total Cancer Risk	6.5E-03
Reference Cancer Risk	1.0E-06

ORIGINAL (Red)

Menzie-Cura & Associates, Inc.
One Courthouse Lane
Suite 2
Chelmsford, Massachusetts 01824
Telephone (978) 453-4300
Fax (978) 453-7260

June 3, 1998

FILE : 384j

Steve Donohue
USEPA Region III
Office of Superfund
Mail Code 3HW22
841 Chestnut Building
Philadelphia, PA 19107

RE: Former UGI Columbia Manufactured Gas Plant
Baseline Human Health Risk Characterization
Approval amendments

Mr. Donohue,

Enclosed is a revised page *iv* and Appendix H for the referenced report. Please attach these items to the April, 1998 report to finalize the submittal.

Sincerely,

Jo Anne Shatkin, Ph.D.
Senior Scientist

cc: TM, JV, JR, SM, DA, file

enc.

Appendix H

EPA Memorandum on Baseline Human Health Risk Assessment, April 1998, dated May 19, 1998 from Lynn Flowers, Ph.D. to Steve Donohue

ORIGINAL (Red)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

Office of Superfund
Steven J. Donohue

Direct Dial (215) 597-3166
Mail Code (3HW22)

Douglas C. Ammon, P.E.
Project Manager
Clean Sites Environmental Services, Inc.
635 Slaters Lane, Suite 130
Alexandria, VA 22314

June 1, 1998

Re: UGI Columbia Gas MGP Site
Approval of Final Baseline Human Health Risk Assessment Report

Dear Doug:

The United States Environmental Protection Agency ("EPA") has received and reviewed the April 1998 Baseline Human Health Risk Assessment ("RA") for the UGI Columbia Manufactured Gas Plant Site ("Site"). The RA was submitted by Menzie Cura & Associates, Inc. and received by EPA on April 16, 1998. EPA has reviewed the RA to ensure revisions made to the text and appendices were responsive to comments made by EPA in a January 6, 1998 letter and subsequent communications.

Enclosed please find a copy of an internal EPA memorandum from Lynn Flowers, Ph.D., the toxicologist for the Site, to me dated May 19, 1998. EPA will consider the RA final and approved provided the memo is included as an appendix in the Final RA. EPA recommends the table of contents be revised, a tab be made for the additional appendix and the items forwarded to EPA for inclusion in the previously submitted RA. If you have any questions or would like to discuss the contents of the memo please contact me.

I have contacted Tony Martinelli, the Pennsylvania Department of Environmental Protection ("PADEP") project manager for the Site. Mr. Martinelli indicated that PADEP would not have any additional comments on the RA. Therefore, satisfaction of the EPA comments would make the RA final.

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(Red)

As indicated in my April 8, 1998 letter approving the Remedial Investigation report, please contact EPA and PADEP as soon as possible to schedule a meeting to discuss the Feasibility Study for the Site. If you have any questions on the above comments please contact me at the number above.

Sincerely,

Steven J. Donohue
Remedial Project Manager

cc: Anthony Martinelli, PADEP

ORIGINAL (Red)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Former UGI Columbia Manufactured Gas Plant
Baseline Human Health Risk Assessment
April 1998

FROM: Lynn Flowers, Ph.D., Toxicologist
Technical Support Section (3HS41) Lynn Flowers

TO: Steve Donohue, RPM
Eastern Pennsylvania Section (3HS22)

May 19, 1998

I have reviewed the document and the accompanying responses to EPA comments and have the following comments for your consideration:

Responses to January 6, 1998 Comments

(1) Comment 3: "worker" should be "resident."

(2) Comment 4: The sediment data for the Susquehanna River has not been included in Appendix A as indicated.

(3) Comment 11: Section 3.2.6 indicates that residential exposure to on-site subsurface soil was not included in the risk assessment (see last sentence of second paragraph). This exposure pathway should have been included in the risk assessment as the soil would have to be re-worked in order to build a residence on the Site.

(4) Comment 13: The statement that the PBF units do not cancel properly is true. However, the actual calculation is correct. The manipulation of the equation in order to incorporate a more appropriate Q/C value is entirely acceptable.

(5) Comment 24: The reference should be to aluminum and beryllium.

(6) Comment 39: Total skin surface areas and soil adherence factors that were actually inputted into the risk equations were requested and are not found in Appendix A.

(7) Comment 40: The response is incorrect. It was stated that the 95% UCL of the mean would be used for both RME and CTE calculations. This has been misinterpreted. All other CTE exposure factors should be average values. The actual CTE values that were used are not shown in the document.

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(8) Comment 41: The final exposure frequency that was used in the risk assessment was needed in order to cross-check the calculations.

Responses to March 11, 1998 Comments

None

Comments on Risk Assessment Document

(1) ES; page viii; summary table:

(a). The risks for soil and ground water for future residents either on-site or off-site have been combined. It would be beneficial for these risks to be separated for risk management purposes. The values can be determined from Appendices E and G.
(b). Only surface soil was considered for an on-site residential scenario. Both surface and subsurface soil should have been included as the soil would require re-working in order for a home to be built on the Site. Residents would then be exposed to both surface and subsurface soils. Both surface and subsurface soils were considered in the assessment of risk to a potential off-site resident.
(c). The fraction ingested from source (Fi) should be 0.5 for off-site (both near the S. River and south of the Site) soil exposure scenarios, i.e., for construction workers and residents. It is presumed that the surface soil is uncontaminated. The risk values shown for these scenarios should be divided by two.
(d). The RME concentrations for inorganic compounds found in subsurface soils south of the Site (includes sample TP-A) are incorrect. It appears that they correspond to the subsurface soil concentrations found near the S. River. After correcting for this error, the noncancer hazard index for a construction worker who would be exposed to subsurface soils south of the Site is 0.6 and the increased cancer risk is 1.1E-5.

(2) ES; conclusions: No reference is made to the dermal exposure risk characterizations that were included in the risk assessment.

(3) Page 10; "A-6" should be "A-4" and "A-7" should be "A-5."

(4) Tables: A number of the Tables contain footnotes which incorrectly state that benzo[ghi]perylene and acenaphthylene will be evaluated "quantitatively" as opposed to "qualitatively."

(5) The qualitative discussion on the toxicity of benzo[ghi]perylene and acenaphthylene is lacking in information. The on-site and off-site concentrations of acenaphthylene ranged from 3.8 to 160 mg/kg. The concentration of benzo[ghi]perylene ranged from 4.4 to 11 mg/kg.

Additional Comments

(1) The effect of including all PAHs detected in soils in the risk assessment was determined. The risk values were increased but the outcome of the risk assessment was unchanged.

(2) Taking all comments into consideration, the possibility of slightly varying input variables being used in the EPA analysis, and rounding differences, the following is a corrected summary of the risks found at the Site for all scenarios, including those where no errors were found.

Ground water human health risks (on-site and off-site) are unacceptable and can be found in Appendix E.

Conclusions

(1) None of the above-mentioned corrections would lead to a change in the overall outcome of the risk assessment.

(2) On-site and off-site ground water human health risks were found to be unacceptable.

(3) On-site soils (surface and subsurface) would pose an unacceptable risk to potential future residents.

(4) The Hazard Quotients for the on-site construction worker scenario and the off-site child-resident scenario were found to be 1.2 and 1.8, respectively. These risks are associated with several target organs, i.e., skin effects, central nervous system effects, and iron overload, with each individual systemic effect having a hazard quotient of less than 1.0. Therefore, the on-site soils do not pose a health threat to construction workers and the off-site soils do not pose a risk to a potential future child resident.

(5) Dermal risk to PAHs present in soils was not considered in this risk assessment. Currently, toxicity values for the determination of risk through dermal exposure to PAHs are not available. EPA Region III has been advised (National Center for Environmental Assessment, Cincinnati, OH) to not include an assessment of this risk as the choice of toxicity values would be inappropriate. It should be noted that the non-cancer risk (due to skin irritation from PAHs) and the cancer risks would be increased if it were possible to quantitate this facet of the risk assessment. It is not possible to estimate the impact of this uncertainty.

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UGI Columbia MGP risks by scenario		
	Total Risk	
On-site	Hazard Index	Cancer Risk
Industrial worker (surface soil only)	0.12	1.3E-5
Construction worker (surface and subsurface soil)	1.2	2.3E-5
Trespasser (surface soil and sediment)	0.05	1.3E-6
Child resident (surface and subsurface soil)	4.2	4E-4
Adult resident (surface and subsurface soil)	0.4	1.6E-4
Off-site (subsurface soil near Susquehanna River)		
Construction worker	0.4	3.3E-6
Child resident	1.8	6.2E-5
Adult resident	0.12	2.4E-5
Off-site (subsurface soil south of the Site)		
Construction worker	0.6	1.1E-5

If I can be of further assistance please contact me at 6-3115.

cc: E. Johnson (3HS41)
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