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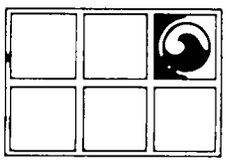

GROUNDWATER
TECHNOLOGY, INC.

GROUNDWATER TECHNOLOGY

Groundwater Technology, Inc.
12 Walker Way, Albany, NY 12205
Tel: (518) 456-2444

SUBSURFACE INVESTIGATION REPORT

OSMOSE WOOD PRESERVING, INC.
980 Ellicott Street
Buffalo, NY 14209
NYS DEC Site No: 91543

June 28, 1991

Submitted to:

Mr. Michael E. Rider
Plant Manager
Osmose Wood Preserving, Inc.
Buffalo, NY 14209

SIGNATURE PAGE

Prepared by:

Bruce W. Ahrens
Project Manager
Senior Engineer

Reviewed by:

Wendy C. Leonard
District Technical Coordination
Senior Hydrogeologist

Lance McGlynn
Project Engineer

Marty Gildea
Project Geologist

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1.0 EXECUTIVE SUMMARY

Osmose Wood Preserving, Inc., (Osmose) operates a facility which manufactures wood preserving products in Buffalo, New York. During removal of 3 underground storage tanks (USTs) in August, 1989, evidence of a release to the subsurface, believed to be #2 fuel oil and/or brushing grade creosote, was discovered. In June, 1990 the Osmose site was included on the New York State Registry of Inactive Hazardous Waste Sites, given a "2a" classification, and assigned New York State Department of Environmental Conservation (NYS DEC) site number 915143.

Groundwater Technology, Inc., (Groundwater Technology) was retained by Osmose to develop and implement a subsurface investigation work plan. The work plan, titled Subsurface Investigation Work Plan for Osmose Wood Preserving, Inc., Buffalo, New York, June 7, 1990, defined the field and analytical procedures and protocols required to evaluate the geologic characteristics of the site and to define the nature and extent of chemical hazards present. Included in the work plan was the implementation of an in-situ soil treatment biocell as an Interim Remedial Measure (IRM) to treat soils excavated during the UST closure. This IRM was conducted under Order of Consent, Index #B9-0314-90-01.

A summary of the results of the subsurface investigation includes:

- Modified soil gas survey techniques indicated non-detectable vapor levels existed both on- and off- site,
- Inorganic compounds (metals) exist within typical published levels in surface and subsurface soils with the exception of zinc, in surface soils upgradient of the Osmose site, and lead, in surface soils at several upgradient (background) and on-site locations,
- Analysis for semi-volatile organic compounds indicated polynuclear aromatic hydrocarbons (PAHs) were the predominant chemicals existing and were present at all biocell and on-site (non-biocell) locations,
- Low dissolved levels of PAHs in groundwater were detected in on-site and in 2 off-site monitor wells
- No pesticides or PCBs were detected on- or off-site.

An integral portion of the work plan included the performance of a baseline Health and Environmental Risk Assessment. The goals of the Risk Assessment were to:

- Provide an analysis of baseline risks to help determine the need for action at the Osmose site, and
- Provide a basis for determining levels of chemicals that can remain on-site and still be protective of human health and environment.

Based upon the combined results of the subsurface investigation and the Risk Assessment, the following site remedial actions are proposed:

- Biocell Soils: Operation of the biocell until total PAH levels in soils are at, or below 473 mg/kg.
- Off-site soils: No remedial action.
- Groundwater: No remedial action; quarterly monitoring. Installation of 1 overburden monitor well to monitor upgradient water quality and provide additional soils information in the area west of MW-8.
- On-site Soils: Installation of one shallow boring to confirm the disassociation of PAHs found at shallow depths at an upgradient location of the site (MW-8), with the former tank pit area; delineation and investigation of potential remedial options.
- Separate Phase: Recovery of intermittent product layers; installation of one monitor well to delineate downgradient extent.

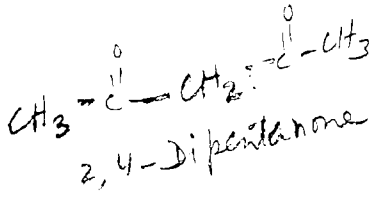
2.0 INTRODUCTION

2.1 Background

Osmose Wood Preserving, Inc. (Osmose) operates a facility which manufactures wood preserving products in Buffalo, New York (Figure 1, Site Location Map). The facility is located at 980 Ellicott Street and serves as the executive and accounting offices, along with research and product production. Osmose manufactures a variety of preservatives used in the treatment of wood and lumber products.

Osmose has sought to maintain this facility in accordance with the most current technology and environmental policies. In keeping with this goal, in August, 1989, as part of their storage system upgrade program, Osmose permanently closed by removal two 12,000 gallon and one 10,000 gallon underground storage tanks (UST's) formerly associated with product production. Prior to closure by removal, the three compartmentalized UST's (6 compartments total) were utilized for the storage of bulk deliveries of raw materials required in the manufacture of wood preservatives. Presented below is a list of materials historically stored in the UST's:

- Brushing Grade Creosote - stored until August, 1989
- #2 Fuel Oil - stored until August, 1989
- Mineral Spirits - stored until 1986
- Isopropyl Alcohol & Diacetone mixture - stored until 1984
- Coal Tar - stored until 1964



During UST removal evidence of a release to the subsurface, believed to be #2 fuel oil and/or brushing grade creosote, was discovered.

Osmose developed and submitted to the New York State Department of Environmental Conservation (NYS DEC) an Interim Remedial Measure (IRM) work plan for contaminated soils which were excavated during storage system closure. The goal of the IRM is to implement an in situ soil treatment bioremediation (biocell). This IRM is being conducted under an Order of Consent, Index # B9-0314-90-01.

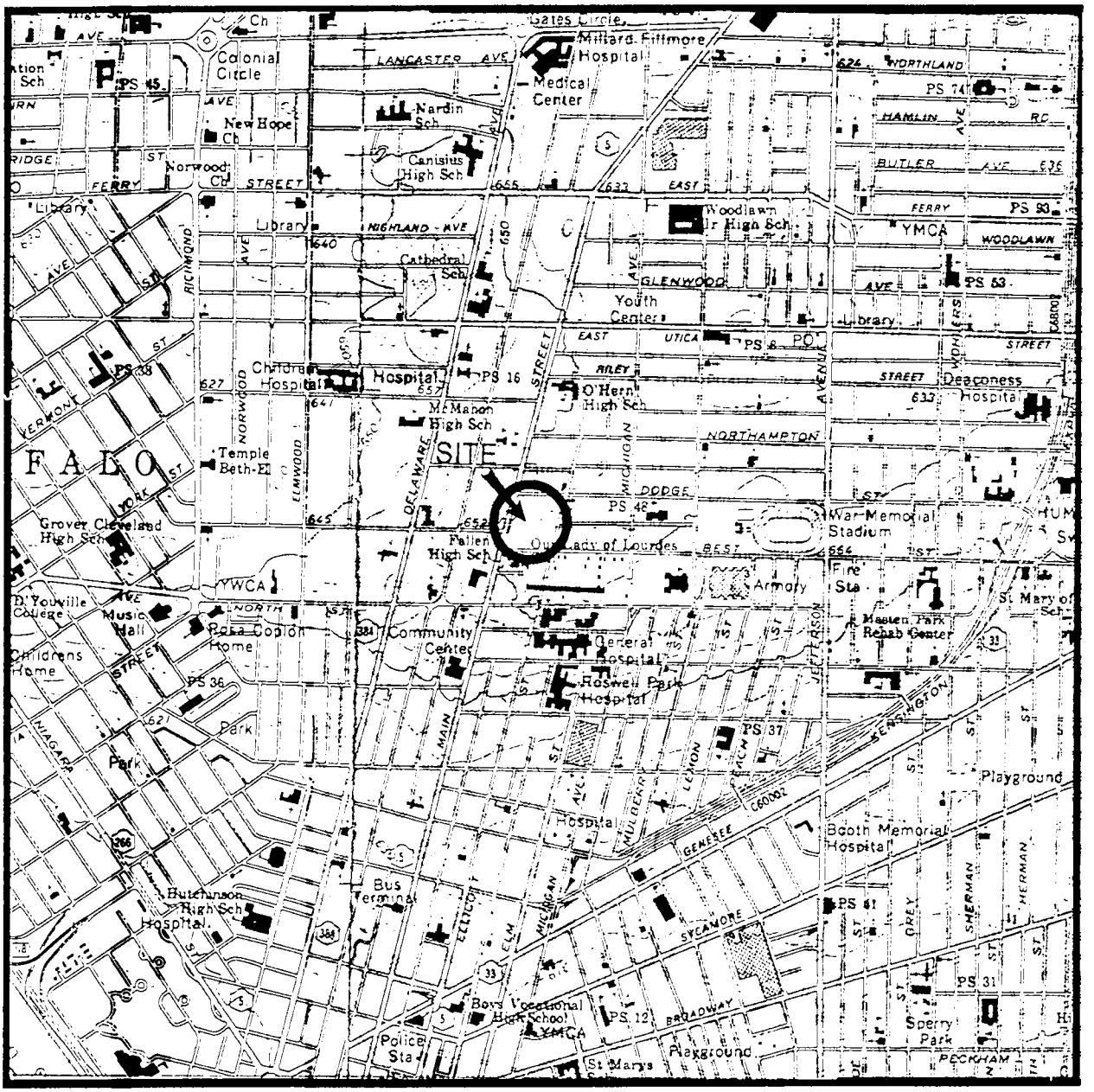
In June, 1990, Osmose was notified by the NYS DEC of their inclusion in the New York State Registry of Inactive Hazardous Waste Disposal Sites. Number 915143 was assigned as the NYS DEC site number. The site was classified as "2a". This temporary classification is assigned to sites for which there is inadequate data to assess threats to public health and environment.

FIGURE 1

SITE LOCATION MAP

OSMOSE WOOD PRESERVING, INC.

BUFFALO, NEW YORK



SOURSE: NYS DOT 7.5 MIN. BUFFALO NE & NW QUADS SCALE: 1 IN. EQUALS 2,000 FT.

GROUNDWATER TECHNOLOGY, INC.

2.2 Objectives

Osmose contracted Groundwater Technology, Inc. (Groundwater Technology) to prepare a work plan to investigate the extent of contamination at the Osmose facility. The work plan, titled Subsurface Investigation Work Plan for Osmose Wood Preserving, Inc., Buffalo, New York, June, 1990, (Work Plan) was submitted to the NYS DEC. Acceptance of the Work Plan was transmitted in a letter dated July 2, 1990 from Mr. Jaspal Walia, P.E. to Osmose. The Work Plan was developed to satisfy the general requirements of a NYS DEC State Superfund Phase II type investigation and was not meant for consideration as a Remedial Investigation.

The objectives of the scope of work defined in the Work Plan included:

- Identification of contaminants in soils and groundwater associated with product release from the USTs,
- Delineation of the horizontal and vertical extent of the contaminants present, and
- Assessment of potential risks to human health and environment resulting from the product release by performance of a Health and Environmental Risk Assessment.

Health and environmental risk data was used to determine which transport medias require remediation and to propose risk driven remediation goals for those media. The risk assessment was developed to address the following:

- Soils within the soil treatment biocell (biocell) as part of the IRM,
- Contaminated soils outside the soil treatment biocell,
- Off-site soils along Ellicott Street adjacent to the site (off-site) and,
- Groundwater on and downgradient of the Osmose property.

The risk assessment report is presented in Section 6.0 of this document.

An additional objective of the Work Plan was to perform a broad based screening of remedial alternatives which could be selected to abate conditions identified as posing potential risk to health and the environment. A key element of the remedial alternative screening process is equating the remediation goal to the technical feasibility of obtaining that goal. Once the proposed remediation goals have been accepted by the NYS DEC, or acceptable alternative goals negotiated, a detailed screening of remediation technologies can be accomplished.

2.3 Workscope

As described in the approved Work Plan, the workscope included the following tasks:

- Site Health and Safety Planning,
- Field Investigation,
- Sample Analysis and Validation,
- Data Evaluation,
- Health and Environmental Risk Assessment Preparation, and
- Remedial Screening.

Included in the following pages is a detailed description of the results of the workscope as described above. Details of specific procedures and protocols, or deviations from the approved Work Plan, are included as required. Additional details of field procedures and QA/QC protocols can be located in the Work Plan.

As mentioned above, one of the objectives of the Health and Environmental Risk Assessment was to develop risk driven remediation goals for the biocell IRM. The IRM design, installation, and monitoring and maintenance details are not included in this Subsurface Investigation Report.

3.0 FIELD INVESTIGATION

3.1 Site Health and Safety Plan

A site and task specific Health and Safety Plan (HSP) was prepared by Groundwater Technology in accordance with Occupational Safety and Health Administration (OSHA) standard "Hazardous Waste Operations and Emergency Response" guidelines (29 CFR 1910.120). The HSP was designed to minimize exposure of Groundwater Technology employees and subcontractors to potentially hazardous substances. In addition, the HSP provides a contingency plan in the event such exposure should occur. A copy of the NYS DEC approved HSP is provided as Appendix E in the Work Plan.

Ambient air monitoring was performed by the Groundwater Technology Site Safety Officer during all site activities and community air monitoring during all soil disturbance activities as detailed in the HSP. Air monitoring included screening for Volatile Organic Compound's (VOCs)[1] and Airborne Particulates.

A Photovac Microtip Photoionization Detector (PID) fitted with a 10.2 eV lamp was used to monitor VOCs. Vapor Monitoring Logs, including sampling date, time, locations, and weather conditions are included in Appendix B. Inspection of the logs indicates that during the modified SGS, no detectable levels of VOCs were measured in ambient air. During soil boring/monitor well installation (refer to Section 3.4) non-detectable levels of VOCs were typically encountered, however, several readings ranging from 0.2 - 2.3 ppmv were detected. All levels detected were below the Threshold Limit Value (TLV) and Permissible Exposure Limit (PEL). No work stoppages occurred due to vapor emissions.

Also included in Appendix B are the Airborne Particulates sampling logs. A Miniram ® Sun Shield Model PDM - SNS particulate meter was set at 150 $\mu g/m^3$ above background as prescribed in the HSP. Downwind particulate levels did not exceed this threshold during soil disturbance activities.

All site work was performed in Level D protective equipment.

[1] VOC's was the only parameter monitored during the soil gas survey.

3.2 Modified Soil Gas Survey

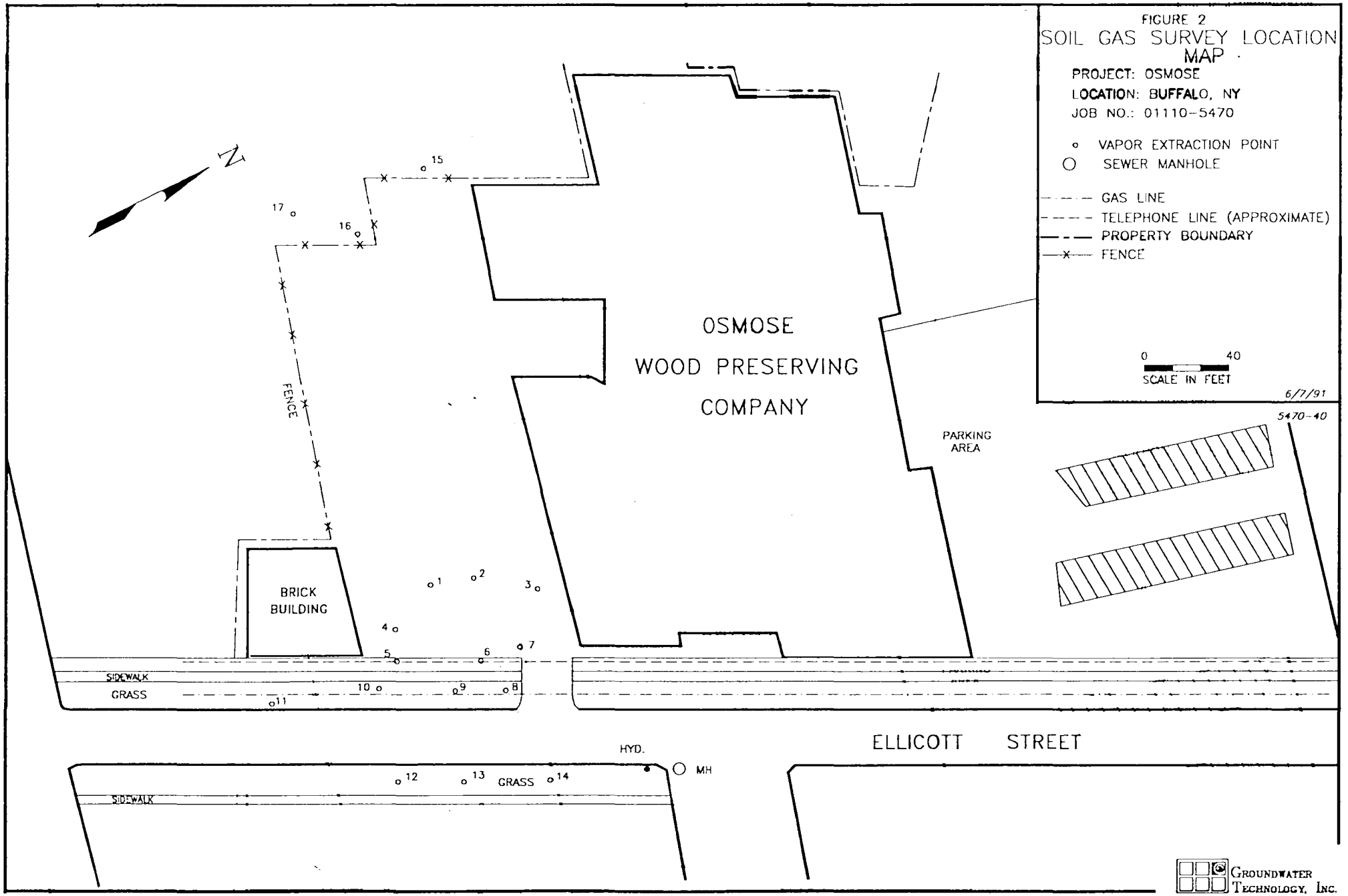
Modified Soil Gas Survey (SGS) is a quantitative, semi-qualitative analysis which provides rapid and cost-effective areal delineation of relative concentrations of volatile and semi-volatile compounds within the unsaturated soil zone.

The objectives of the SGS, performed at the Osmose site on August 23, 24, and 25, 1990, were:

- define the areal extent of volatile and semi-volatile organic vapors in the unsaturated soil zone,
- assist in selecting appropriate monitor well locations,
- determine if levels were sufficiently high to enable effective use of a portable field gas-chromatograph (GC) during monitor well installation, and
- evaluate the potential of soil gas (vapors) as an off-site migration route.

In addition to the above stated objectives, results from the soil gas survey would be used to infer the extent of Light Non-Aqueous Phase Liquid (LNAPL), if present. On August 23-25, 1990, soil gas data were collected from 17 vapor extraction points (VPs) located in the vicinity of the former tank pit area, along the boundaries of the Osmose property and in the right-of-ways bordering Ellicott Street. Figure 2, Soil Gas Survey Location Map, indicates the locations where soil gas was sampled. As indicated on the location map, 3 vapor extraction points (VPs) were located along the upgradient (VP-15, VP-16, & VP-17), and downgradient (VP-5, VP-6 and VP-7) boundaries of the site as specified in the Work Plan.

Soil gas collection was accomplished by driving a 1/4 inch diameter, hollow, stainless steel probe with a perforated point into the soil to a depth of 3 feet below grade and drawing a sample of soil vapor. The volume of ambient air within the sample probe was calculated and evacuated. Once ambient air was evacuated, the soil gas was screened for VOC's using a Photovac Microtip photoionization device. Charcoal sampling tubes were then connected to the stainless steel probes and a vacuum applied (via a portable vacuum pump). A metered volume of soil gas was then drawn through the charcoal tube. The flowrate of the soil gas drawn through each tube was precisely measured using a rotometer equipped with a needle valve to control flow. A stopwatch was utilized to measure the sampling interval. Soil gas flow rates and duration of pumping were carefully recorded for each vapor extraction point. Included in Appendix C is a summary of the soil gas flowrates and pumping time intervals. The flowrates for both volatile and semi-volatile samples was set at 1 liter per minute.



GROUNDWATER TECHNOLOGY, INC.

Two (2) soil gas samples were extracted from each VP. The samples (carbon tubes) were sent to the contract laboratory, GTEL Environmental Laboratories in Milford, NH for analysis. As prescribed by the laboratory, charcoal samples were not sent on ice to preclude condensation within the sample tubes. One sample was analyzed for BTEX components which required a volume of 5 liters of soil gas to be drawn through the charcoal tube. The second sample was analyzed by Modified NIOSH Method 1501 for Polynuclear Aromatic Hydrocarbons (PAHs). These samples required that 20 liters of soil gas be drawn through the charcoal tube.

Between each soil gas sampling location the stainless steel probes were thoroughly decontaminated with an Alconox and water solution, rinsed with clean water, then with methanol, and finally purged with flame to remove moisture and trace volatiles. The probes were then allowed to cool.

A summary of the soil gas survey analytical results are presented in Section 4.2, Table 4-1.

In compliance with the Work Plan, several soil gas samples were obtained for quality control purposes. The intent of the internal quality control program was to detect potential problems at the source and, if necessary, trace the sample analytical pathways for introduction of contamination. The quality control data generated in the field was used to monitor sampling technique, reproducibility and cleanliness. The quality control samples included:

- two blank samples,
- one equipment blank,
- one trip blank, and,
- one blind duplicate sample (VP-18).

All blanks and duplicates were sampled and analyzed for both BTEX and PAH components.

The purpose of the equipment blank was to assess the potential for carryover contamination on the sample probes and within the tubing between the probe and the carbon sampling tubes. These blanks were obtained using the identical probes and vacuum pump apparatus used to obtain the soil gas samples, the only difference being that ambient air, rather than soil vapor, was drawn through the tube. These blanks were subsequently handled and analyzed in a similar fashion to the soil vapor samples.

A trip blank sample was obtained to detect external sources (ie., ambient air) of contamination and sampling tube cleanliness. The sample was prepared by breaking off the ends of the charcoal tube, then immediately recapping them. The trip blank accompanied the soil vapor samples from the point of sampling to the final laboratory analysis.

A blind duplicate was extracted at vapor point VP-6. The purpose of the blind duplicate was to insure the precision of both field and laboratory measurements. The blind duplicate (labeled VP - 18) was handled and analyzed in a fashion identical to that of the other samples.

3.3. Surface Soil Grab Samples

At each soil vapor extraction point, a grab sample of soil was obtained from the surface. All grab samples were collected using a clean stainless steel spade. The soil was placed into appropriate glass containers. The samples were subsequently placed on ice and shipped overnight to the contract laboratory. All samples were stored at the laboratory awaiting the results of the soil gas survey. After review of the soil gas survey results, six of the soil samples were chosen for analysis of Priority Pollutant Metals [(PPM) Total Metals as per SW 846]. Three (3) of these soil samples were analyzed from an area located downgradient of the presumed source area (VP-5, 8 and 13). The remaining 3 soil samples were taken from areas located upgradient from the presumed source area (VP-15, 16, and 17).

Laboratory analytical reports from the soil grab sampling event are summarized in Section 4.3. Also included is a discussion of the results.

3.4. Soil Borings/Monitor Well Installation

3.4.1 Soil Boring Installation

To aid in the determination of the vertical and horizontal extent of adsorbed and dissolved phase contamination, soil borings were completed within the overburden. Seven (7) soil borings were installed from October 1 through October 16, 1990 by Groundwater Technology utilizing a Mobile B-61 hollow-stem auger drill rig. The locations of these borings were based upon a review of existing groundwater gradient data and the results of the soil gas survey. Borings completed in this phase of the investigation were located as follows:

- one (1) upgradient of the former tank pit (MW-8),
- three (3) downgradient of the former tank pit area (MW-9, MW-10, and MW-11).

- two (2) borings installed as a cluster located immediately downgradient of the former tank pit area (CW-1 & CW-2)
- one (1) soil boring within the bedding of an existing storm sewer line (SB-1).

The 3 downgradient borings were placed within the right of ways bordering Ellicott Street. Soil boring SB-1 was installed within the sewer bedding located just west of the centerline and beneath Ellicott Street. Soil boring and monitor well locations are shown on Figure 3, Monitor Well Location Map.

Borings MW-8 and CW-1 were completed as described in Section 6.2.2 of the Work Plan. These borings were advanced through the uppermost clay layer into the underlying sands and silts until an aquitard (MW-8) or bedrock (CW-1) was reached. Boring CW-2 and SB-1 were also completed in accordance with the Work Plan. Boring CW-2 was completed within the upper clay layer adjacent to CW-1, while SB-1 was completed within the sewer bedding located beneath Ellicott Street. A third well in the cluster was proposed in the Work Plan. The third well in the cluster was proposed which would be screened at an intermediate depth below the clay horizon to a lower confining aquitard, if present. This intermediate cluster well was not necessary due to the absence of a substantial aquitard between the upper clay layer and bedrock (refer to Section 4.1, Geologic Evaluation).

Due to the unexpected depth to bedrock (63 feet) found at boring CW-1 and the lack of an intermediate confining layer between the upper clay layer and bedrock, the criteria for determining the depth of the remaining borings (MW-9, MW-10, and MW-11) presented in the Work Plan was amended as follows:

- Each boring was advanced through the upper clay layer into the underlying sands and silts to a minimum of 25 feet, unless an aquitard was intersected before that depth.
- Each boring was advanced until field screening results indicated non-detect soil readings then continued 5 feet beyond that depth.

These amends were developed with Mr. Jerry Pietraszek of the NYS DEC during a telephone conversation on the evening of October 3, 1990. The results of this conversation were also discussed with Mr. Jaspal Walia, P.E., Senior Sanitary Engineer, NYS DEC. Mr. Walia indicated these amendments were acceptable.

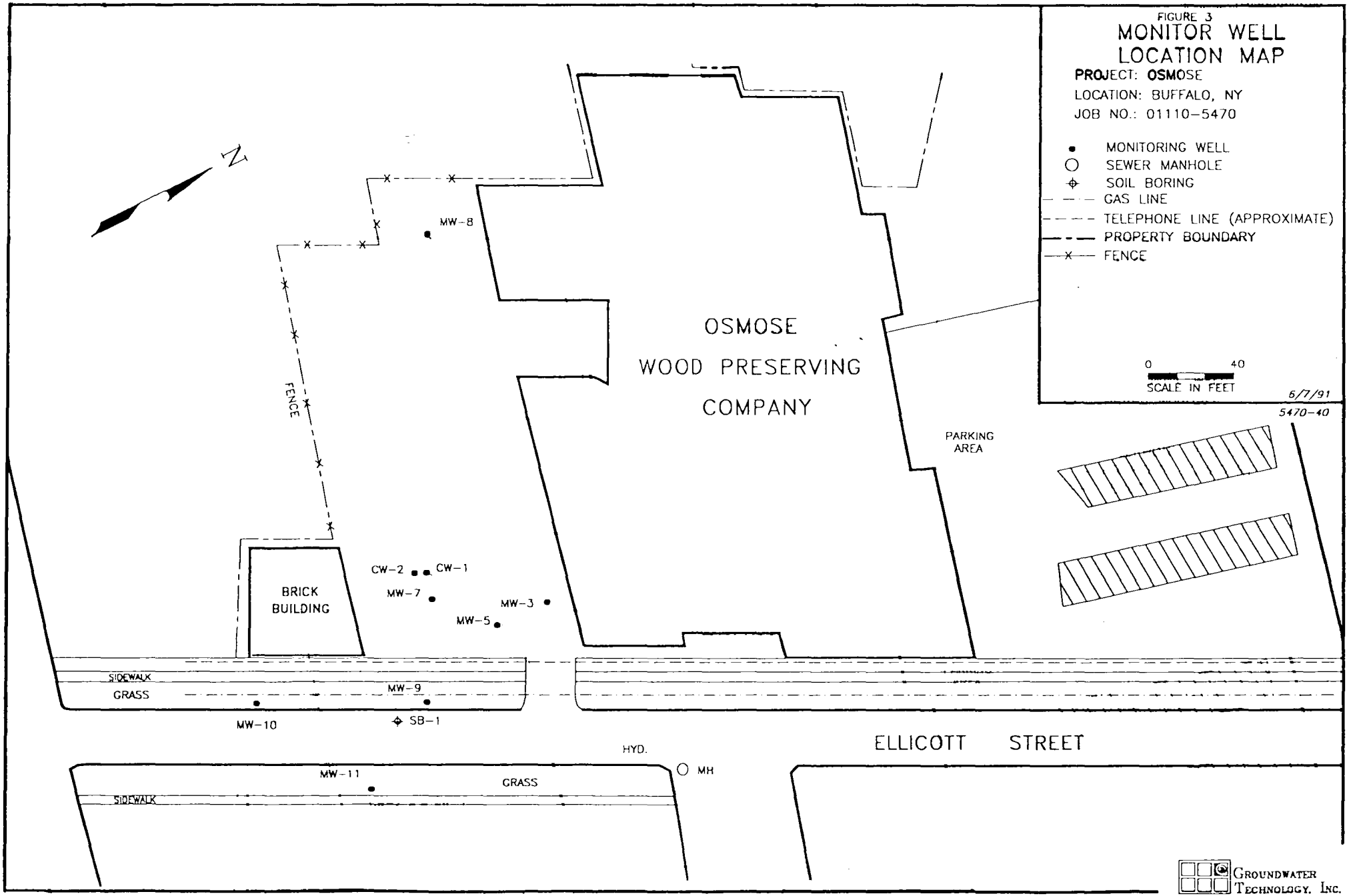


FIGURE 3
MONITOR WELL LOCATION MAP
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
0
40
SCALE IN FEET
6/7/91
5470-40
N
OSMOSE
WOOD PRESERVING
COMPANY
PARKING
AREA
FENCE
MW-8
BRICK
BUILDING
CW-2
CW-1
MW-7
MW-3
MW-5
SIDEWALK
GRASS
MW-9
MW-10
SB-1
HYD.
MH
MW-11
GRASS
SIDEWALK
ELLICOTT STREET
GROUNDWATER
TECHNOLOGY, INC.

3.4.2 Subsurface Soil Sampling

During installation of the soil borings, subsurface soil samples were collected. The objective of the soil sampling program was to determine the lithology of the soils and the presence or absence of any volatiles, semi-volatiles, and inorganic compounds which may exist in the soil matrix and define their vertical and horizontal distribution.

Soil samples were collected continuously at 2 foot intervals for each soil boring using standard split-barrel sampling tubes. The soil samples were visually classified and field screened for VOCs utilizing a Photovac Microtip PID. A portable PID was used rather than a field gas chromatograph (GC) based on the results of the modified SGS (refer to section 4.2). As described in Section 6.2.3 of the Work Plan, the PID was used to determine which soil sample from above the water table in each soil boring would be sent to the laboratory for analysis. In addition, soil samples from below the air/water interface were collected and sent for analysis. Presented in Table 3-1 below is a summary of the locations and depths from which the soil samples were taken.

TABLE 3-1

SUBSURFACE SOIL SAMPLE LOCATIONS

October 1-16, 1990

		ANALYSES		
WELL LOCATION	SAMPLE DEPTH	BTEX EPA 8020	PAH EPA 8310	PPM
MW-8	2'-4'	X	X	X
	16'-18'	X	X	X
MW-9	4'-6'	X	X	X
	10'-12'	X	X	X
	30'-32'	X	X	
MW-10	6'-8'	X	X	X
	10'-12'	X	X	X
MW-11	4'-6'	X	X	
	10'-12'	X	X	
CW-1	6'-8'	X	X	
	8'-10'	X	X	
	30'-32'	X	X	
	62'-64'	X	X	
CW-2	6'-8'	X	X	
SB-1	4'-6'	X	X	
BLIND DUPLICATE*	10'-12'	X	X	

* = Blind duplicate sample of MW-11

Laboratory reports of the soil analyses are included in the Laboratory Analyses from the Subsurface Investigation Work Plan, January, 1991, previously published under separate cover.

Soil boring logs are included in Appendix D. A discussion of site geology is included in Section 4.1 of this report.

3.4.3 Monitor Well Installation

Each of the soil borings installed during this phase of the investigation was completed as a monitor well with the exception of SB-1. The objective of completing the soil borings as monitor wells was to allow for definition of any dissolved and/or separate phase contaminant plumes.

Each monitor well was constructed of 2 inch diameter fiberglass reinforced epoxy (FRP) well screen and casing with flush-threaded joints. FRP construction was chosen for chemical compatibility reasons based upon review of the materials formerly stored in the USTs. This material assures the collection of representative samples of the groundwater in the formation surrounding the well.

With the exception of cluster well CW-2, at each monitor well location the screened interval was placed within the sands and silts below the upper clay layer (refer to Section 4.1, Geologic Evaluation). At CW-2 the screened interval was within the upper clay layer. At CW-1 the screened interval lies directly above the bedrock (63') and extends only 5 feet (58'-63' below grade). Each well was completed with an appropriate sand pack extending approximately 2 feet above the well screen followed by a 2 foot bentonite seal. The well was then grouted to the surface as specified in the Work Plan. Each well was then finished at grade in a water-tight road box which was cemented into place. Construction details of the individual monitor wells are presented in the Soil Boring Logs, Appendix D. Screened intervals of each monitor well are summarized below in Table 3.2.

TABLE 3-2

MONITOR WELL SCREENED INTERVALS

MONITORING WELL ID	SCREENED INTERVAL (feet below grade)
MW-8	16.0 - 21.0
MW-9	8.0 - 28.0
MW-10	11.0 - 25.0
MW-11	9.0 - 16.0
CW-1	57.0 - 62.0
CW-2	1.5 - 5.5

3.5. Groundwater Monitoring and Sampling

3.5.1 Monitor Well Development

Following installation, development of the monitor wells was performed by repetitive surging and bailing, as described in Section 6.2.4 of the Work Plan. A dedicated teflon bailer was decontaminated with an alconox solution, rinsed with clean water, nitric acid solution, distilled water, methanol solution, and finally rinsed with distilled water solution prior to bailing each well. An H.F. Scientific field turbidity meter was calibrated using Formazin standards of 198, 19.8, and 2.0 Nephelometric Turbidity Units (NTUs). Due to the low recharge of groundwater, Groundwater Technology field personnel were unable to develop the wells to a level below 50 NTUs as specified in the Work Plan. On October 31, 1990, Mr. Vincent Dondelinger of GTEL Environmental Laboratories, Milford, NH (contract laboratory) was consulted and confirmed that high turbidity (>200 NTU's) would not effect any of the required laboratory analyses as the samples are centrifuged prior to analysis. Groundwater Technology was informed by Mr. Jaspal Walia, P.E. and Mr. Jerry Peterzack of the NYS DEC that the 50 NTU limit for water analyses as specified in the Work Plan was a guideline that could be waived by the contract laboratory if it could be determined that the turbidity would not adversely affect the analytical results.

On November 2, 1990, the wells were developed. Included in Appendix E is a summary of well development details, including the approximate number of gallons removed from each well, the initial turbidity value prior to development (equilibrium), and the turbidity values recorded directly after well development.

All development water removed from the wells was stored in DOT approved 55 gallon drums on site until sufficient quantities existed for proper treatment and disposal.

3.5.2 Groundwater Sample Collection

As outlined in Section 6.2.5 of the Work Plan, Groundwater Technology originally proposed 3 sampling events. Two (2) abbreviated groundwater sampling events would be performed after the initial 5 well sampling event. In each of these 2 abbreviated groundwater sampling events, groundwater samples would be collected from 1 upgradient and 2 downgradient monitor wells. The purpose of these 2 additional sampling rounds was to evaluate potential trends in contaminant levels which could impact the exposure assessment portion of the proposed Risk Assessment (refer to Section 6.0, Risk Assessment). In December 1990, Envirologic Data (ELD), a Division of

Groundwater Technology, contracted to perform the risk assessment, informed Groundwater Technology that a more useful and representative data package would be obtained by sampling all 5 monitor wells 1 additional time rather than 3 monitor wells 2 additional times. Groundwater Technology obtained permission from Mr. Walia to modify the groundwater sampling events accordingly to accommodate collection of a more useful data package. To specify the proper QA/QC requirements for the modified sampling plan, Groundwater Technology contacted Mrs. Maureen Serafini, Environmental Chemist for the NYS DEC. Mrs. Serafini approved of the modifications and recommended that the additional groundwater samples be analyzed and validated according to the NYS DEC Analytical Service Protocol (ASP), Category A. This recommendation was agreed upon by Osmose and Groundwater Technology in order to obtain 1 set of ASP data in the event that defensible data becomes necessary in the future.

The first groundwater sampling event was conducted on November 9, 1990. Prior to collecting groundwater samples, Groundwater Technology's field technician purged each well of approximately 5 well volumes of water or until the well was bailed dry. A dedicated teflon bailer was used for this purpose. Wells were bailed and sampled from the expected lowest hydrocarbon concentrations to the highest in order to minimize the possibility of cross contamination. These procedures insured a more representative groundwater sample at each well location. Decontamination procedures between monitor wells were followed as described in Appendix D of the Work Plan. Water samples were collected from MW-8, MW-9, MW-10, MW-11, and CW-1 as described in Section 6.2.5, Groundwater Sample Collection, and Appendix D, Project Specific QA/QC Plan, of the Work Plan. Laboratory analyses conducted on water samples collected during the first sampling event included BTEX by Modified Method 602, PAHs by EPA Method 610 and hardness. A water sample collected from CW-1 was analyzed for the Target Compound List (TCL) analytes (organics and inorganics) using non-CLP approved protocols. The TCL included the following analyses:

- Base/Neutral/Acids (B/N/As)
- Pesticides and PCBs by EPA Method 8080
- Volatile Organics by EPA Method 8240, and
- Inorganics (Total Metals).

In addition, water samples from 1 downgradient well (MW-11) and 1 on-site well (CW-1) were analyzed for Priority Pollutant Metals (PPM). A chart summarizing the respective laboratory analyses performed on groundwater samples from each monitor well is presented below as Table 3-3.

Field analyses of temperature, pH and conductivity were also performed on each of the water samples. Collected samples were then packed in ice and mailed via overnight courier to GTEL. A

summary of the analytical results and discussion are presented in Section 4.6 of this document. Data validation was performed to satisfy the NYS DEC's requirements for non-CLP methods according to EPA SW-846 on all sampling events.

Laboratory reports and the Data Validation Reports have been forwarded to Osmose and the NYS DEC under separate cover.

TABLE 3-3

FIRST GROUND WATER SAMPLING DETAILS
November, 1990

MONITOR WELL ID	BTEX EPA Mod 602	PAH EPA 610	HARDNESS	TCL	PPM
MW-11	X	X	X		X
MW-10	X	X	X		
MW-9	X	X	X		
MW-8	X	X	X		X
CW-1	X	X	X	X	X
BLIND DUP.	X	X	X	X	X
SMPL BLANK			X	X	X
TRIP BLANK	X	X	X	X	X
FIELD BLANK	X	X		X	X

The second groundwater sampling event was conducted on January 10 and 11, 1991. Based upon Mrs. Serafini's recommendations, groundwater samples were analyzed and reports generated which conformed to NYS DEC ASP, Category A protocols.

Analytical methods prescribed by ASP, Category A differed from the previous analyses (first groundwater sampling event) in that EPA Method 8010/8020 (Purgeable Halocarbons/Aromatic Volatile Organics) and EPA Method 8310 (PAHs) are used rather than Modified EPA Method 602 and

EPA Method 610, respectively. Table 3-4 below provides a summary of the respective ASP, Category A laboratory analyses performed on groundwater samples collected from each monitor well during the second groundwater sampling event.

TABLE 3-4

SECOND GROUND WATER SAMPLING DETAILS
ASP CATEGORY A
January, 1991

MONITOR WELL ID	VOLATILES EPA 8020	PAH EPA 8310	HARDNESS	HALOCARBONS EPA 8010	TOTAL METALS
MW-11	X	X	X	X	X
MW-10	X	X	X	X	X
MW-9	X	X	X	X	X
MW-8	X	X	X	X	X
CW-1	X	X	X	X	X
BLIND DUP.	X	X	X	X	X
FIELD BLANK	X	X	X	X	X
EQUIP BLANK	X	X	X	X	X
TRIP BLANK	2X			2X	

Field analyses of temperature, pH, and conductivity were also performed on each of the water samples. Samples were then packed in ice and mailed via overnight courier to GTEL. A summary of the analytical results and field analyses is also presented in Section 4.6 of this document. Laboratory reports and the data validation report are included under separate covers.

3.5.3 Groundwater Elevation Survey

Prior to purging each well during both groundwater sampling events, each well was gauged to determine depth to groundwater using an Electronic Interface Probe. The interface probe is capable of gauging the depth to groundwater/product with an accuracy of +/-0.01 feet. All depth to water data were converted to groundwater elevations using an arbitrary benchmark elevation of 100 feet. This information was used to develop groundwater gradient maps and to compare data to previously obtained values. A discussion of site hydrogeology, including groundwater gradient maps are included in Section 4.4. of this report.

3.6 Sample Management and Quality Control

The sample management and quality control sampling performed during the implementation of this work scope followed the approved Work Plan as specified in Appendix D, Project Specific QA/QC Plan. Proper chain-of-custody procedures were employed throughout. The sample preservation, equipment decontamination and laboratory tracking all followed the prescribed procedures.

The quality control sampling that was performed is shown in Table 3-5. The data validation reports, provided under separate cover, indicate any deviations or problems encountered relating to the sample handling and quality control for this work scope.

TABLE 3-5

QUALITY CONTROL SAMPLING SUMMARY

TASK/TYPE OF SAMPLE	MATRIX	ANALYSES	NUMBER OF SAMPLES
MODIFIED SOIL GAS SURVEY			
Trip Blank	Air	EPA 610, BTEX by 602	1
Blind Duplicate	Air	EPA 610, BTEX by 602	1
Equipment Blank	Air	EPA 610, BTEX by 602	
SURFACE SOIL GRAB SAMPLING			
Trip Blank	Water	Priority Pollutant Metals	2
Blind Duplicate	Soil	Priority Pollutant Metals	2
Equipment Blank	Water	Priority Pollutant Matals	1
SUBSURFACE SOIL SAMPLING			
Trip Blank	Water	Priority Pollutant Metals, EPA 8020, EPA 8310	1
Blind Duplicate	Soil	EPA 8310, BTEX by 8020	1
Equipment Blank	Water	Priority Pollutant Metals, EPA 8020, EPA 8310	1
GROUNDWATER SAMPLING			
FIRST ROUND			
Trip Blank	Water	Gas. Hydrocarbons by 602, EPA 610, Priority Pollutant Metals, Hardness	1
Blind Duplicate	Water	Gas. Hydrocarbons by 602, EPA 610, Priority Pollutant Metals, Hardness	1
Equipment Blank	Water	Gas. Hydrocarbons by 602, EPA 610, Priority Pollutant Metals, Hardness	1
SECOND ROUND			
Trip Blank	Water	EPA 8010 and 8020	3
Blind Duplicate	Water	EPA 8010 and 8020, EPA 8310, Priority Pollutant Metals	1
Equipment Blank	Water	EPA 8010 and 8020, EPA 8310, Priority Pollutant Metals	1
Field Blank	Water	EPA 8010 and 8020, EPA 8310, Priority Pollutant Metals	1

4.0 FIELD INVESTIGATION RESULTS

Presented in the following sections are the results of the field investigation. The field investigation was developed to define the types of chemical hazards existing at the Osmose facility, and their vertical and horizontal distribution. Imperative to the discussion of the results of specific Work Plan tasks is a general understanding of the site geology. The regional and site geology are therefore discussed first, to be used as a reference for following sections. The remainder of the sections follow in chronological order, as they occurred.

4.1 Geologic Evaluation

4.1.1 Regional Geology

Unconsolidated deposits in the region consist of glacial till, glacial outwash, fine grained glacial lake deposits, recent swamp deposits, and alluvium. The glacial lake deposits are composed of fine sand, silt and clay.

The bedrock in the area of investigation is the Onondaga Limestone. Structurally, the Onondaga Limestone dips gently to the south-southwest (Staubits and Miller, 1987) and has been encountered at depths ranging from above surface elevation (outcrops along Kensington Expressway) to 63 feet below grade (this investigation). The upper surface is typically irregular and contains deeply incised glacially carved channels, sink holes and solution features.

4.1.2 Site Geology

As indicated in the well logs and Geologic Cross Sections, (Figure 4), the site is underlain by approximately 63 feet of unconsolidated clay, silt, sand and gravel deposits which rest directly upon the Onondaga Limestone. These deposits are fairly typical of glacial deposits of the area, and exhibit varied permeability. The area of highest relative permeability is the fill material (located in the upper few feet of section) and the native sand and gravel deposits. An upper clay horizon with an upper boundary located approximately 5 feet below grade, provides the least permeable zone observed. This upper clay, composed primarily of extremely low permeability glacial lake deposits, was encountered in all wells drilled and ranges in thickness from approximately 4 to 8 feet. The glacial lake deposits grade downward to coarser grained glacial outwash deposits at approximately 10 feet below grade (refer to well logs of CW-1, MW-1, MW-2, and MW-7). The well log from CW-1 indicates that stratification within the glacial outwash occurs throughout the section to the total depth of 63 feet. Interstratification of glacial lake and outwash deposits was encountered between 10 and 15 feet in MW-10 and between 20 and 25 feet in MW-8. The contacts, based on the well logs, are clearly marked with correlation lines in the cross sections, Figure 4.

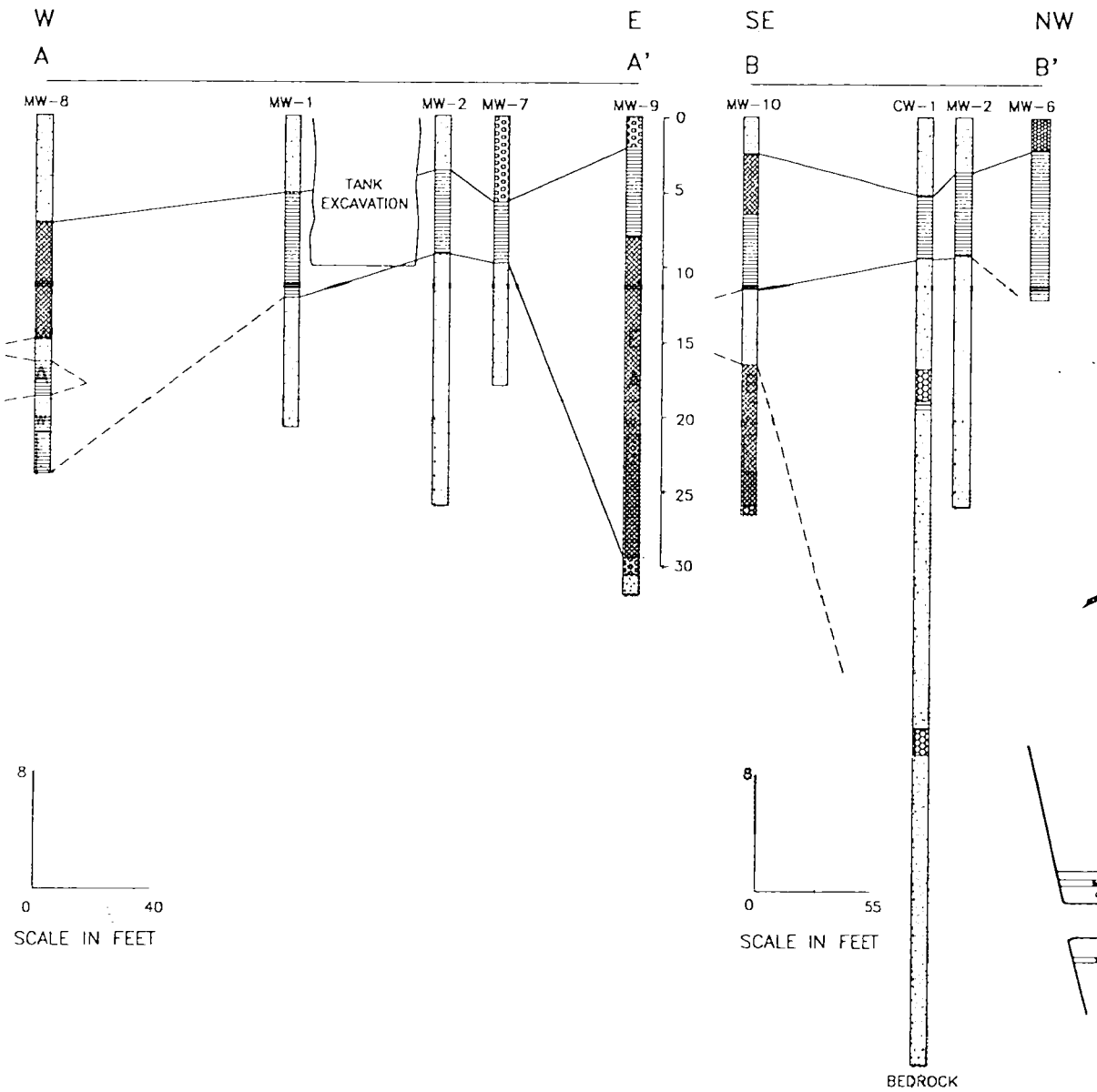
FIGURE 4

GEOLOGIC CROSS-SECTIONS

PROJECT: OSMOSE WOOD PRESERVING, INC.

LOCATION: BUFFALO, NY

JOB NO.: 01110-5470



GLACIAL OUTWASH DEPOSITS AND FILL (UNDIFFERENTIATED)

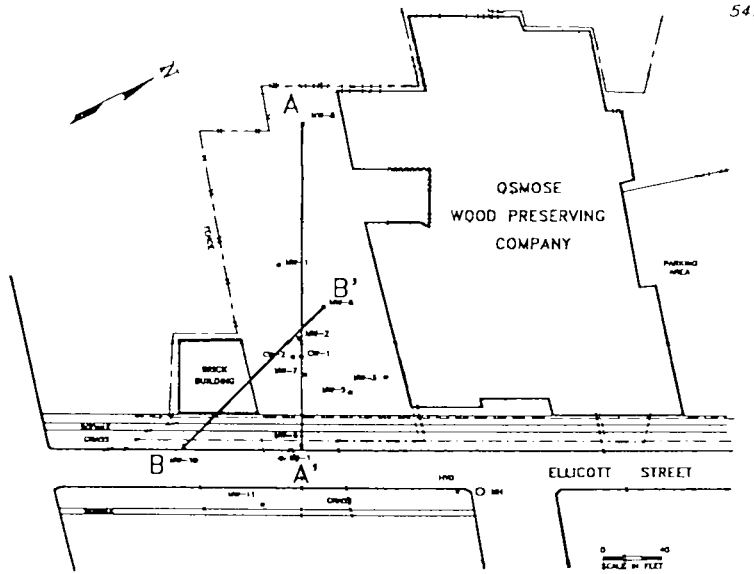
- FINE SANDS
- FINE TO COARSE SANDS WITH SILT/CLAY
- COARSE SANDS AND GRAVEL
- SILTY SANDS WITH GRAVEL

GLACIAL LAKE DEPOSITS

- SILTY SANDS, SILTS, CLAYEY SANDS
- CLAY

6/7/91

5470-X



GROUNDWATER TECHNOLOGY, INC.

4.2 Modified Soil Gas Survey Results

The initial field work step at the Osmose site was the performance of a modified soil gas survey. The results of the modified SGS are summarized below in Table 4-1 and presented on Figure 5. Only 1 of the 18 soil vapor samples, VP-12, contained detectable levels of volatile hydrocarbon vapors. Vapor point VP-12 which measured 19 mg/m^3 was located in the right-of-way on the east side of Ellicott Street adjacent to P & R Wire Forming, Inc.

SGS data collected from the farthest downgradient vapor extraction points, VP-12, VP-13, and VP-14, compares well with PID readings collected during the installation of MW-11. Inspection of the soil boring well log for MW-11 (included in Appendix D), indicated that no VOCs were detected during monitor well installation. Similarly, laboratory analyses from soil samples collected during MW-11 installation (refer to Table 4-4, Section 4.5) indicate non-detectable levels of BTEX and 0.072 mg/kg total PAH (at 4' - 6' below grade) were identified. Soils containing this low level of semi-volatile PAHs would not be expected to be detected using soil gas survey techniques.

A good correlation exists if SGS results from VP-5, VP-8, VP-9,VP-10 and VP-11 are compared with the drilling logs and soils analysis from soils collected during installation of MW-9 and MW-10. Soil gas results, laboratory analysis of soil samples taken from soil samples to a depth of 32' below grade, and PID readings recorded during monitor well installation indicates non-detectable levels of BTEX and VOCs. The same soil samples indicated low levels (0.006 - 0.024 mg/kg) of PAHs, which again would not be detected using a soil gas survey.

Soil gas samples from vapor points VP-4, VP-6 and VP-7, which are located between downgradient monitor well MW-9 and on-site wells CW-1, MW-3, MW-5, and MW-5, and MW-7 indicated non-detectable levels of BTEX and PAHs. These results are justifiable based on the vapor points location between MW-9, which generally exhibited non-detectable contaminant levels as described above, and inspection of the drilling logs from the wells just downgradient of the former tank pit installed in June, 1989 (MW-3, MW-5 and MW-7).

VP — Vapor Point

TABLE 4-1

MODIFIED SOIL GAS SURVEY ANALYTICAL SUMMARY
(MG/M3)

VEP	BENZENE	TOLUENE	ETHYL-BENZENE	XYLENES (total)	BTEX	TOTAL PAHs
VP-1	ND	ND	ND	ND	ND	NA
VP-2	ND	ND	ND	ND	ND	ND
VP-3	ND	ND	ND	ND	ND	ND
VP-4	ND	ND	ND	ND	ND	ND
VP-5	ND	ND	ND	ND	ND	ND
VP-6	ND	ND	ND	ND	ND	ND
VP-7	ND	ND	ND	ND	ND	NA
VP-8	ND	ND	ND	ND	ND	NA
VP-9	ND	ND	ND	ND	ND	NA
VP-10	ND	ND	ND	ND	ND	ND
VP-11	ND	ND	ND	ND	ND	NA
VP-12	ND	4	5	10.3	19	NA
VP-13	ND	ND	ND	ND	ND	NA
VP-14	ND	ND	ND	ND	ND	NA
VP-15	ND	ND	ND	ND	ND	ND
VP-16	ND	ND	ND	ND	ND	ND
VP-17	ND	ND	ND	ND	ND	NA
VP-18*	ND	ND	ND	ND	ND	ND
EQUIP BLANK	ND	ND	ND	ND	ND	ND
TRIP BLANK	ND	ND	ND	ND	ND	ND

* = Blind duplicate of VP-6

ND = Not detected by analytical method

NA = Not analyzed

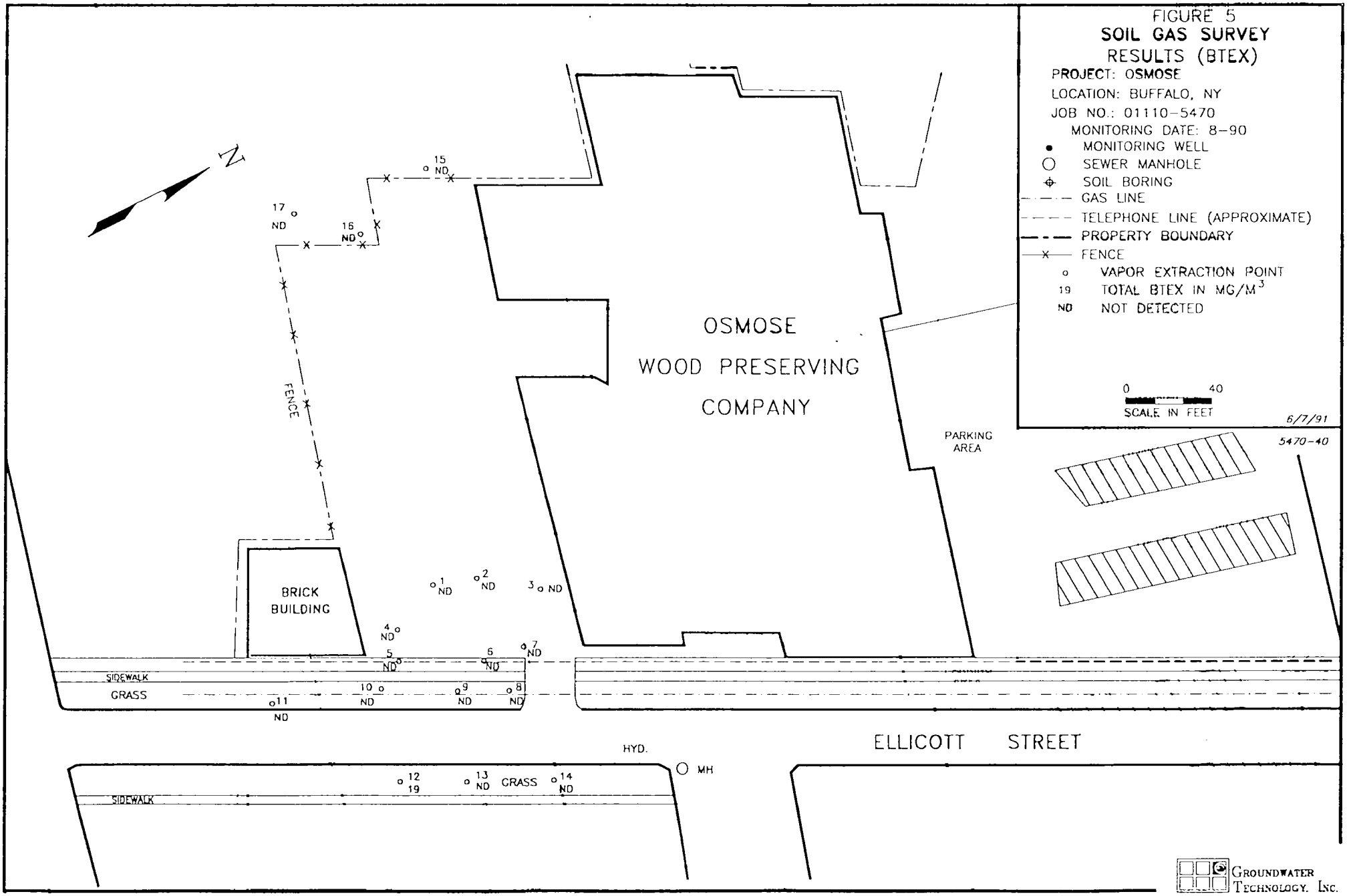


FIGURE 5
SOIL GAS SURVEY
RESULTS (BTEX)
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
MONITORING DATE: 8-90
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
VAPOR EXTRACTION POINT
19 TOTAL BTEX IN MG/M3
ND NOT DETECTED
0 40
SCALE IN FEET
6/7/91
5470-40
N
OSMOSE
WOOD PRESERVING
COMPANY
PARKING
AREA
FENCE
BRICK
BUILDING
SIDEWALK
GRASS
SIDEWALK
GRASS
HYD.
MH
ELLICOTT STREET
15 ND
17 ND
16 ND
1 ND
2 ND
3 ND
4 ND
5 ND
6 ND
7 ND
8 ND
9 ND
10 ND
11 ND
12 19
13 ND
14 ND
GROUNDWATER
TECHNOLOGY, INC.

Soil gas samples from VP-1, VP-2 and VP-3, located immediately downgradient from the former tank pit also showed non-detectable levels of BTEX and total PAHs. These results correlate well with the non-detectable levels of BTEX which were reported on soil samples taken from CW-1 and CW-2 at 6'- 8' below grade.

Based upon the limited results from the volatile compound analysis, eight soil gas samples were chosen for analysis of the semi-volatile PAHs; two upgradient locations (VP-15, VP-16), three downgradient locations (VP-5, VP-6 and VP-10) and three locations adjacent to the former tank pit (VP-2, VP-3, VP-4). A blind duplicate of VP-6 (labeled VP-18) was also analyzed for PAHs for QA/QC purposes along with an equipment blank and trip blank. No PAHs were detected in any of the samples analyzed.

Drilling logs from monitor wells MW-3, MW-5, and MW-7 (previously installed), indicated PID levels from 0 - 1 ppmv from soils above the groundwater table. However, PID measurements taken from soil samples collected below the water table showed that the most highly impacted soils were encountered at 9 feet below grade at or below the contact with a continuous clay layer (soil boring logs from monitor wells MW-3, MW-5, and MW-7 are included in Appendix D). The clay was observed in all wells drilled and appears of sufficient thickness to provide a physical barrier to vapor migration.

In summary, the results of the soil vapor gas survey show that:

- Hydrocarbon vapors were not present across Ellicott Street (east of site) with the exception of VP-12. The levels at VP 12 are not believed to be associated with the Osmose site.
- Hydrocarbon vapors were not present in the right-of ways bordering Ellicott Street.
- Hydrocarbon vapors were not present upgradient (west) of the Osmose facility.
- Contaminants in the area of MW-8 (refer to Section 4.5, Subsurface Soil Sampling Results) were not volatile enough to be detected by the vapor points VP-15 and VP-16.
- A physical barrier (clay layer) may be preventing contaminant vapors from reaching the surface in some areas where they exist on site.
- Soil gas (vapors) do not exist at sufficient levels to be considered as an exposure pathway, and

- Soil gas was not present in sufficient quantity to warrant the use of a field gas chromatograph (GC) rather than a portable PID fitted with a 10.2 eV lamp during drilling activities.

In conjunction with the modified SGS, grab samples of surface soils were collected to test for inorganic metals in the surface soils. The results of the surface grab sample analyses is presented in the following section.

4.3 Surface Soil Results

Surface soil grab samples were collected at each vapor extraction point location (which was not paved over with asphalt or poured concrete) and sent to GTEL Environmental Laboratories in Milford, NH. After review of the laboratory results of the modified SGS, selected soil samples were authorized for analysis. Soil grab samples were analyzed for priority pollutant metals according to Test Methods for Evaluating Solid Waste, SW-846.

Samples authorized for analysis included:

- three (3) upgradient locations (VP-15, VP-16, VP-17)
- three (3) downgradient locations (VP-5, VP-8, VP-13)
- one (1) blind duplicate of VP-13 (labeled VP-19)
- one (1) trip blank
- one (1) field blank

A summary of the soil grab sample analytical results showing the levels of priority pollutant metals detected at each vapor point is presented in Table 4-2.

To evaluate possible inorganic indicators of contamination, the analytical results were compared to background levels of inorganic elements in Eastern U.S. soils. Typical average background values and their ranges are summarized in Table 4-3. Based on the average typical background concentrations, all metals tested at all sampled locations are within average ranges, with the exception of lead at VP-5 (810 mg/kg), VP-8 (610 mg/kg) and VP-15 (820 mg/kg) and zinc at VP-15 (860 mg/kg) and VP - 17(450 mg/kg). VP-15 and VP-17 are located upgradient (west) of the Osmose facility in an area where a former public garage existed (Buffalo Base Zone Map No. 26, September, 1933). The area is currently an abandoned lot which is sometimes used for parking. Given the former land uses in the vicinity of the property, these lead and zinc values are not considered out of the ordinary.

Further discussion of the surface soil grab sampling results is included with the Subsurface Soil Sampling Results in Section 4.5.

TABLE 4-2

SURFACE SOIL GRAB SAMPLE
ANALYTICAL RESULTS SUMMARY

	PRIORITY POLLUTANT METALS (mg/kg)								
METAL	VP-5	VP-8	VP-13	VP-15	VP-16	VP-17	VP-19	TRIP BLANK	FIELD BLANK
Antimony	ND	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	17	36	4.1	18	3.9	3.0	3.8	ND	ND
Beryllium	ND	ND	ND	ND	ND	ND	ND	ND	ND
Cadmium	1.2	1.1	ND	2.5	ND	0.92	ND	ND	ND
Chromium	32	19	14	33	14	11	14	ND	ND
Copper	41	64	52	73	30	27	53	ND	ND
Lead	810	610	200	820	310	410	260	ND	ND
Mercury	0.68	1.9	0.30	1.2	1.1	0.88	ND	1.3	ND
Nickel	11	18	22	21	ND	12	21	ND	ND
Selenium	0.65	0.84	ND	1.1	ND	ND	0.57	ND	ND
Silver	ND	ND	ND	ND	ND	ND	ND	ND	ND
Thallium	ND	ND	ND	ND	ND	ND	ND	ND	ND
Zinc	380	270	140	860	380	450	170	ND	ND

ND = Not detected by analytical method

Priority Pollutant Metals as per Test Methods for Evaluating Solid Waste, SW-846

TABLE 4-3

TYPICAL METAL CONCENTRATIONS IN EASTERN U.S. SOILS*

ELEMENT	RANGE (mg/kg)
Aluminum	7,000 - 100,000
Arsenic **	1 - 50
Barium	15 - 1,000
Berrylium **	3 - 40
Boron	<10 - 150
Cadmium **	<1 - 7.0
Calcium	<100 - 160,000
Chromium	1 - 100
Cobalt	<3 - 70
Copper	<1 - 150
Iron	100 - >100,000
Lead	<7 - 300
Lithium	<5 - 136
Magnesium	50 - 50,000
Manganese	<2 - 7,000
Mercury	0.01 - 3.4
Nickel	<3 - 700
Palladium	Not Reported
Selenium **	0.01 - 2
Sodium	<200 - 15,000
Strontium	<5 - 700
Vanadium	<5 - 300
Zinc	<5 - 400

* Connor, Jon J., and Hansford T. Shacklette; Background Geochemistry of Some Rocks, Soils, Plants, And Vegetables in the Conterminous United States; United States Geological Survey, Professional Paper 574-F; 19

** Baker and Chesnin, 1975; Advances in Agronomy 27:305-374

4.4. Hydrogeologic Evaluation

4.4.1 Regional Hydrogeology

A review of hydrogeologic reports of the area determined that the groundwater circulates through a regional flow system in a north, northwest direction from the Appalachian Uplands to the Erie-Ontario Lowlands, where it discharges near Tonawanda Creek. The glacial deposits recharge the soluble limestone bedrock (ie., Onandaga Limestone) by percolation into joints, fractures and solution channels. The zone of fracturing and solution that follows the upper surface of the soluble limestone rocks has been observed to be in hydraulic continuity with the glacial deposits (LaSala, 1968). Local secondary flow systems exist which discharge to tributary streams.

The transmissivity of the glacial deposits ranges from very low for the lake bed sediments and glacial till to very high (600,000 gpd per foot) for the outwash sand and gravel deposits. The Onandaga Limestone transmissivity varies greatly depending upon the amount of solution channels present. Reported values range from 300 to 25,000 gpd per foot.

4.4.2 Site Hydrogeology

In order to construct a groundwater contour map of the unconsolidated glacial aquifer, top of well casing elevations were surveyed in the field by a Groundwater Technology survey team. All elevations were made relative to a selected arbitrary benchmark of 100 feet. Groundwater contour maps for the two gauging dates (November, 1990 and January, 1991) are included as Figures 6 and 7. The groundwater gradient in the shallow overburden wells was towards the east at approximately 0.3 to 0.4 %. The gradient is towards the east due to the local influence of a small broad V-shaped knoll, just to the west of the site. The monitor well data suggests that the small knoll and associated glacial stratification in the subsurface is exerting hydraulic controls on the groundwater gradient at the site.

The hydrogeologic evaluation of the site suggests that a complex aquifer system exists beneath the site. Groundwater levels in the upper portion of the overburden aquifer range from 7 to 9 feet below grade. Groundwater in the deep overburden well (CW-1) was encountered at approximately 26-27 feet below the surface, indicating that a steep vertical gradient exists within this unit. Possible reasons for the downward vertical gradient observed in CW-1 include:

- Existence of a perched condition in an upper aquifer

- Unrestricted or partially restricted overburden aquifer groundwater drainage into the bedrock (recharge zone). This condition may be due to extensive solution channels in the bedrock.
- A low permeability zone through which CW-1 is screened.
- Well construction differences.

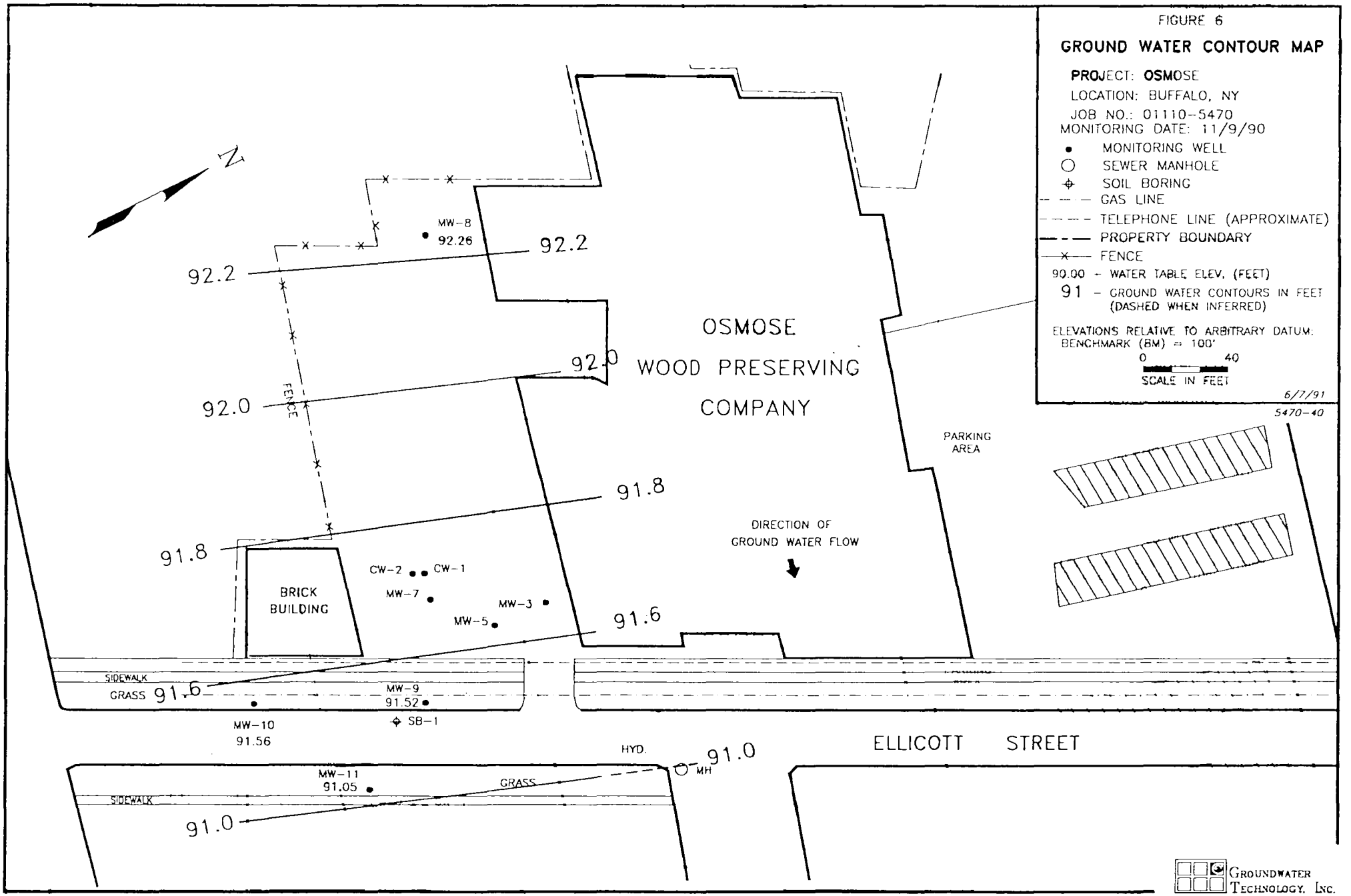


FIGURE 6
GROUND WATER CONTOUR MAP
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
MONITORING DATE: 11/9/90
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
90.00 - WATER TABLE ELEV. (FEET)
91 - GROUND WATER CONTOURS IN FEET (DASHED WHEN INFERRED)
ELEVATIONS RELATIVE TO ARBITRARY DATUM: BENCHMARK (BM) = 100'
0 40
SCALE IN FEET
6/7/91
5470-40
N
OSMOSE WOOD PRESERVING COMPANY
PARKING AREA
BRICK BUILDING
FENCE
MW-8 92.26
MW-7
MW-3
MW-5
CW-2
CW-1
MW-9 91.52
MW-10 91.56
SB-1
MW-11 91.05
HYD.
MH
SIDEWALK
GRASS
DIRECTION OF GROUND WATER FLOW
92.2
92.0
91.8
91.6
91.0
ELLICOTT STREET
GROUNDWATER TECHNOLOGY, INC.

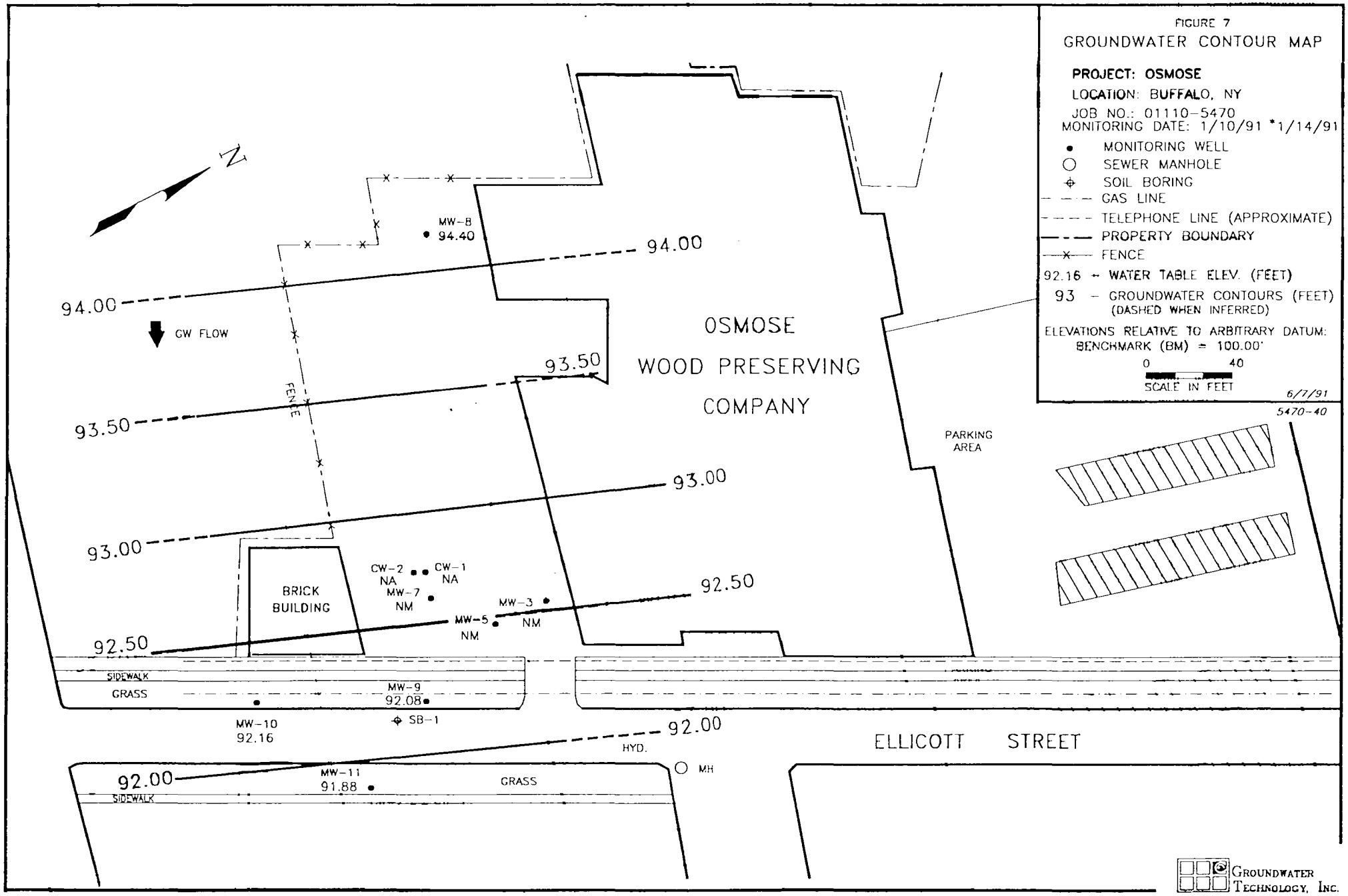


FIGURE 7
GROUNDWATER CONTOUR MAP
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
MONITORING DATE: 1/10/91 *1/14/91
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
92.16 - WATER TABLE ELEV. (FEET)
93 - GROUNDWATER CONTOURS (FEET) (DASHED WHEN INFERRED)
ELEVATIONS RELATIVE TO ARBITRARY DATUM: BENCHMARK (BM) = 100.00'
0 40
SCALE IN FEET
6/7/91
5470-40
N
MW-8
94.40
94.00
94.00
GW FLOW
93.50
93.50
FENCE
OSMOSE
WOOD PRESERVING
COMPANY
PARKING
AREA
93.00
93.00
CW-2
NA
CW-1
NA
MW-7
NM
MW-3
NM
MW-5
NM
BRICK
BUILDING
92.50
92.50
SIDEWALK
GRASS
MW-9
92.08
MW-10
92.16
SB-1
92.00
HYD.
MH
ELLICOTT STREET
92.00
MW-11
91.88
GRASS
SIDEWALK
GROUNDWATER TECHNOLOGY, INC.

Inspection of boring logs and lithologic cross-sections obtained from the United States Department of Interior Geological Survey, Water Resources Division revealed similar groundwater elevation data. Two (2) monitor wells located near the intersection of Best and Main Streets indicated groundwater at approximately 15-26 feet below grade. Additional monitor wells located near the intersection of North Hampton and Main streets indicated groundwater elevations from between approximately 5-15 feet below grade.

Due to the environmental nature of this investigation, all wells except CW-1 were screened through the upper portion of the unconsolidated glacial aquifer to evaluate separate phase and dissolved phase BTEX and PAHs (refer to Monitor Well Screened Intervals; Table 3-2). Well CW-1 was constructed to assess vertical distribution of dissolved-phase PAH and evaluate the general chemical characteristics of the groundwater at depth. Therefore, CW-1 was screened only through a five (5) foot interval at the base of the unconsolidated section. For this reason and because CW-1 was the only well to penetrate the lower portion of the aquifer, thorough evaluation of the causes of the steep vertical gradient could not be conducted.

A preliminary evaluation was performed by review of the stratification and texture of the sediments in CW-1. This review determined the following:

- A potential low permeable, 2 to 4 foot thick, clay layer was observed at 32 feet in CW-1. The lateral continuity of this layer is unknown.
- The texture of the screened interval in CW-1 does not appear to be of low permeability .

Groundwater hardness (as $CaCO_3$) was also evaluated to assess if there were any variations which could be used to assess source areas of the groundwater encountered in the individual wells. A comparison of regional wells that penetrate the surficial unconsolidated deposits and those that penetrate deep unconsolidated deposits (and the Onandaga Limestone) indicates that hardness may exhibit considerable variations due to surface water infiltration (La Sala, 1968). The hardness values obtained at the site (refer to Section 4.6, Table 4-12, Hardness in Groundwater) show a majority of values which are midway between the expected range and therefore are largely inconclusive in determining the groundwater source. The anomalously low values determined for MW-8 (first sampling event) and CW-1 (second sampling event), however, suggest that infiltration of surface water may be occurring at depth in portions of this aquifer.

A conclusion concerning the cause of the vertical gradient can not be determined at this time.

4.5 Subsurface Soil Sampling Results

Soil boring samples were collected and analyzed for PAHs by EPA Method 8310, and Aromatic Volatile Organics by Modified EPA Method 8020 from all boring locations. Soil samples at MW-8, MW-9, and MW-10 were also analyzed for Priority Pollutant Metals (refer to Table 3-1, Subsurface Soil Sample Locations). Sample intervals from 2 to 64 feet were evaluated. Complete copies of the laboratory analytical reports can be found in Laboratory Analysis from the Subsurface Investigation Work Plan, January 1991, published under separate cover. A summary of the PAH and Aromatic Volatile Organics analytical results are presented in Table 4-4.

Table 4-5 presents a summary of the laboratory results for the individual constituent compounds detected and the concentration of those analytes. Included is the analyte name, its relative complexity (carbon#) and depth interval analyzed.

TABLE 4-4

SUBSURFACE SOIL ANALYTICAL SUMMARY
(MG/KG)

MONITOR WELL ID	SAMPLE INTERVAL	TOTAL PAH	TOTAL BTEX	TOTAL H-C
MW-8	2'-4'	500.9	ND	49
	16'-18'	0.005	ND	ND
MW-9	4'-6'	0.006	ND	ND
	10'-12'	ND	ND	4.4
	30'-32'	ND	ND	ND
MW-10	6'-8'	0.042	ND	ND
	10'-12'	0.024	ND	ND
MW-11	4'-6'	0.072	ND	ND
	10'-12'	ND	ND	ND
CW-1	6'-8'	106.42	ND	5.5
	8'-10'	397.55	4.4	170
	30'-32'	0.182	ND	55
	62'-64'	3.33	ND	ND
CW-2	6'-8'	58.54	ND	ND
SB-1	4'-6'	0.026	ND	ND
Blind Dup.*	10'-12'	0.25	ND	ND

ND = Not detected by analytical method

PAH = Sum of Polynuclear Aromatic Hydrocarbons per EPA Method 8310

Total H-C = Sum of BTEX, Misc. Aliphatics (C4–C12), and Misc. Aromatics (C8–C10) per EPA Method 8020

* = Blind duplicate sample from MW-11

TABLE 4–5

VOLATILE AND SEMI–VOLATILE HYDROCARBONS IN SOILS (ug/kg)
EPA Methods 8310 and 8020

ANALYTE	CN*	CW–1 @6'–8'	CW–1 @8'–10'	CW–1 @30'–32'	CW–1 @62'–64'	CW–2 @6'–8'	SB–1 4'–6'	MW–8 @2'–4'	MW–8 @16'–18'	MW–9 @4'–6'	MW–9 @10'–12'	MW–9 @30'–32'	MW–10 @6'–8'	MW–10 @10'–12'	MW–11 4'–6'	MW–11 10'–12'	BLIND DUP**
Naphthalene	C10	8400	77000	ND	160	2100	ND	12000	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthylene	C12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1–Methylnaphthalene	C11	2600	15000	ND	82	350	ND	1200	ND	ND	ND	ND	ND	ND	ND	ND	ND
2–Methylnaphthalene	C11	4100	30000	ND	160	820	ND	4000	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acenapthene	C12	8000	40000	ND	300	1700	ND	3200	ND	ND	ND	ND	ND	ND	ND	ND	ND
Fluorene	C13	6700	29000	12	260	1300	ND	3200	ND	ND	ND	ND	ND	ND	ND	ND	ND
Phenanthrene	C14	22000	62000	36	670	6500	ND	36000	ND	ND	ND	ND	ND	ND	ND	ND	ND
Anthracene	C14	27000	63000	57	720	27000	ND	180000	ND	ND	ND	ND	ND	ND	ND	ND	51
Fluoranthene	C16	9000	28000	21	320	5000	ND	43000	ND	ND	ND	ND	ND	ND	11	ND	30
Pyrene	C16	15000	41000	44	490	10000	14	120000	ND	ND	ND	ND	27	16	28	ND	80
Benzo(a)anthracene	C18	1400	4700	4.4	53	980	2.7	17000	1.8	1.7	ND	ND	3.8	1.7	4.9	ND	14
Chrysene	C18	1100	3700	ND	47	1100	ND	15000	ND	ND	ND	ND	ND	ND	ND	ND	6.0
Benzo(b)fluoranthene	C20	490	1600	3.1	20	530	3.7	14000	1.5	1.7	ND	ND	4.9	2.2	6.6	ND	14
Benzo(k)fluoranthene	C20	290	980	1.5	11	290	1.5	7600	ND	0.88	ND	ND	2.0	ND	2.9	ND	8.3
Benzo(a)pyrene	C20	9.3	97	3.1	21	450	4.1	18000	1.6	1.7	ND	ND	4.6	0.88	6.2	ND	19
Dibenzo(a,h)anthracene	C22	20	120	ND	1.6	53	ND	3700	ND	ND	ND	ND	ND	ND	ND	ND	3.8
Benzo(g,h,i)perylene	C22	260	991	ND	11	280	ND	13000	ND	ND	ND	ND	ND	2.8	6.3	ND	13
Indeno(1,2,3–cd)pyrene	C21	49	360	ND	3.6	88	ND	10000	ND	ND	ND	ND	ND	ND	5.9	ND	10
Benzene	C6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	C6	ND	250	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl benzene	C6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Xylenes (total)	C6	ND	4200	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Misc. Aliphatics	C4–C12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Misc. Aromatics	C8–C10	5500	170000	5500	ND	ND	ND	49000	ND	ND	4400	ND	ND	ND	ND	ND	ND
TOTAL SEMIVOLATILES		106,729	397,548	182	3,330	58,541	26.0	500,900	4.9	5.1	ND	ND	42.3	23.6	71.8	ND	249

ND = Not detected by analytical method

* = Carbon Number

** = Blind duplicate sample from MW–11 @ 10'–12' below grade

4.5.1 Volatile Compounds

As indicated on Tables 4-4 and 4-5, at the 16 soil sampling locations, no BTEX compounds were detected with the exception of cluster well CW-1 at 8' - 10' below grade (4.4 mg/kg). Table 4-5, shows that toluene at 0.250 mg/kg and xylenes (total) at 4.2 mg/kg were the volatile analytes present. The BTEX was detected in the fine to medium sands and silts just below the clay layer which extends from approximately 5-8 feet below grade. Benzene was not detected at any of the sampling locations.

The volatile compounds detected are most likely as a result of either being in the low distillation end of the brushing grade creosote itself, in the hydrocarbon carrier used for creosote, or possibly, as degradation products from the more complex aromatic hydrocarbons found in creosote.

Total hydrocarbons ranged from non-detectable (ND) in SB-1, MW-10, MW-11 and CW-2 to 170 mg/kg at CW-1 at 8' - 10' below grade.

4.5.2 Semi-volatile Compounds

The highest levels of semi-volatile compounds were encountered from 2 to 4 feet below grade in MW-8 (500.0 mg/kg) and from 8 to 10 feet below grade in CW-1 (397.5 mg/Kg). A soil sample taken from MW-8 at 16' - 18' below grade indicated that only low levels (0.005 mg/kg) of PAHs were present at greater depths.

Non-detectable to low levels of total PAHs (<75 µg/kg) were detected in monitor wells MW-9, MW-10, MW-11, and soil boring SB-1. At cluster well CW-1 total PAH concentrations of 397.5 mg/kg and 106.7 mg/kg were detected at 8'- 10' and 6'- 8' below grade, respectively. At 30' - 32' below grade the concentration of total PAHs drops to 0.18 mg/kg. At the soil sample collected from 62' - 64' below grade (directly above bedrock), concentrations increased to 3.3 mg/kg.

As shown in Table 4-5, anthracene, phenanthrene, pyrene, fluoranthene and naphthalene were present in the highest relative concentrations. A manufacturers materials specification sheet for creosote is included in Appendix F. Anthracene, phenanthrene, pyrene, fluoranthene, and naphthalene are all present in creosote at concentrations ranging from 0.5 - > 5.0 %.

The more complex PAHs (carbon numbers C18 - C22) are present in lower relative concentrations than are the low (C10 - C13) and medium (C14 - C16) complexity PAH analytes with the exception of the soil sample collected from MW-8 at 2' - 4' below grade. Inspection of the manufacturers

specifications indicates that low to medium complexity PAHs are present in higher concentrations in the raw materials than the medium to high complexity PAHs.

4.5.3 Metals in Soils

As detailed in Table 3-1, Subsurface Soil Samples Locations (Section 3.4.2), soil samples from various depths at 3 soil boring locations were analyzed for priority pollutant metals. Results of these analyses are presented below in Table 4-6, Metals in Soils.

Comparison of the results from the PPM analyses and typical metals in soils ranges as described by Connor and Shacklette (Table 4-3, Section 4.2), indicates that for all subsurface soils analyzed, all concentrations reported fall within "typical" ranges for Eastern U. S. soils.

When compared with the results for the surface soil grab samples, the following statements can be made:

- soils at depths contain lower concentrations of metals than surface grab samples, and
- lead and zinc, which exhibited slightly elevated levels in selected surface soil grab samples, are not present at elevated levels in any of the soil samples collected from below grade.

TABLE 4-6

METALS IN SOILS (ug/kg)
Priority Pollutant Metals

Sample Date: October, 1990

ANALYTE	MW-8 @2'-4'	MW-8 @16'-18'	MW-9 @4'-6'	MW-9 @10'-12'	MW-10 @6'-8'	MW-10 @10'-12'	FIELD BLANK	TRIP BLANK
Antimony	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	10	2.2	6.6	2.6	16	1.1	ND	ND
Beryllium	4.2	ND	ND	ND	0.43	ND	ND	ND
Cadmium	ND	ND	ND	ND	ND	ND	ND	ND
Chromium	11	5	13	4.3	15	4.6	ND	ND
Copper	ND	7	14	6	15	6.4	ND	ND
Lead	ND	9.7	24	8.5	12	9.9	ND	ND
Mercury	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	5.3	17	4.2	19	5.2	ND	ND
Selenium	ND	ND	ND	ND	ND	ND	ND	ND
Silver	ND	ND	ND	ND	ND	ND	ND	ND
Thallium	ND	ND	ND	ND	ND	ND	ND	ND
Zinc	20	52	60	49	56	66	ND	ND

ND = Not detected by analytical method

4.6 Groundwater Analytical Results

Two (2) groundwater sampling events occurred at the Osmose facility as described in Sections 3.5 of this report. The first groundwater sampling analysis was conducted using non-CLP laboratory protocols. Table 3-3, First Water Sampling Details, (located in Section 3.5) summarizes the groundwater sampling dates, locations and laboratory analyses performed. Groundwater samples from monitor wells MW-8, MW-9, MW-10, MW-11 and CW-1 were sent to GTEL for analysis of volatiles by EPA Method 602, PAHs by EPA Method 610, and hardness. In addition, a water sample from CW-1 was also analyzed for the TCL analytes by non-CLP methods.

Complete copies of the laboratory analytical reports for the first sampling can be found in Laboratory Analyses from the Subsurface Investigation Work Plan, January 1991, published under separate cover. A summary of the results is presented below. Field analysis of pH, temperature, and conductivity were also performed on each groundwater sample and are presented in the following paragraphs.

The second groundwater sampling event, which occurred on January 10 and 11, 1991 was conducted using ASP, Category A protocols. Table 3-4, Second Groundwater Sampling Summary, ASP, Category A, presented in Section 3.3.2.2 of this report summarizes the respective laboratory analyses performed on groundwater samples collected from each monitor well. The results of the analyses are presented below. Laboratory reports are presented in Laboratory Reports from the Subsurface Investigation Work Plan, Second Groundwater Sampling Event, ASP Category A, June, 1991, published under separate cover.

4.6.1 Volatile Compounds

Laboratory results of purgeable aromatics (BTEX and Total Hydrocarbons) for the first groundwater sampling event are summarized in Table 4-7, below. Total BTEX ranged from non-detectable levels in upgradient monitor well MW-8, and downgradient wells MW-10, and MW-11 (reported values are probable laboratory artifacts - refer to nonconformance summary, Laboratory Analyses from the Subsurface Investigation Work Plan, January 1991) to 300 μg/l in MW-9. Total hydrocarbons (Total H-C) concentrations similarly ranged from non-detectable levels in MW-8, MW-10, and MW-11 to 770 μg/l in MW-9.

TABLE 4-7

FIRST GROUND WATER SAMPLING
PURGEABLE AROMATICS SUMMARY (ug/l)

	PURGEABLE AROMATICS (MOD. EPA METHOD 602)					
MONITOR WELL ID	BENZENE	TOLUENE	ETHYL-BENZENE	XYLENES (total)	TOTAL BTEX	TOTAL H-C
MW-8	0.2**	ND	ND	ND	0.2**	0.2**
MW-9	150	76	9	66	300	770
MW-10	0.2**	ND	ND	ND	0.2**	0.2**
MW-11	0.7**	ND	ND	ND	0.7**	0.7**
CW-1	15**	4.9	1.6	12	34	240
D-1*	0.7**	ND	ND	ND	0.7	0.7**

ug/l = micrograms per liter

* Blind duplicate sample of MW-11

** Probable Laboratory artifact; see Sec 1.2 of Nonconformance Summary, GTI Data Validation Report, January, 1991

ND = Not detected by analytical method

Total H-C = Sum of BTEX, Misc. Aromatics (C8-C10), and Misc. Aliphatics (C4-C12)

Presented in Table 4-8 below is a summary of the results from the second groundwater sampling event. Purgeable Halocarbons and Aromatic Volatile Organics by EPA Methods 8010 and 8020, respectively, were run on groundwater samples collected during the second sampling event as opposed to purgeable aromatics by Modified EPA Method 602 (which was run during the first sampling event). Laboratory analytical methods varied from the first groundwater sampling event to conform with the requirements of ASP, Category A protocols.

Total BTEX ranged from non-detectable levels in monitor wells MW-8 and MW-10 to 260 μg/l in groundwater samples collected from monitor well MW-9. Similarly, Total H-C concentrations in the collected groundwater samples ranged from non-detectable levels in monitor wells MW- 8 and MW-10 to 640 μg/l in monitor well MW-9. The aromatic volatile organics data (EPA Method 8020) collected during the second groundwater sampling event correlated very well with the BTEX and Total H-C data reported from the first groundwater sampling event (Modified EPA Method 602).

In addition to the analysis of Aromatic Volatile Organics by EPA Method 8020, analysis of Purgeable Halocarbons was performed by EPA Method 8010. A summary of the total purgeable halocarbons is also provided in Table 4-8, above. Non-detectable levels of purgeable hydrocarbons existed in groundwater samples collected from monitor wells MW-8 (upgradient), MW-9 and MW-10 (immediately downgradient).

Monitor well MW-11 (furthest monitor well downgradient) indicated 0.84 μg/l of total purgeable hydrocarbons and a groundwater sample from cluster well CW-1 indicated 4.31 μg/l. Presented below is Table 4-9 which details the specific halocarbon analytes detected in these wells. In addition, the results of the purgeable halocarbon analyses for the 5 QA/QC samples are also reported on Table 4-9. Inspection of the QA/QC samples indicates that low levels of 1,1,1 - Trichloroethane were found in trip blanks #1 and #3, the field blank, and the equipment blank at concentrations equivalent to the reported values for MW-11 and CW-1. Additionally, 1,2-Dichloroethane was detected in Trip Blank #2. This indicates that these analytes are probably a sampling or analytical artifact.

TABLE 4-8

SECOND GROUND WATER SAMPLING
PURGEABLE HALOCARBONS and AROMATIC VOLATILE ORGANICS SUMMARY

Sample Date: January, 1991

MONITOR WELL ID	PURGEABLE HALOCARBONS by EPA 8010	AROMATIC VOLATILE ORGANICS by EPA 8020 (ug/l)					
		BENZENE	TOLUENE	ETHYL-BENZENE	XYLENES (total)	TOTAL BTEX	TOTAL H-C
MW-8	ND	ND	ND	ND	ND	ND	ND
MW-9	ND	81*	90*	14	74*	260*	640*
MW-10	ND	ND	ND	ND	ND	ND	ND
MW-11	0.84	0.2	ND	ND	ND	0.2	51
CW-1	4.31*	49*	8.5	2.7	25	85*	550*
DW-1**	ND	82*	100*	14	76*	270*	660*
TRIP BLANK-1	0.25	ND	0.8	ND	ND	0.8	0.8
TRIP BLANK-2	0.62	0.2	0.7	ND	ND	0.9	0.9
TRIP BLANK-3	2.6	ND	0.6	ND	ND	0.6	0.6
FIELD BLANK	1.2	ND	0.6	ND	ND	0.6	0.6
EQUIP BLANK	1.1	ND	0.6	ND	ND	0.6	0.6

** Blind duplicate sample of MW-9

* Estimated concentration; see Nonconformance Summary, GTI Data Validation Report

ug/l = micrograms per liter

Total H-C = Sum of BTEX, Misc. Aromatics (C8-C10), and Misc. Aliphatics (C4-C12)

ND = Not detected by analytical method

TABLE 4-9

SECOND GROUNDWATER SAMPLING
PURGEABLE HALOCARBONS SUMMARY (ug/l)
EPA METHOD 8010
Sample Date: January, 1991

ANALYTE	MONITOR WELLS						Trip Blank #1	Trip Blank #2	Trip Blank #3	Field Blank	Equip Blank
	MW-8	MW-9	MW-10	MW-11	CW-1	DW-1					
Chloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromomethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
trans-1,2-Dichloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	3.4	ND	ND	0.62	ND	ND	ND
1,1,1-Trichloroethane	ND	ND	ND	0.84	0.91	ND	0.25	ND	2.6	1.2	1.1
Carbon Tetrachloride	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dichloropropane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,3-Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Dichlorodifluoromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2-Trichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
trans-1,3-Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
2-Chloroethylvinyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromoform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Tetrachloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2,2-Tetrachloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichlorofluoromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

4.6.2 Semi-Volatile Compounds

In addition to volatile compounds, semi-volatile compounds in groundwater were also tested. Laboratory results from the first groundwater sampling event for PAHs by EPA Method 610 are presented in Table 4-10, below.

Total PAHs ranged from non-detectable levels in monitor wells MW-9 and MW-10 to 79.74 µg/l in cluster well CW-1.

PAH analytes detected in concentrations below 1.0 µg/L included:

- Fluorene (0.96 µ/l)
- Fluoranthene (0.86 µ/l)
- Benzo{a}anthrazene (0.16 µ/l)
- Chrysene (0.17 µ/l)
- Benzo{6}fluoranthene (0.22 µ/l)
- Benzo{K}fluoranthene (0.11 µ/l)
- Benzo{a}pyrene (0.22 µ/l)
- Dibenzo{a,h}anthracene (0.054 µ/l)
- Benzo{g,h,i}perylene (0.23 µ/l)
- Indeno{1,2,3-cd} pyrene (0.16 µ/l)

Naphthalene (C-10), 2-Methylnaphthalene (C-11), Anthracene (C-14) and 1-Methlynaphthalene (C-11) were detected in the highest concentrations. Although the more complex (C18-C22) PAH compounds were detected more frequently than these low to medium complexity (C10-C14) PAHs, the lower complexity analytes exists in the groundwater at higher relative concentrations. This is attributed to the lower complexity PAHs possessing higher solubility and lower Koc values, (refer to Section 4.7, Contaminant Characteristics).

Presented in Table 4-11 below is a summary of PAHs in groundwater for the second groundwater sampling event. Again, laboratory analytical methods varied from the first sampling event to conform with the requirements of ASP, Category A protocols.

TABLE 4-10

FIRST GROUND WATER SAMPLING
POLYNUCLEAR AROMATIC HYDROCARBONS SUMMARY (ug/l)
EPA METHOD 610
Sample Date: November, 1990

ANALYTE	CARBON #	MONITOR WELL ID					
		MW-8	MW-9	MW-10	MW-11	CW-1	D-1*
Naphthalene	C10	ND	ND	ND	ND	51	ND
Acenaphthylene	C12	ND	ND	ND	ND	ND	ND
1-Methylnaphthalene	C11	ND	ND	ND	ND	4.0	ND
2-Methylnaphthalene	C11	4.6	ND	ND	ND	8.9	ND
Acenaphthene	C12	ND	ND	ND	ND	3.6	ND
Fluorene	C13	ND	ND	ND	ND	0.96	ND
Phenanthrene	C14	ND	ND	ND	1.1	1.9	ND
Anthracene	C14	ND	ND	ND	ND	5.6	ND
Fluoranthene	C16	ND	ND	ND	ND	0.86	0.29
Pyrene	C16	0.29	ND	ND	0.29	1.6	0.55
Benzo(a)anthracene	C18	ND	ND	ND	0.040	0.16	0.08
Chrysene	C18	ND	ND	ND	ND	0.17	ND
Benzo(b)fluoranthene	C20	0.060	ND	ND	0.049	0.22	0.098
Benzo(k)fluoranthene	C20	0.029	ND	ND	0.026	0.11	0.051
Benzo(a)pyrene	C20	0.061	ND	ND	0.054	0.22	0.12
Dibenzo(a,h)anthracene	C22	ND	ND	ND	ND	0.054	ND
Benzo(g,h,i)perylene	C22	ND	ND	ND	ND	0.23	0.12
Indeno(1,2,3-cd)pyrene	C21	ND	ND	ND	ND	0.16	0.088
TOTAL PAHs		5.04	ND	ND	1.56	79.74	1.48

ug/l = Micrograms per liter

ND = Not detected by analytical method

* = Duplicate sample of MW-11

TABLE 4-11

SECOND GROUNDWATER SAMPLING

POLYNUCLEAR AROMATIC HYDROCARBONS SUMMARY (ug/l)

EPA METHOD 8310

Sample Date: January, 1990

ANALYTE	CARBON #	MONITOR WELL ID						Field Blank	Equip Blank
		MW-8	MW-9	MW-10	MW-11	CW-1	DW-1*		
Naphthalene	C10	ND	7.8	ND	ND	160	6.6	ND	ND
Acenaphthylene	C12	ND	ND	ND	ND	ND	ND	ND	ND
Acenaphthene	C11	ND	ND	ND	ND	5.9	ND	ND	ND
Fluorene	C13	ND	ND	ND	ND	1.3	ND	ND	ND
Phenanthrene	C14	ND	ND	ND	ND	1.3	ND	ND	ND
Anthracene	C14	ND	ND	ND	ND	ND	ND	ND	ND
Fluoranthene	C16	ND	ND	ND	ND	0.36	ND	ND	ND
Pyrene	C16	ND	ND	ND	ND	0.54	ND	ND	ND
Benzo{a}anthracene	C18	ND	ND	0.026	ND	0.068	ND	ND	ND
Chrysene	C18	ND	ND	ND	ND	ND	ND	ND	ND
Benzo{b}fluoranthene	C20	ND	ND	0.03	ND	0.05	ND	ND	ND
Benzo{k}fluoranthene	C20	ND	ND	ND	ND	0.028	ND	ND	ND
Benzo{a}pyrene	C20	ND	ND	0.027	ND	0.047	ND	ND	ND
Dibenzo{a,h}anthracene	C22	ND	ND	ND	ND	ND	ND	ND	ND
Benzo{g,h,i}perylene	C22	ND	ND	ND	ND	ND	ND	ND	ND
Indeno{1,2,3-cd}pyrene	C21	ND	ND	ND	ND	ND	ND	ND	ND
TOTAL PAHs		ND	7.8	0.083	ND	169.59	6.6	ND	ND

ug/l = Micrograms per liter

ND = Not detected by analytical method

* = Duplicate sample of MW-11

Total PAH concentrations in groundwater ranged from non-detectable in monitor wells MW-8 and MW-11 to 169.6 μg/l in cluster well CW-1. The low levels of PAHs detected in MW-11 during the first sampling round were not confirmed by ASP during this sampling event. Sixteen (16) PAH analytes are reported by EPA Method 8310. Of these 16 analytes, 6 were not detected in any of the groundwater samples, 6 were detected in only 1 groundwater sample, and 4 were detected in 2 groundwater samples.
Napthalene and Acenapthene were present in the highest concentrations. (160 μ/l and 5.9μ/l, respectively).

Presented in Table 4-12 below are ambient water quality standards and guidance values as published by the NYS DEC, Division of Water in September, 1990 for toxic and non-conventional pollutants.

A comparison of these standards or guidance values, where provided, with the results of first groundwater sampling events can be summarized as follows:

- MW-8 , MW-11 and cluster well CW-1 exceeded groundwater guidance values for benzo {b}fluoranthene, benzo{k}fluoranthene and benzo{a}pyrene.
- Monitor wells MW-11 and CW-1 exceeded guidance levels for benzo{a}anthracene.
- Cluster well CW-1 exceeded guidance values for chrysene and indeno{1,2,3-cd} pyrene.
- MW-9 exceeded groundwater standards for benzene, toluene, ethylbenzene and xylenes (total).

TABLE 4-12

NYS DEC CLASS GA GROUNDWATER GUIDANCE VALUES AND STANDARDS
(ug/l)

ANALYTE	CARBON #	NYS DEC STANDARD	NYS DEC GUIDANCE VALUE
Naphthalene	C10	10	ND
Acenaphthylene	C12	NA	ND
1-Methylnaphthalene	C11	NA	NA
2-Methylnaphthalene	C11	NA	NA
Acenaphthene	C12	20	NA
Fluorene	C13	NA	50
Phenanthrene	C14	NA	50
Anthracene	C14	NA	50
Fluoranthene	C16	NA	50
Pyrene	C16	NA	50
Benzo{a}anthracene	C18	NA	0.002
Chrysene	C18	NA	0.002
Benzo{b}fluoranthene	C20	NA	0.002
Benzo{k}fluoranthene	C20	NA	0.002
Benzo{a}pyrene	C20	NA	NA
Dibenzo{a,h}anthracene	C22	NA	NA
Benzo{g,h,i}perylene	C22	NA	NA
Indeno{1,2,3-cd}pyrene	C21	ND	0.002

~ ND

ug/l = Micrograms per liter

NA = Not Applicable

When groundwater standards and guidance values are compared to the results of the second sampling event (ASP Category A) the following observations can be made:

- Cluster well CW-1 exceeded the groundwater standard for napthalene.
- MW-10 and CW-1 exceeded guidance values for benzo{a}anthracene, benzo{b}fluoranthene, and benzo{a}pyrene.
- CW-1 exceeded groundwater guidance values for benzo{k}fluoranthene.
- MW-9 exceeded groundwater standards for each BTEX analyte.
- Exceedances of groundwater standards of benzene, toluene and xylenes occurred in CW-1, and
- Water samples from MW-11 exceeded the benzene standard.

The following observations can be made comparing data between the first and second groundwater sampling events:

- Napthalene was detected in the highest concentrations,
- Medium to high complexity PAHs were present in lower concentrations than low complexity PAHs,
- 11 of the 16 PAHs were either not detected or detected in only cluster well CW-1 for the first sampling event; 12 of the 16 for the second event, and
- Acenaphthylene was not detected during either sampling event.

Base/Neutrals and Acid (B/N/A) in a water sample collected from CW-1 were also analyzed by EPA Method 8270 as part of the TCL testing protocol. Laboratory reports have been previously published in the January, 1991 Laboratory Analyses from the Subsurface Investigation Work Plan. Worth noting, no phenolic compounds were detected. Phenolic compounds were not detected because phenol comprises less than 0.1 % by weight of the manufacturers listed hazardous substances and their presence would not be expected from brushing grade creosote or #2 fuel oil.

Toxicological profiles of the PAH analytes are included in Appendix A, Risk Assessment.

4.6.3 Hardness

Hardness values from the first groundwater sampling ranged from 290 mg CaCO3/l in monitor well MW-8 to 1,100 mg CaCO3/l in cluster well CW-1. A hardness value of 1,000 mg CaCO3/l was detected in MW-9. Monitor Well MW-10 and MW-11 contained values ranging from 460 to 510 mg CaCO3/l.

Hardness values from the second groundwater sampling event ranged from 360 mg CaCO3/l at cluster well CW-1 to 720 mg CaCO3/l at monitor well MW-8. Table 4-13 summarizes the hardness results for both sampling events.

Comparative background hardness values for bedrock and stratified glacial deposits were obtained from Groundwater Resources of the Erie-Niagara Basin, Water Resources Commission Basin Planning Report ENB-3, 1968. Values ranged from below 140 mg CaCO3/l in aquifers producing from glacial deposits, to above 1,000 mg $CaCO_3$/l in some of the limestone aquifers of the region. A comparison of regional wells that penetrate the surficial unconsolidated deposits and those that penetrate deep unconsolidated deposits (and the Onondaga Limestone) indicates that hardness may exhibit considerable variations due to surface water infiltration (LaSala, 1968).

As shown in Table 4-13, the hardness values obtained from the groundwater tested at this site vary considerably.

4.6.4 Metals

As described in Section 3.5.2, 2 groundwater sampling events occurred. During the first event, groundwater samples collected from on site cluster well CW-1 and downgradient monitor well MW-11 were analyzed for priority pollutant metals. In addition, 1 groundwater sample was collected from CW-1 and analyzed for TCL analytes which included Total Metals in Water. A summary of the laboratory results are presented in Table 4-14, Priority Pollutant Metals in Water, First Sampling Event and Table 4-15, Target Compound List, Total Metals in Water. Water quality standards and guidance values, as published by the NYS DEC, Division of Water are included in the tables for reference.

TABLE 4-13

HARDNESS IN GROUND WATER

(mgCaCO3/L)

MONITORING WELL ID	1ST SAMPLING EVENT	2ND SAMPLING EVENT
MW-8	290	720
MW-9	1,000	710
MW-10	460	610
MW-11	510	520
CW-1	1,100	360
D-1*	500	NA
DW-1**	NA	710
SAMPLE BLANK	<5.0	NA
TRIP BLANK	<5.0	NA
FIELD BLANK	NA	<5.0
EQUIP. BLANK	NA	<5.0

NA = sample not collected/analyzed

* = Blind duplicate of MW-11

** = Blind duplicate of MW-8

TABLE 4-14

FIRST GROUND WATER SAMPLING

PRIORITY POLLUTANT METALS SUMMARY (ug/l)

EPA METHOD 6010

Sampling Date: November, 1990

ANALYTE	MONITOR WELLS						SAMPLE	TRIP	NYS*	DEC*
	MW-8	MW-9	MW-10	MW-11	CW-1	D-1*	BLANK	BLANK	STD	GUIDANCE
Antimony	NA	NA	NA	ND	ND	ND	ND	ND		3
Arsenic	NA	NA	NA	ND	ND	6.3	ND	ND	25	
Beryllium	NA	NA	NA	ND	ND	ND	ND	ND	1100	
Cadmium	NA	NA	NA	ND	6.7	ND	ND	ND	10	
Chromium	NA	NA	NA	ND	19	ND	ND	ND	50	
Copper	NA	NA	NA	ND	ND	ND	ND	ND	200	
Lead	NA	NA	NA	6.9	ND	ND	ND	ND	25	
Mercury	NA	NA	NA	ND	ND	ND	ND	ND	2	
Nickel	NA	NA	NA	ND	ND	ND	ND	ND		
Selenium	NA	NA	NA	ND	ND	ND	ND	ND	10	
Silver	NA	NA	NA	ND	ND	ND	ND	ND	50	
Thallium	NA	NA	NA	ND	ND	ND	ND	ND		4
Zinc	NA	NA	NA	39	140	34	ND	ND	300	

* Blind duplicate sample of MW-11

NA = Not Analyzed

ND = Not Detected by analytical method

** = Class GA groundwater

Laboratory reports from the first groundwater sampling event for PPM by EPA Method 6010 indicated levels of zinc ranging from 39 µg/l in the groundwater sample from monitor well MW-11 to 140 µg/l in groundwater samples from cluster well CW-1. Cadmium and chromium at 6.7 and 19 µg/l, respectively, were detected in samples collected from on-site cluster well CW-1. Groundwater samples collected from monitor well MW-11 indicated lead at 6.9 µg/l and zinc at 39 µg/l were present. A blind duplicate sample of MW-11 did not confirm the presence of lead in monitor well MW-11. No other metals were detected. Eight (8) of the 13 metals tested were not detected. Of the 5 metals detected, arsenic, cadmium, chromium, lead and zinc, none exceeded NYS DEC groundwater standards or guidance values.

Laboratory reports from the groundwater sample collected from cluster well CW-1 for analysis of TCL analytes are summarized in Table 4-15 along with groundwater standards or guidance values for Class GA groundwater. The laboratory reports are included in Laboratory Analyses from the Subsurface Investigation Work Plan, January 1991. Of the 23 metals tested only iron, lead, manganese and sodium exceeded groundwater standards for Class GA groundwater. Each of the remaining analytes (which possess groundwater standards or guidance values) were detected at concentrations below those levels. The presence of lead at levels above groundwater standards was not confirmed by the results of the PPM analysis by EPA Method 6010 (see Table 4-14, above) or by the results of the second groundwater sampling event performed in January, 1991, under ASP Category A protocols (see below).

The second groundwater sampling event occurred on January 10 and 11, 1991. Groundwater samples were analyzed and reports generated which conformed to the NYS DEC ASP, Category A protocols. Groundwater samples were collected from monitor wells MW-8, MW-9, MW-10, MW-11, and cluster well CW-1. Laboratory reports are included in Laboratory Reports from the Subsurface Investigation Work Plan, Second Groundwater Sampling Event, ASP Category A, June, 1991 published under separate cover. A summary of PPM results is summarized below in Table 4-16.

For all 13 metals analyzed, only lead and zinc were detected; both were detected at levels below groundwater standards.

TABLE 4-15

FIRST GROUNDWATER SAMPLING

TARGET COMPOUND LIST (TCL)

TOTAL METALS IN WATER (ug/l)

Sample Date: Novenber, 1990

ANALYTE	METHOD	CW-1	NYS STD*	GUIDANCE*
Aluminum	6010	2300		
Antimony	6010	ND		3
Arsenic	7060	ND	25	
Barium	6010	270	1000	
Beryllium	6010	ND	1100	
Cadmium	6010	7.3	10	
Calcium	6010	400000		
Chromium	6010	11	50	
Cobalt	6010	ND		
Copper	6010	ND	200	
Iron	6010	7900	500	
Lead	7421	31	25	
Magnesium	6010	160000		35000
Manganese	6010	740	300	
Mercury	7470	ND	2	
Nickel	6010	ND		
Potassium	6010	9200		
Selenium	7740	ND	10	
Silver	6010	ND	50	
Sodium	6010	45000	20000	
Thallium	6010	ND		4
Vanadium	6010	ND		
Zinc	6010	140	300	

ND = Not Detected by analytical method

* Class GA groundwater

TABLE 4-16

SECOND GROUND WATER SAMPLING

PRIORITY POLLUTANT METALS SUMMARY (ug/l)

Sample Date: January, 1991

ANALYTE	MONITOR WELLS						FIELD	EQUIP
	MW-8	MW-9	MW-10	MW-11	CW-1	D-1*	BLANK	BLANK
Antimony	ND	ND	ND	ND	ND	ND	ND	ND
Arsenic	ND	ND	ND	ND	ND	ND	ND	ND
Beryllium	ND	ND	ND	ND	ND	ND	ND	ND
Cadmium	ND	ND	ND	ND	ND	ND	ND	ND
Chromium	ND	ND	ND	ND	ND	ND	ND	ND
Copper	ND	ND	ND	ND	ND	ND	ND	ND
Lead	8.3	10.0	ND	8.2	ND	10.8	ND	ND
Mercury	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	ND	ND	ND	ND	ND	ND	ND	ND
Selenium	ND	ND	ND	ND	ND	ND	ND	ND
Silver	ND	ND	ND	ND	ND	ND	ND	ND
Thallium	ND	ND	ND	ND	ND	ND	ND	ND
Zinc	41.1	104	20.4	51.6	ND	80.3	ND	ND

* Blind duplicate sample of MW-11

NA = Not Analyzed

ND = Not Detected by analytical method

4.6.5 Conductivity, pH, and Temperature

A Groundwater Technology field engineer conducted a field analysis of pH, conductivity, and temperature of the collected groundwater samples. Those field tests were performed as described on Appendix D, Project Specific QA/QC Plan of the Work Plan (Sections 6.3 and 8.0). A Corning PS 15 pH meter was calibrated according to manufacturers instructions and a standard 7.0 pH buffer solution. Specific conductance was measured utilizing a Corning PS 17 conductivity meter calibrated using a 1,000 µmho KCl solution. A standard field thermometer was used to measure temperature of the groundwater samples.

A summary of the field analyses is presented below in Table 4-17. The specific conductance values reported for groundwater samples collected from MW-8 and CW-1 during the first sampling event represent laboratory data (EPA Method 120.1) due to the malfunction of the field meter during sampling activities. Laboratory results of conductance, reported as µmhos/cm were converted to µS for comparison purposes. Conductivity values at the Osmose site were comparable to values reported by the United States Department of Interior Geological Survey for monitor wells installed along Main Street. The Geological Survey listed conductivity values from 240 µS from a monitor well located at the corner of Main and North Streets to 2,600 µS at a well located at the corner of Main and Best Streets. Details of well construction were not available.

pH values ranged from 7.3 in monitor well MW-9 to 8.2 in cluster well CW-1. These values are typical for limestone aquifers. Reported pH values for carbonate aquifers range from 7-8 pH units (Freeze and Cherry, 1979).

TABLE 4-17

CONDUCTIVITY, pH and TEMPERATURE

Sample Dates: November, 1990 / January, 1991

FIELD TEST	MONITOR WELL ID				
	MW-8	MW-9	MW-10	MW-11	CW-1
Conductivity (uS)	990/NS	>999/>999	112/139	122/125	990/134
pH	7.6/NS	7.7/7.3	7.5/7.4	7.6/7.5	7.7/8.2
Temperature (oF)	58/NS	58/50	58/56	57/52	48/46

NS = Not Sampled

58/50 = Results of First Field Test/Results of Second Field Test

4.6.6 Pesticides and PCB's

A groundwater sample collected during the first sampling event from cluster well CW-1 was analyzed for Organochlorine Pesticides and PCBs by EPA Method 8080 as part of the TCL analytes.

No pesticides or PCBs were detected in the groundwater sample.

Laboratory reports are included in the Laboratory Analyses from the Subsurface Investigation Work Plan, previously published under separate cover.

4.7 Contaminant Characteristics

Table 4-18, Chemical Characteristics of PAH Compounds, summarizes the physical properties relating to environmental fate and transport of PAH analytes, the most prevalent compounds detected on site. These physical properties include aqueous solubility, vapor pressure, and the organic carbon/water partition coefficient (K_{oc}).

The vapor pressure of the compounds of concern range from 2.3 X 10^{-1} mmHg for Naphthalene to 9.59 X 10^{-11} for Benzo{k}fluoranthene. Values for the medium to high complexity PAHs are so low as to preclude any significant vaporization. For comparison, the vapor pressure of water at 25° C is 24 mm Hg.

The mobility of the compounds of concern in groundwater is also low. This is reflected in the low solubility and high values of K_{oc} . A good correlation between the relative complexity and the solubility and K_{oc} values exists. As can be seen in Table 4-18, as the complexity of the PAH compound increases, its K_{oc} value increases and solubility decreases.

The mobility of a chemical in groundwater is a function of the partition coefficient, K_d , ,and the fraction of organic carbon in the soil f_{oc}. When K_d exceeds 1, contamination will reside principally in the soil matrix, rather than in the groundwater, and its rate of migration will be retarded. Since f_{oc} is in the range of 1-3% for most soils, K_d for the chemicals of concern are 0.5 for Acenaphthene to 10^5 for Indeno {1,2,3,-cd} pyrene. Acenaphthene was the only PAH analyte where K_d was less than 1.

Evaluation of these properties concludes that, with the exception of acenaphthene, the PAHs detected at this site will be highly absorbed to soil and will be soluble at low concentrations in the groundwater. This conceptual model corrolates well with the distribution of PAHs found on site.

TABLE 4-18

CHEMICAL CHARACTERISTICS OF PAH COMPOUNDS*

COMPOUND	COMPLEXITY (C#)	SOLUBILITY @25oC (ug/l)	Koc	VAPOR PRESSURE mm Hg @ 25oC
Naphthalene	C10	31,000	1.37E+03	2.3E-01
Acenaphthylene	C12	3,930	4.79E+03	2.9E-2 **
1-Methylnaphthalene	C11	27,000	NA	NA
2-Methylnaphthalene	C11	25,000	7.94E+03	NA
Acenapthene	C12	3,700	1.78E+01	1.55E-3
Fluorene	C13	1,400	5.01E+03	1.0E-3 **
Phenanthrene	C14	1,050	1.67E+04	6.8E-4
Anthracene	C14	67	1.97E+04	1.95E-4
Fluoranthene	C16	242	4.17E+04	1.0E-2 **
Pyrene	C16	111	6.90E+04	6.85E-7
Benzo{a}anthracene	C18	11	1.38E+06	1.1E-7
Chrysene	C18	3.3	2.45E+05	6.3E-9
Benzo{b}fluoranthene	C20	1.2	5.50E+05	5.7E-7 **
Benzo{k}fluoranthene	C20	0.55	4.37E+06	9.59E-11
Benzo{a}pyrene	C20	4.0	8.81E+05	5.5E-9
Dibenzo{a,h}anthracene	C22	1.5	1.66E+06	1.0E-10 **
Benzo{g,h,i}perylene	C22	0.26	7.76E+06	1.01E-10
Indeno{1,2,3-cd}pyrene	C22	62	3.09E+07	1.0E-10

* Groundwater Chemicals Desk Reference, Montgomery & Welkom, Lewis Publishers, Inc., 1990.

NA = Information Not Available

** = Vapor Pressure @ 20oC

5.0 SUMMARY OF CONTAMINANT DISTRIBUTION

5.1 Separate Phase

Separate phase hydrocarbons were encountered on site in the unconsolidated glacial aquifer. PVC monitor wells MW-3, MW-5, and MW-7 (installed in June, 1989) are identified as the areas of separate phase hydrocarbon impaction as evidenced by the intermittent occurrence of petroleum product in these wells. Although these wells were historically installed and constructed with PVC and not part of the scope of work detailed in the Work Plan, they were monitored on a regular basis by Osmose personnel. Weekly gauging and bailing of separate phase product, when existing, was conducted. Evacuated product and water is stored in drums until sufficient quantities exist for proper disposal. The horizontal distribution of separate phase product is illustrated in Figure 8. Note that the leading edge of the plume is not believed to extend beyond the property boundaries.

Based on Figure 8, the estimated mass volume of light non-aqueous phase liquid (LNAPL) was calculated to be between 75-150 gallons. This estimate was based on a true product thickness of 0.1 feet determined from baildown/recharge tests conducted on well MW-3 during January and February 1991. Calculations were performed by estimating the total area of phase-separate petroleum, and assuming a 60% formation reduction in apparent product thickness (observed in the impacted well). A porosity value of 0.3 was used for the formation constant (clayey sand), and all values were based on static groundwater table conditions (not effected by pumping).

To illustrate the separate phase product response to natural fluctuations in the water table, depth to water and depth to product in MW-3 were gauged on a daily basis through the period from September 1989 through September, 1990, (refer to Hydrograph, Figure 9). The hydrograph illustrates the total vertical fluctuation of the product layer. The stratigraphic section, as determined from the well log of MW-3, is depicted on the right side of the graph.

During the monitoring period, the separate phase product layer fluctuated an average of two feet vertically and came into continuous contact with the glacial lake (clay) deposits. As product fluctuated through the clays, it became adsorbed into the clay matrix and a thinning of the layer resulted (note recurrent thinning of product layer in Figure 9 during water table rise). Because clay rich soils typically adsorb greater than 60% of separate phase petroleum hydrocarbons, the estimates obtained in the separate phase product volume calculations are considered low. To date, no separate phase petroleum has been encountered in any of the FRP wells installed (including CW-1).

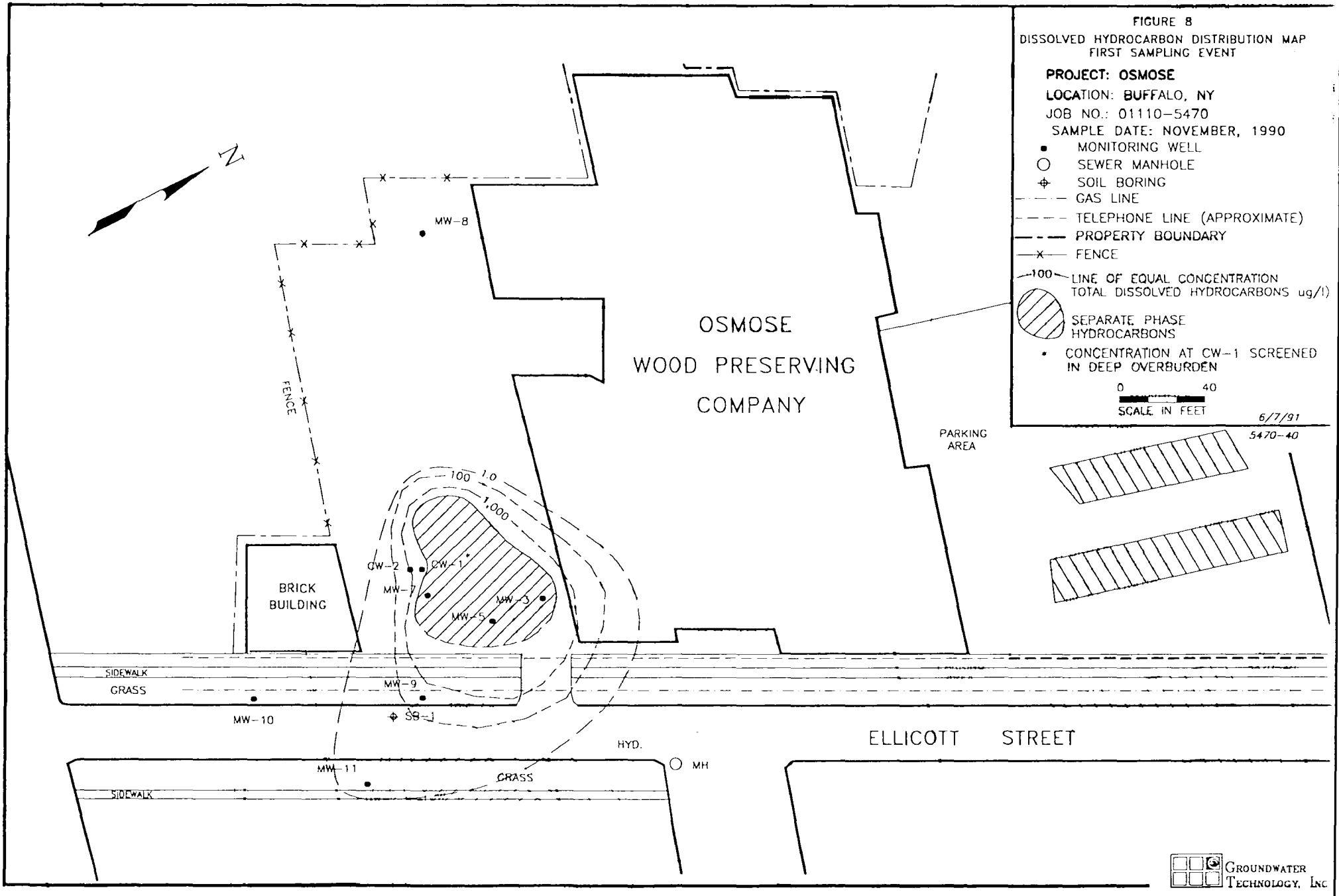
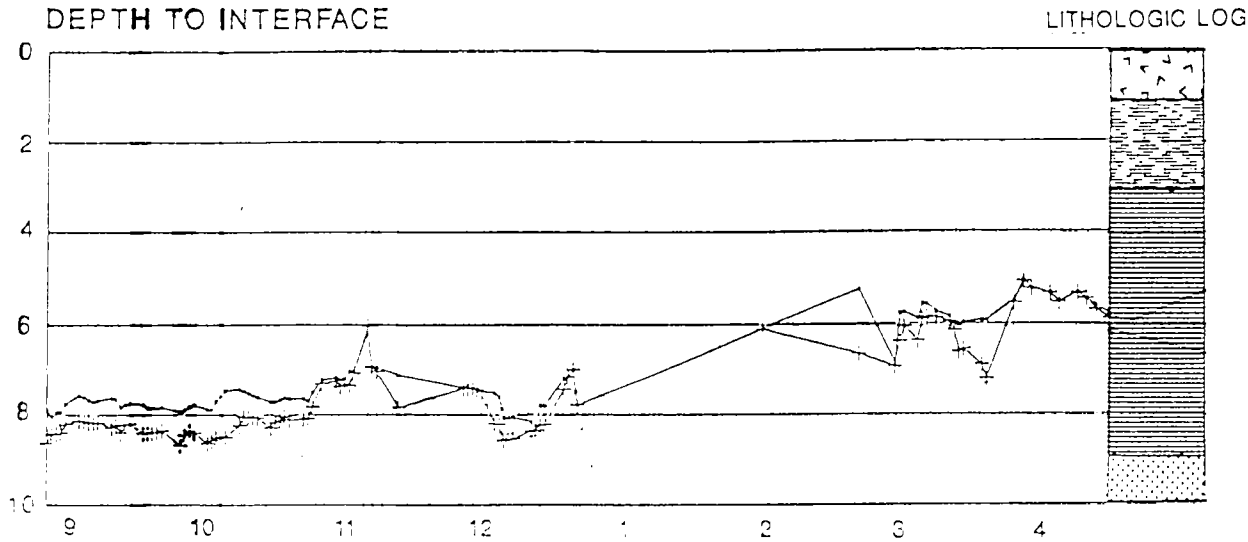


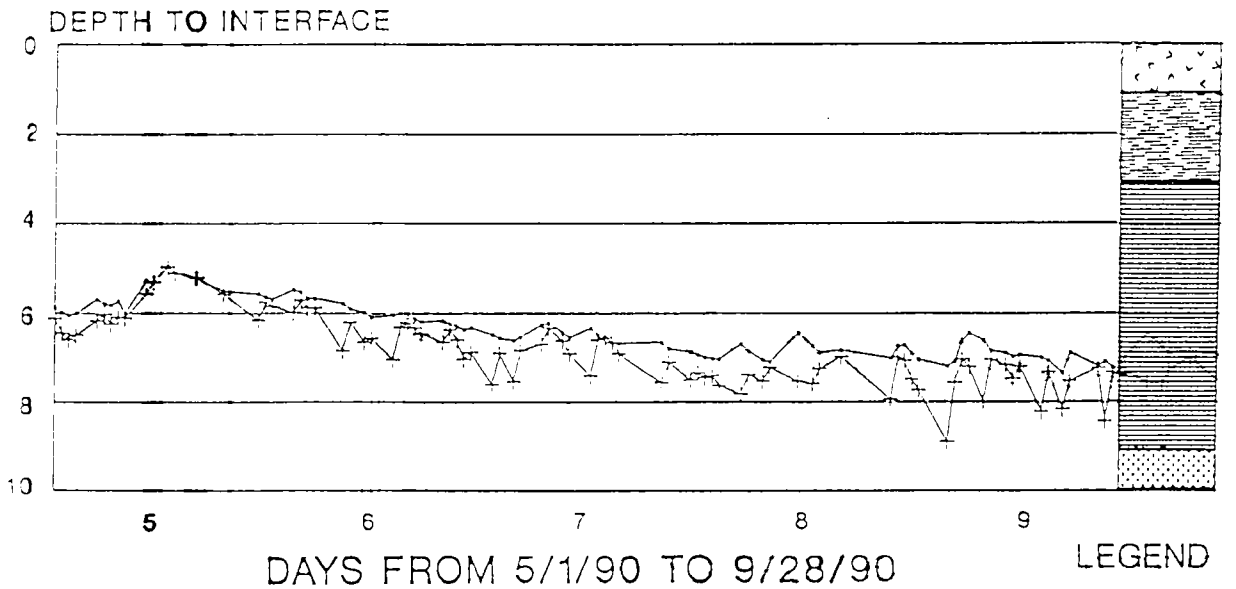
FIGURE 8
DISSOLVED HYDROCARBON DISTRIBUTION MAP
FIRST SAMPLING EVENT
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
SAMPLE DATE: NOVEMBER, 1990
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
100 LINE OF EQUAL CONCENTRATION TOTAL DISSOLVED HYDROCARBONS ug/l)
SEPARATE PHASE HYDROCARBONS
* CONCENTRATION AT CW-1 SCREENED IN DEEP OVERBURDEN
0 40
SCALE IN FEET
6/7/91
5470-40
N
OSMOSE
WOOD PRESERVING
COMPANY
PARKING AREA
FENCE
BRICK BUILDING
MW-8
CW-2
CW-1*
MW-7
MW-3
MW-5
100
1.0
1,000
SIDEWALK
GRASS
MW-9
MW-10
SB-1
HYD.
MH
MW-11
GRASS
SIDEWALK
ELLICOTT STREET
GROUNDWATER TECHNOLOGY, INC

FIGURE 9

OSMOSE WOOD PRESERVING, INC.

HYDROGRAPHS: MW-3





LEGEND

- CLAYEY SAND
- SILT & CLAY
- CLAY
- FINE SAND

GROUNDWATER TECHNOLOGY, INC.

A sample of the LNAPL was collected from MW-3 and MW-7 and its specific gravity calculated. Specific gravity values of 0.9474 and 0.9878 were determined, respectively.

Dense, non-aqueous phase liquid (DNAPL) has been detected on an intermittent basis in PVC monitor wells MW-3, MW-5, and MW-7. When detected, the DNAPL has been bailed out and stored in 55 gallon drums until sufficient quantity exists for proper disposal. Due to the intermittent data, and the nature of DNAPL migration, an estimate of the quantity of DNAPL present could not be calculated.

The following sections discuss the distribution of the adsorbed and dissolved-phase contaminant plumes as determined from the analytical and field data obtained during drilling and sampling activities.

5.2 Adsorbed Phase

The vertical distribution of adsorbed hydrocarbons is illustrated in cross-section in Figure 10, Vertical Hydrocarbon Distribution: Adsorbed Phase. Hydrocarbon compounds show a significant decrease in concentration with depth. In well CW-1, a slight increase in total PAH was detected between 62 and 64 feet below grade. The highest concentrations of PAH compounds were located at or just below the groundwater surface (6' - 10' below grade) with the exception of the discrete area around monitor well MW-8 where the highest PAH levels were located at 2' - 4' below grade. The area in which MW-8 was installed is the location where a gravel bottomed coal bin existed until around 1960. A letter from Osmose which includes a description of the coal bin is enclosed in Appendix E. The coal bin was used for storage of anthracitic coal used to heat the facility prior to its conversion to fuel oil. The PAHs found in this area contained higher relative percentages (20%) of the more complex (C18 - C22) analytes than found at other locations at the site (examples: CW-1 = 3% and CW-2 = 6.4%).

Horizontally, elevated levels of PAHs in soils occur at the surface in the vicinity of MW-8, and in proximity to the former tank pit. These soils contain moderate to high concentrations of PAHs (as shown in Table 4-5; Section 4.5 of this report).

BTEX hydrocarbons were found at cluster well CW-1 (4.4 mg/kg) and nowhere else within the investigation area.

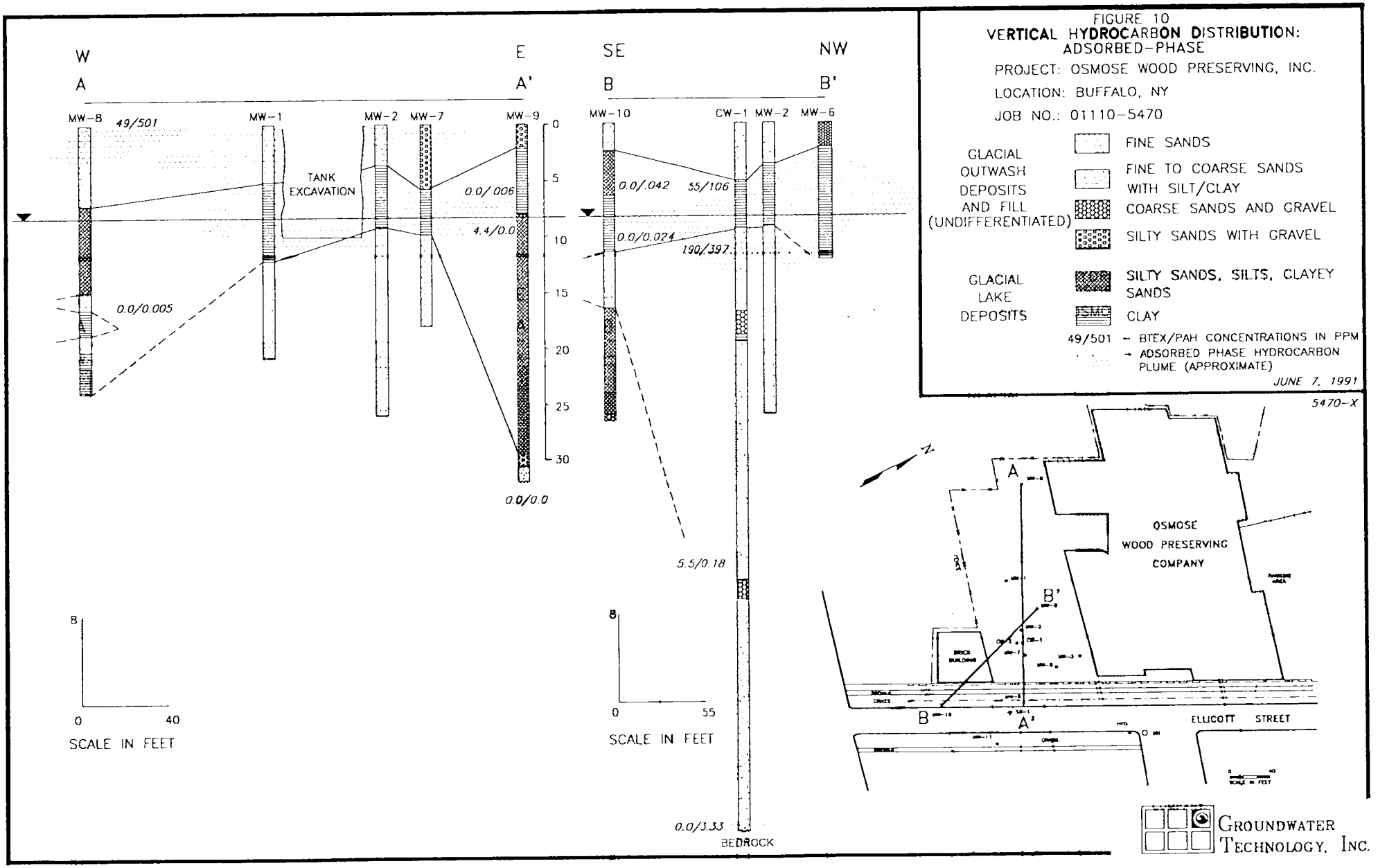


FIGURE 10
VERTICAL HYDROCARBON DISTRIBUTION: ADSORBED-PHASE

PROJECT: OSMOSE WOOD PRESERVING, INC.

LOCATION: BUFFALO, NY

JOB NO.: 01110-5470

GLACIAL OUTWASH DEPOSITS AND FILL (UNDIFFERENTIATED)

- FINE SANDS
- FINE TO COARSE SANDS WITH SILT/CLAY
- COARSE SANDS AND GRAVEL
- SILTY SANDS WITH GRAVEL

GLACIAL LAKE DEPOSITS

- SILTY SANDS, SILTS, CLAYEY SANDS
- CLAY

49/501 – BTEX/PAH CONCENTRATIONS IN PPM

→ ADSORBED PHASE HYDROCARBON PLUME (APPROXIMATE)

JUNE 7, 1991

5470-X

GROUNDWATER TECHNOLOGY, INC.

Low levels (<5 µg/kg) of total PAHs were found in soils in the right-of-way bordering the west side of Ellicott Street. Similar results (<75 µg/kg total PAHs) were found in the soils in the right of way on the east side of Ellicott Street (at MW-11).

Insufficient data exists to accurately calculate the quantities (pounds/gallons) of PAHs adsorbed to the soils, however, a rough calculation indicates approximately 100-300 gallons exists within the soil matrix.

5.3 Dissolved Phase

Two (2) groundwater sampling events occurred as described in Section 3.5.2. During the first event, dissolved BTEX was detected only in the tank pit area (CW-1 at 34 µg/l), and downgradient of the tank pit in MW-9 at 300 µg/l. BTEX was not detected in MW-8, MW-10 or MW-11. Similarly, PAHs were not detected in MW-9 and MW-10. Low concentrations (5.0 µg/l and 1.6 µg/l) of total PAHS were detected in monitor wells MW-8 and MW-11, respectively. Water samples from cluster well CW-1 contained 79.7 µg/l total PAHs. The results of the first groundwater sampling event is presented on Figure 8, Dissolved Hydrocarbon Distribution Map, First Sampling Event. The total dissolved contaminant load was calculated to be approximately 1 gallon.

The results of the second groundwater sampling event are presented in Figure 11. As with the first sampling event, BTEX was not detected in monitor wells MW-8 and MW-10. Monitor well MW-11 contained 0.2 µg/l BTEX (as benzene). BTEX concentrations in water samples collected from MW-9 and CW-1 indicated 260 µg/l and 85 µg/l, respectively, both increasing from the first sampling event.

Total PAHs were not detected in water samples from monitor wells MW-8 and MW-11 during the second sampling event. Monitor wells MW-10 and MW-8 contained 0.08 µ/l and 7.8 µg/l total PAHs respectively. Water samples from cluster well CW-1 were reported at 169.6 µg/l total PAHs, here again, increasing from the first sampling event.

The total dissolved contaminant load for the second sampling event was calculated to be less than 1 gallon.

Comparison of the dissolved PAH levels to maximum solubility concentrations, as presented in Table 4-18 (refer to Section 4.7), shows that most of the PAH analyses are present at less than 5 % of saturation levels. Anthracene, Benzo{b}fluoranthene, and Benzo{k}fluoranthene were present at

8%, 18% and 20 % of their saturation levels, respectively, for the first sampling event. Benzo{g,h,i}perylene was present at .23 μ/l or 88% of saturation.

None of the PAH analyses detected in the second groundwater sampling event were detected above 5 % of saturation concentrations.

Purgeable halocarbons were not detected in any of the groundwater samples analyzed. (Purgeable halocarbons detected are believed to be laboratory artifacts).

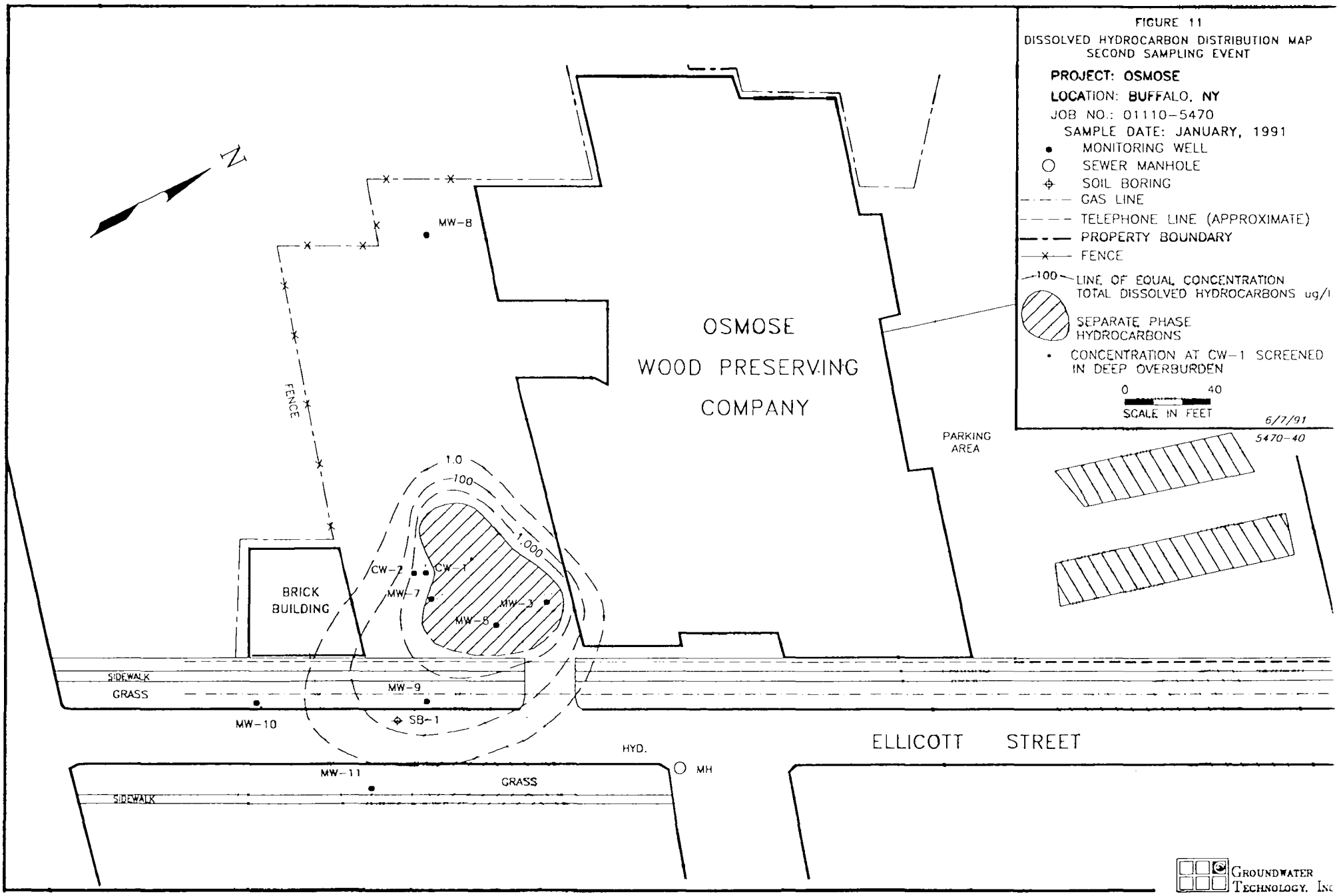


FIGURE 11
DISSOLVED HYDROCARBON DISTRIBUTION MAP
SECOND SAMPLING EVENT
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
SAMPLE DATE: JANUARY, 1991
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
100 LINE OF EQUAL CONCENTRATION TOTAL DISSOLVED HYDROCARBONS ug/l
SEPARATE PHASE HYDROCARBONS
CONCENTRATION AT CW-1 SCREENED IN DEEP OVERBURDEN
0 40
SCALE IN FEET
6/7/91
5470-40
N
OSMOSE
WOOD PRESERVING
COMPANY
PARKING AREA
FENCE
BRICK BUILDING
1.0
100
1,000
CW-2
CW-1
MW-7
MW-3
MW-5
MW-8
MW-9
MW-10
MW-11
SB-1
SIDEWALK
GRASS
SIDEWALK
GRASS
HYD.
MH
ELLICOTT STREET
GROUNDWATER TECHNOLOGY, INC

6.0 HUMAN HEALTH RISK ASSESSMENT

6.1 Introduction

This report has been prepared as part of the assessment of the Osmose Wood Preserving, Inc. site in Buffalo, New York. Specifically, this report applies accepted quantitative risk assessment methodology to evaluate compounds of concern detected in on-site soils and groundwater, and potential exposures to those compounds associated with hypothetical future exposure scenarios, in order to characterize baseline risks associated with a no-action alternative. The results of this baseline analysis have then been applied in conjunction with site-specific environmental conditions to derive risk based clean-up objectives for on-site soils.

6.1.1 Risk Assessment and the Regulatory Process

Over the past ten years, the application of quantitative risk assessment methodology to evaluate issues pertaining to public health has become increasingly widespread. Whereas earliest applications of quantitative risk assessment were concerned with regulation of the use of chemical products, the formalized four-step risk assessment process (hazard identification, dose-response evaluation, exposure assessment, and risk characterization) now plays an integral role in several environmental regulatory programs, including Superfund (CERCLA) and RCRA. For example, the Baseline Risk Assessment for a Superfund site assesses potential risk associated with no further remedial action, while risk assessment within RCRA can be applied to identify contaminant levels which meet requirements of being protective of public health and the environment. The use of risk assessment, with its emphasis on establishing health-protective cleanup levels, becomes especially critical when site conditions, technology, and/or economic factors preclude cleanup to pre-use or background contaminant levels. Extensive efforts at the federal level have been directed toward standardizing risk assessment methodology and its application, resulting most recently in the 2-volume *Risk Assessment Guidance for Superfund* (EPA, 1989a), to accompany the *Exposure Factors Handbook* (EPA, 1989b) and the *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA* (EPA, 1988b). Within the New York regulatory community, quantitative risk assessment has been utilized in a manner generally consistent with developments at the federal level.

6.1.2 Baseline Risk Assessment

The purpose of the baseline risk assessment is to determine, for regulatory purposes, the potential adverse health effects that may be caused by hazardous substance releases or threatened releases

from a site. The baseline risk assessment is part of the overall regulatory evaluation process, the purposes of which are to:

- provide an analysis of baseline risks and help determine the need for action at sites,
- provide a basis for determining levels of chemicals that can remain on-site and still be protective of public health,
- provide a basis for comparing potential health impact of various remedial alternatives, and
- provide a process for evaluating and documenting potential public health threats at sites.

The risk assessment should also summarize uncertainties and methodologies which are relevant to site-specific remedial decision making. This assessment uses EPA methodology for carcinogenic and noncarcinogenic risk assessment.

The risk assessment may be utilized in the remedial decision-making process to:

1. assess the appropriateness of "no-action" remedial alternatives, and
2. provide the New York State Department of Environmental Conservation (NYS DEC) with information upon which to base its decision whether additional response action is necessary at the site.

The particular application of risk assessment in this report is to characterize baseline risks associated with a no-action alternative. Results of the analysis were then utilized in developing risk based soil cleanup objectives.

6.2 Site Background

Osmose Wood Preserving, Inc. (Osmose) is located at 980 Ellicott Street, Buffalo, New York. The site, which Osmose has occupied since approximately 1950, includes research and production operations, as well as the executive and accounting offices for the company. Osmose manufactures a variety of preservatives used in the treatment of wood and lumber products. Raw materials historically stored on site in underground storage tanks (USTs) include brushing grade creosote (stored until 8/89), No. 2 Fuel Oil (until 8/89), mineral spirits (until 1986), an isopropyl alcohol and diacetone mixture (until 1984), and coal tar (until 1964). Between 1910 and 1950 several operations were located either on or adjacent to the site, including several automotive repair shops, a florist, a sheet metal works, a plumbing supplier, and a letter service company (GTI, 1990- Subsurface Investigation Work Plan for Osmose Wood Preserving, Inc., Buffalo, N.Y., June 7, 1990).

The Osmose site is located in a mixed residential and commercial area. The only other manufacturer in the immediate area is P&R Wire Forming, Inc., located southeast of Osmose across Ellicott Street. Bordering the site are residential neighborhoods to the north and east, commercial operations on Main Street to the west, the Summer/Best Metro Rail subway station on Best Street southwest of the site, and a Niagara Mohawk substation located on Best Street south of the Osmose site (GTI, 1990).

The site, located approximately 1.25 miles east of the Niagara River and 1.75 miles northeast of Lake Erie, is flat with a slight gradient to the southeast. Soil borings in the southern portion of the site indicate that the uppermost 63 feet of unconsolidated materials are characterized as glacial lake deposits, comprised primarily of stratified clays, silts, and fine sands. The bedrock beneath the glacial material is the Onondaga Limestone, composed of a hard cherty Silurian dolostone. Gauging data from the southern portion of the site in the unconsolidated glacial aquifer material indicate that the local groundwater gradient is approximately 0.3 - 0.4% to the east. The regional gradient is expected to flow westward toward Lake Erie and the Niagara River (GTI, 1990). Additional hydrogeological information for the site is located in Section 4.4.

As part of a program to upgrade their chemical storage system, Osmose removed three underground storage tanks (USTs) in August 1989 and replaced them with a state-of-the-art above-ground system. Groundwater sampling during this upgrade program indicated the presence of phase separated product which was immediately reported to the New York State Department of Environmental Conservation (NYS DEC). The site was subsequently included on the New York State Registry of Inactive Hazardous Waste Disposal Sites (GTI, 1990). The source of compounds of concern identified on site is presumably historical small scale spillage during filling of the storage tanks and/or small leaks in the transfer piping. There has been no indication of release from storage tanks themselves. The primary receiving medium for released chemicals has been on-site soil. Data collected to date indicates low level of contamination of on-site groundwater.

Groundwater Technology Inc. (GTI) has conducted additional site investigations to further characterize the site. Results of these recent characterization studies are included in other sections of this site assessment report. In addition, GTI has installed and is now operating a bioremediation soil treatment cell (biocell) in the area of the former tank farm immediately south of the primary production and office building.

In this report three different areas are addressed relative to soil conditions and potential exposure to compounds of concern in the soil. These areas include the bioremediation cell (biocell), on-site

locations east and west of the biocell (on-site), and off-site locations along Ellicott Street adjacent to the site (off-site). These three areas were selected to reflect potential exposure events and exposure conditions corresponding to distinct locations. Relative to groundwater, exposure and risk evaluations were conducted based on data from shallow monitoring wells. Deep groundwater conditions were not addressed in the quantitative exposure and risk evaluations, because there is no indication of either current or future exposure to this groundwater.

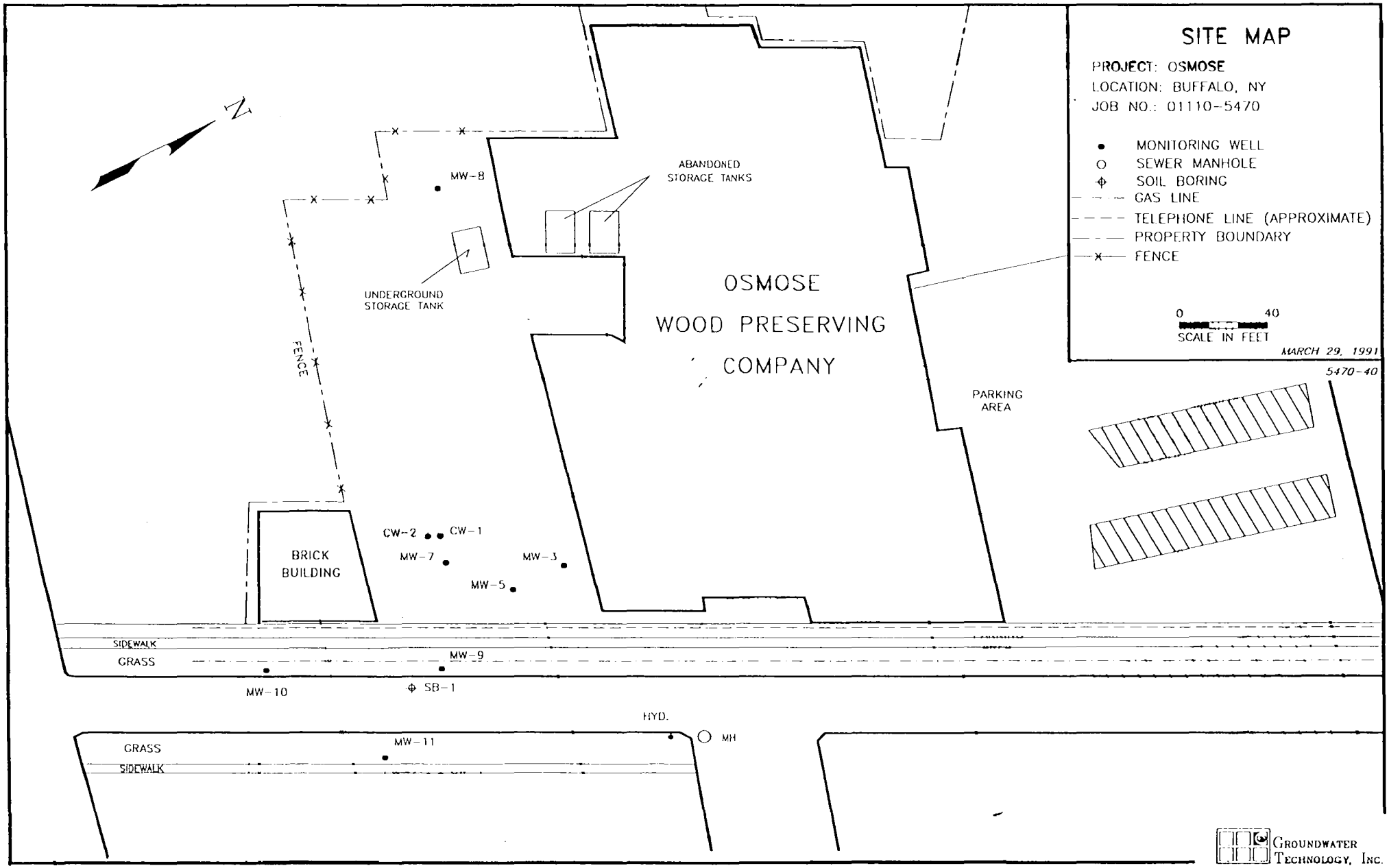
6.3 Analytical Data Review and Interpretation

Analytical data were generated for the Osmose site based on soil and groundwater samples collected between August 1990 and January 1991 from on-site areas immediately south of the existing building complex and from off-site areas immediately downgradient of the site along Ellicott Street. Laboratory reports are published under separate cover and are summarized in Tables 6-1 through 6-7 in this section.

Soil

Data were collected for volatile organic compounds in soil gas from samples collected in August 1990 at on-site (VP-1 through VP-7) and off-site (VP-8 through VP-18) vapor point locations, and in October 1990 at selected monitoring well boring locations. Results of analyses indicated non-detect values for all of the vapor point samples except for 19.3 mg/kg (ppm) total BTEX (benzene, toluene, ethylbenzene, total xylenes) at VP-12 and 4.4 mg/kg total BTEX at CW-1 (8-10 feet) (Tables 6-1 and 6-2, Summary of Volatiles Data and Appendix A-1, Specific Sample Data). Envirologic Data concluded that volatile organic compounds are present in the soil only in scattered locations and at very low concentrations; therefore, they were not selected for quantitative exposure and risk assessment.

Analyses for inorganic compounds were conducted for soil samples collected in August 1990 at vapor point locations and at selected monitoring well borings in October 1990. As indicated in Table 6-3, analytical results for metals in soil were compared to background levels developed by Kingsbury and Ray (1986). Maximum concentrations of all inorganic compounds were within background ranges with the exception of zinc at four vapor point locations and lead at six vapor point locations. Even these values are probably not unusual for soils in an area of historical industrial use. The reported lead concentrations do not exceed the range of 500 to 1,000 mg/kg currently used as an interim guidance for establishing lead cleanup levels at Superfund sites (EPA, 1989c). Envirologic Data concluded that concentrations reported for metals in the soil are generally low as compared with background levels and that there are no site-related activities


SITE MAP
PROJECT: OSMOSE
LOCATION: BUFFALO, NY
JOB NO.: 01110-5470
MONITORING WELL
SEWER MANHOLE
SOIL BORING
GAS LINE
TELEPHONE LINE (APPROXIMATE)
PROPERTY BOUNDARY
FENCE
0
40
SCALE IN FEET
MARCH 29, 1991
5470-40
N
OSMOSE
WOOD PRESERVING
COMPANY
ABANDONED
STORAGE TANKS
UNDERGROUND
STORAGE TANK
FENCE
PARKING
AREA
BRICK
BUILDING
MW-8
CW-2
CW-1
MW-7
MW-3
MW-5
SIDEWALK
GRASS
MW-9
MW-10
SB-1
HYD.
MH
MW-11
GRASS
SIDEWALK
GROUNDWATER
TECHNOLOGY, INC.

which might be a source of metal contaminants. For these reasons, metals were not selected for quantitative exposure and risk assessment.

Analyses for semi-volatile organic compounds were conducted for five composite samples from the biocell in June 1990 and for samples from selected on-site (non-biocell) and off-site monitoring well locations in October 1990. A number of polynuclear aromatic hydrocarbons (PAHs) were detected in all of the biocell and on-site (non-biocell) samples. Samples from off-site locations showed low concentrations of eight PAHs (Table 6-4, Summary of PAH data; Appendix A-1 , Specific Sample Data). Based on the prevalence of PAHs in biocell and on-site samples, and occurrence of PAHs to a lesser extent in off-site samples, 18 different PAHs were selected for quantitative exposure and risk assessment.

Groundwater

Several analyses for volatile organic compounds in groundwater were performed. Results indicate BTEX present in the shallow aquifer in one off-site well (MW-9) and one on-site well (MW-8). Reported concentrations are below USEPA Drinking Water Standards (MCLs) for individual BTEX compounds in the on-site well and for BTEX in the off-site well. Results from monitoring wells in the deep aquifer indicate one on-site well (CW-1) with concentrations of only benzene in excess of the MCL. Two monitoring wells (MW-9 and CW-1) indicate concentrations of individual BTEX compounds in excess of the more stringent NYS DEC Groundwater Standards. All groundwater quality results are summarized in Table 6-5.

For the purposes of the quantitative, site-specific risk assessment, only future exposure to the shallow groundwater is reasonable. No current exposures are occurring (see Section 6.5.2). Therefore, only analytical data from shallow monitoring wells are applied in this assessment.

Relative to total metals in groundwater, analyses of samples collected in November 1990 and January 1991 indicated the presence of five metals in specific monitoring wells at concentrations below MCLs and NYS DEC Groundwater Standards (see Table 6-6, Summary of Metals in Groundwater; Appendix A-1, Specific Sample Data). Inorganic compounds in groundwater are not addressed quantitatively in this assessment.

Groundwater was tested for purgeable halocarbons in January 1991. Results are reported in Appendix A-1 of this report. Detected concentrations are of negligible significance, and are not addressed in the quantitative assessment.

Groundwater was tested for pesticides and PCBs in November 1990. No compounds were detected.

Groundwater samples were analyzed for polynuclear aromatic hydrocarbons (PAHs). Results indicated non-detect levels in several wells and low concentrations in some shallow and deep monitoring wells. Maximum concentrations of three carcinogenic PAHs were marginally greater than their respective MCLs and five carcinogenic PAHs exceeded the NYS DEC Groundwater Standards or Guidance Values (see Table 6-7). MCLs for non-carcinogenic PAHs are not available; NYS DEC Groundwater Standards or Guidance Values were not exceeded, except for naphthalene in monitoring well CW-1. PAH results are summarized in Table 6.3-7; specific sample data is presented in Appendix A-1. Three of the detected PAHs in deep monitoring well CW-1 exceed their respective U.S.-EPA drinking water standards (MCLs) for the November 1990 sampling event; but concentrations detected in the subsequent analyses of this well for January were below their respective standards. None of the concentrations detected in the shallow monitoring wells exceeded their respective MCL values. Envirologic Data concluded that the data indicated restricted occurrence of PAHs in both on-site and off-site monitoring wells. PAH data for shallow wells for both November 1990 and January 1991 sampling events was used in a quantitative assessment of potential exposure to a hypothetical utility repair worker.

In summary, compounds of concern to be carried through the quantitative exposure and risk assessments include 18 different PAH compounds for the hypothetical occupational exposure to soil, in addition to 16 PAHs and BTEX compounds for hypothetical occupational exposure to shallow groundwater. Data points from five composite soil samples for June 1990 analyses were included for the biocell soil assessment, data from shallow (10 feet or less) samples from three on-site (non-biocell) locations (MW-8, CW-1, and CW-2) were included for the on-site (non-biocell) soil assessment, and data from shallow (10 feet or less) samples from three off-site locations (MW-9, MW-10, MW-11) were included for the off-site soil assessment. Relative to shallow groundwater, data from four shallow wells (MW-8, MW-9, MW-10, MW-11) for the November 1990 and January 1991 sampling events were included for the shallow groundwater assessment.

6.4 Hazard Identification

The toxicity assessment is the component of the risk assessment process which qualitatively and quantitatively evaluates the potential for chemical compounds to induce adverse health effects in exposed populations. The toxicity assessment incorporates a two-step analysis originally described by the National Academy of Sciences and the EPA (NAS, 1983; EPA, 1986a): hazard identification

Table 6-1
Summary Data for Volatiles in Soil Gas

	Soil	
Chemical	Frequency of Detection	Range (ug/kg)
Benzene	0/18	--
Toluene	1/18	4
Ethyl Benzene	1/18	5
Xylenes (total)	1/18	10.3

Table 6-2
Summary Data for Volatiles in Soil

	Soil	
Chemical	Frequency of Detection	Range (ug/kg)
Benzene	0/15	--
Toluene	1/15	0.25
Ethyl Benzene	0/15	--
Xylenes (total)	1/15	4.2
Misc. Aromatics (C4-C12)	0/15	--
Misc. Aromatics (C8-C10)	5/15	4.4 - 170

Table 6-3
Summary Data for Metals in Soil

	Soil		
Chemical	Frequency of Detection	Range (mg/kg)	Background Levels (1) (mg/kg)
Arsenic	13/13	1.1 - 36 J	0.1 - 40
Beryllium	2/6	0.43 - 4.2	NA
Cadmium	4/7	0.92 - 2.5	0.01 - 7
Chromium	13/13	4.3 - 33	50 - 170
Copper	12/13	6 - 73	2 - 100
Lead	12/13	9.7 - 810J	2 - 200
Mercury	6/7	0.3 - 1.9	<0.01 - 4.6
Nickel	11/13	0.68 - 22	10 - 40
Selenium	4/7	0.57J - 1.1J	0.1 - 2
Zinc	13/13	20 - 860	10 - 300

(1) - Background levels cited for U.S. soils (Kingsbury & Ray, 1986)

Table 6-4
Summary Data for PAH's in Soil

Chemical	Biocell Frequency of Detection	Biocell Range (ug/kg)	On-Site Frequency of Detection	On-Site Range (ug/kg)	Off-Site Frequency of Detection	Off-Site Range (ug/kg)
NONCARCINOGENS						
Acenaphthene	5/5	52000 - 280000	5/7	ND - 40000	0/8	ND
Anthracene	5/5	7500 J - 28000	6/7	ND - 180000	0/8	ND
Benzo(g,h,i)perylene	0/4	ND	5/7	ND - 13000	1/8	ND - 6.3
Dibenzofuran	5/5	32000 - 190000	Not tested		Not tested	
Fluoranthene	4/5	39000 - 58000	6/7	ND - 43000	1/8	ND - 11
Fluorene	5/5	15000 - 60000	6/7	ND - 29000	0/8	ND
1-Methylnaphthalene	0/4	ND	5/7	ND - 15000	0/8	ND
2-Methylnaphthene	5/5	22000 - 140000	5/7	ND - 30000	0/8	ND
Naphthalene	5/5	10000 - 77000	5/7	ND - 77000	0/8	ND
Phenanthrene	5/5	37000 - 140000	6/7	ND - 62000	0/8	ND
Pyrene	4/5	26000 - 41000	6/7	ND - 120000	4/8	ND - 28
2-Nitroaniline	1/5	36000	Not tested		Not tested	
CARCINOGENS						
Benzo(a)anthracene	5/5	7700 J - 12000	7/7	1.8 - 17000	5/8	ND - 4.9
Benzo(a)pyrene	0/5	ND	7/7	1.6 - 18000	4/8	ND - 6.2
Benzo(b)fluoranthene	1/5	7300	7/7	1.5 - 14000	5/8	ND - 6.6
Benzo(k)fluoranthene	0/5	ND	6/7	ND - 7600	4/8	ND - 2.9
Chrysene	0/5	ND	5/7	ND - 15000	0/8	ND
Dibenzo(a,h)anthracene	0/5	ND	5/7	ND - 3700	0/7	ND
Indeno(1,2,3-cd)pyrene	0/5	ND	5/7	ND - 10000	2/8	ND - 5.9

J - Estimated Value
ND - Not Detected

Table 6-5
Summary Data for BTEX in Groundwater

	Groundwater				
Chemical	Frequency of Detection	Range (ug/L)	MCL** (ug/L)	NYS DEC Standard (ug/L)	NYS DEC Guidance Value (ug/L)
Benzene	7/10	0.2 - 150	5	ND	NA
Toluene	5/11	4.1J - 95	1000	5	NA
Ethyl Benzene	4/11	1.6 - 76	700	5	NA
Xylenes (total)	5/11	4.1J - 75	10000	5	NA
Misc. Aromatics (C4-C12)	2/10	68 - 70			
Misc. Aromatics (C8-C10)	5/10	51 - 470			

** MCL = Maximum Contaminant Level (US-EPA Drinking Water Regulation)
NYS DEC: Class GA Groundwater Standards and Guidance Values.
NA = Not available

Table 6-6
Summary Data for Total Metals in Groundwater

	Groundwater				
Chemical*	Frequency of Detection	Range (ug/L)	MCL** (ug/L)	NYS DEC Standard (ug/L)	NYS DEC Guidance Value (ug/L)
Arsenic	1/7	6.3	50	25	NA
Cadmium	1/8	6.7	10	10	NA
Chromium	1/8	19	50	50	NA
Lead	5/7	6.9 - 10.8	50	25	NA
Zinc	7/8	20.4 - 140	5000	300	NA

* Only compounds detected at least once are included in the summary.
** MCL = Maximum Contaminant Level (US-EPA Drinking Water Regulation)
NYS DEC: Class GA Groundwater Standards and Guidance Values.
NA = Not available

Table 6-7
Summary Data for PAH's in Groundwater

Chemical*	Groundwater Frequency of Detection	Range (ug/L)	MCL** (ug/L)	NYS DEC Standard (ug/L)	NYS DEC Guidance Value (ug/L)
NONCARCINOGENS					
Acenaphthene	2/10	3.6 - 5.9	NA	20	NA
Anthracene	1/10	5.6	NA	NA	50
Benzo(g,h,i)perylene	2/10	0.16 - 0.23	NA	NA	NA
Fluoranthene	2/10	0.2 - 0.86	NA	NA	50
Fluorene	2/10	0.96 - 1.3	NA	NA	50
1-Methylnaphthalene	1/5	4	NA	NA	NA
2-Methylnaphthene	2/5	4.6 - 5.9	NA	NA	NA
Naphthalene	3/10	7.2 - 160	NA	10	NA
Phenanthrene	3/10	0.71 - 1.9	NA	NA	50
Pyrene	4/10	0.29 - 1.5	NA	NA	50
CARCINOGENS					
Benzo(a)anthracene	4/10	0.026 - 0.16	0.1	NA	0.002
Benzo(a)pyrene	5/10	0.027 - 0.22	0.2	ND	NA
Benzo(b)fluoranthene	5/10	0.03 - 0.22	0.2	NA	0.002
Benzo(k)fluoranthene	4/10	0.028 - 0.11	0.2	NA	0.002
Dibenzo(a,h)anthracene	1/10	0.054	0.3	NA	NA
Indeno(1,2,3-cd)pyrene	2/10	0.055 - 0.16	0.4	ND	0.002

* Only compounds detected at least once are included in the summary.
** MCL = Maximum Contaminant Level (US-EPA Drinking Water Regulation)
NYS DEC: Class GA Groundwater Standards and Guidance Values.
NA = Not available

and dose-response assessment. Hazard identification is the process of characterizing the nature and strength of the evidence of causation between exposure to a chemical agent and the induction of adverse health effects. Where health effects have been observed in humans or experimental animals, the dose-response assessment determines the quantitative relationship between the dose of the agent and the incidence of adverse effects. The end result of the dose-response assessment is the derivation of toxicity values which are used in the risk characterization step to predict the likelihood of adverse effects in populations at site-specific exposure levels. In the toxicity assessment, carcinogenic and noncarcinogenic endpoints are evaluated separately.

6.4.1 Noncarcinogenic Health Effects

Evaluation of noncarcinogenic effects is based on comparison of an estimated daily exposure level to an allowable daily exposure level, termed most often as the Reference Dose (RfD). The RfD is based on the assumption that thresholds or protective mechanisms exist for noncarcinogenic effects which must be overcome before adverse effects are observed. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human subpopulation (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (HEAST, 1991). Typically, the RfD is derived by applying one or more "modifying" or "uncertainty" factors to a No-Observed-Adverse-Effect-Level (NOAEL) or Lowest-Observed-Adverse-Effect-Level (LOAEL) from animal or human studies (HEAST, 1991). An RfD, expressed in units of mg/kg-day, is specific to the chemical and exposure route for which it is derived.

6.4.2 Carcinogenic Health Effects

The assessment of carcinogenic effects involves assignment of a weight-of-evidence classification based on evidence of carcinogenicity in humans and, where applicable, derivation of a toxicity value based on human or animal data. Carcinogenesis is currently considered by EPA to be a nonthreshold phenomenon. In other words, it is assumed that no dose of a carcinogenic agent is without some risk of carcinogenic response, however small. For chemicals classified as known (Group A) or probable carcinogens (Group B1 and B2), a toxicity value for carcinogenic potency is derived from the dose-response data. Where available, human epidemiological data of high quality are preferable to animal data. If animal data are used, species which are metabolically or physiologically most similar to humans are preferred. The toxicity value, called the Cancer Potency Factor (CPF), is typically derived from the plot of the incidence of cancer versus the dose of the substance, and is expressed in units of $(mg/kg\text{-}day)^{-1}$. Low-dose incidence of cancer is estimated through the use of a mathematical model which extrapolates low-dose cancer incidence from high-dose data. The EPA favors the linearized multi-stage model in cases where available scientific data

do not indicate to the contrary. The cancer potency factor is considered to represent the upper 95th percent confidence limit on the probability of a response per unit intake of a chemical over a lifetime. In other words, there is a probability of only 5% that a carcinogenic response will be greater than the estimate predicted by the model (EPA, 1989a).

6.4.3 Site-Specific Application and Considerations

In this assessment, EPA cancer potency factors (CPFs) and Reference Doses (RfDs) as reported in IRIS and HEAST, 1991 are utilized in the risk characterization step. Table 6-8 presents a summary of carcinogenic and noncarcinogenic hazard identification and dose-response for the compounds included in the quantitative evaluation. For noncarcinogenic compounds included in the quantitative risk assessment, oral RfDs were utilized for risk characterization. In the absence of any RfD values for 1-methylnaphthalene and 2-methylnaphthalene, the oral RfD for napthalene was used as a surrogate value for these compounds (EPA, 1989a). For the dermal exposure route, an adjustment factor representing absorption efficiency is applied to the oral RfD so that both the exposure estimates and the toxicity values are based on absorbed (rather than administered) dose (EPA, 1989a).

Compounds classified as carcinogens by the oral route were quantitatively evaluated as carcinogens for oral, dermal, and inhalation exposure routes with oral CPFs utilized as the toxicity value. For the dermal exposure route, an adjustment factor representing absorption efficiency is applied to the oral CPF so that both the exposure estimates and the toxicity values are based on absorbed (rather than administered) dose (EPA, 1989a). Where agency derived toxicity values were unavailable for certain carcinogens detected at the Osmose site, Envirologic Data utilized a method which addresses the compound relative to its chemical class, as well as its U.S. EPA carcinogen classification. At the Osmose site, polynuclear aromatic hydrocarbons (PAHs) not referenced in IRIS were evaluated using this method to determine appropriate toxicity values. Specifically, carcinogenic PAHs were evaluated individually based on the interim U.S. EPA oral cancer potency factor for benzo(a)pyrene, and using estimates of relative potency for other potential carcinogenic PAHs as compared with benzo(a)pyrene. The relative potency scheme developed by ICF-Clement (1988) in a study conducted for the U.S. EPA was used in this assessment. The relative potency approach was developed by ICF-Clement Associates for the EPA as an alternative to the equipotency method (i.e., method whereby the degree of toxicity of each carcinogenic PAH is assumed to equal that of benzo-a-pyrene). Utilizing assumptions of a two-stage mathematical dose-response model, the relative potency approach generates a Toxicity Equivalence Factor (TEF) for each carcinogenic PAH, based on its potency relative to B(a)P. This method assigns a TEF value of 1.0 to B(a)P. TEF values less than 1.0 correspond to PAH compounds which are deemed less toxic than B(a)P, based on an

assessment of bioassay data using a two-stage mathematical model. Likewise, TEF values greater than 1.0 are associated with compounds which are more toxic than B[a]P. The relative potency approach accomodates the differing potencies of carcinogenic PAHs and, thus, is thought to generate a more realistic estimate of risk as compared with use of the equipotency approach.

Table 6-8 presents a summary of the hazard identification and dose-response evaluation for carcinogens and non-carcinogens included in the quantitative assessment.

6.5 Exposure Assessment

The purpose of the baseline exposure assessment is to develop a quantitative estimate of the potential chemical-specific and exposure scenario-specific intakes of the chemicals of interest identified at the Osmose site. The exposure assessment must consider current as well as potential future exposure scenarios. In addition, the exposure models may rely on actual site monitoring data, or in other instances rely on chemical concentrations calculated using predictive environmental fate and transport models. The exposure assessment includes several distinct steps that lead to the final quantitative estimate of potential intake, as described below.

6.5.1 Methodology

The first step to perform an exposure assessment involves a qualitative characterization of the physical setting and potentially exposed populations. Important site characteristics, including climate, geological setting, vegetation, ground water hydrology, and description of surface water, have been previously presented in Section 6.2. In describing the potentially exposed populations it is necessary to identify the location of current populations relative to the site, and to determine current and future land use.

The second step of the exposure assessment is to identify potential exposure pathways. A complete exposure pathway consists of five elements:

- a source or chemical release from a source,
- an environmental transport medium,
- a point of potential human contact with the contaminated medium (exposure point),
- a potential receptor, and
- a potential exposure route at the contact point.

Table 6-8: Health Criteria for Compounds of Potential Concern

Chemical	EPA Carcinogenic Classification	Reference Dose Oral (mg/kg-d)	Reference
NONCARCINOGENS			
Acenaphthene	-	0.6	HEAST, 1991
Anthracene	D	0.3	HEAST, 1991
Benzo(g,h,i)perylene	D	0.03	Assumed
Fluoranthene	-	0.04	HEAST, 1991
Fluorene	D	0.04	HEAST, 1991
1-Methylnaphthalene	-	0.004	Assumed
2-Methylnaphthene	-	0.004	Assumed
Naphthalene	D	0.004	HEAST, 1991
Phenanthrene	-	0.3	Assumed
Pyrene	D	0.03	HEAST, 1991

Chemical	EPA Carcinogenic Classification	Cancer Potency Factor - Oral (mg/kg-d)-1	Reference
CARCINOGENS			
Benzo(a)anthracene	B-2	1.7	Calculated*
Benzo(a)pyrene	B-2	11.5	HEAST, 1991
Benzo(b)fluoranthene	B-2	1.6	Calculated*
Benzo(k)fluoranthene	B-2	0.76	Calculated*
Chrysene	B-2	0.05	Calculated*
Dibenzo(a,h)anthracene	B-2	12.77	Calculated*
Indeno(1,2,3-cd)pyrene	B-2	2.67	Calculated*

EPA Classification:
- A - Human Carcinogen
- B - Probable Human Carcinogen
- B1 - Limited Human Envidence
- B2 - Sufficient Animal Evidence: Inadequate or no Human Evidence
- C - Possible Human Carcinogen
- D - Not Classifiable as to Human Carcingenicity

Reference Dose: An estimate of the daily exposure of the human population to a toxicant that is likely to be without any incidence of deleterious effects.

Cancer Potency Factor: The value representing the quantitative relationship between the dose of a chemical and the probability of inducing a carcinogenic effect.

- = Compound has not been evaluated

* = Calculated, based on the relative toxicity approach (ICF Clement, 1988)

EPA identifies a pathway as complete if all the elements of the exposure pathway listed are present. A pathway is considered "incomplete" if one or more of the required criteria is lacking, such as a situation where there is a source releasing to air but there are no nearby receptors. EPA (1989a) also recommends further evaluation and possible elimination of pathways which meet the above criteria, based on site-specific information and conditions. Pathways may be eliminated based on the magnitude and probability of the exposure and its potential impact relative to other exposure pathways. After the complete exposure pathways are identified, these are described quantitatively in the calculation of potential exposure point concentrations (Section 6.5.4.1) and the potential intake models (Section 6.5.5).

The third step of the exposure assessment is to quantify the exposure point concentrations (EPCs) and to estimate the chemical-specific intake. The EPC is the chemical concentration that is representative of each exposure point location where exposure may occur. The EPCs are estimated using monitoring data and/or chemical transport and environmental fate data.

The final component of the quantitative exposure assessment is to estimate potential chemical-specific intakes, expressed as the mass of substance per unit body weight per unit time (mg/kg/day). The estimated intakes are calculated using equations that include variables for:

- exposure concentration,
- contact rate,
- exposure duration (years),
- exposure frequency (days/year),
- averaging time,
- body weight, and
- relative absorption.

Some of the variables used in the exposure assessment, particularly averaging times and relative absorption, are dependent upon chemical-specific toxicity characteristics as well as site-specific considerations. According to the EPA (1989a), intake variable values should be selected so that although some intake variables may not be at their individual maximum values, the combination of all intake variables results in an estimate of the Reasonable Maximum Exposure (RME) for that pathway. The RME is defined as that level of exposure that is reasonably expected to occur at a site. The RME is intended "to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures" (EPA, 1989a).

The EPA provides recommendations for some exposure values. These recommendations may represent ranges of values or a specific value. Often, the recommendations are based on quantitative evaluations as well as professional judgment. For certain variables that have documented ranges, the EPA recommends the 95 percent upper confidence limit.

6.5.2 Potentially Exposed Populations

The purpose of this section is to characterize the Osmose site and immediate environs with respect to characteristics of the human populations on and near the site. This evaluation focuses on those characteristics that influence exposure at the site, and presents information that supports the identification of exposure pathways as well as selection of appropriate values of specific intake variables (EPA, 1989a).

In order to evaluate potential receptors on or near the Osmose site, it is necessary to determine the location of current populations relative to the site. The land use around the Osmose site is primarily residential, commercial, and vacant lots. Residential neighborhoods are located immediately adjacent to the site; however, access to the site is restricted. In addition, there is no evidence that occupational exposure currently exists on site. Overall, there is no evidence for exposure under current use conditions.

According to EPA Guidance, potential future land use is to be considered. However, one need not assume that residential use is possible (EPA, 1989a). Generally, zoning requirements, established land uses, and other relevant factors should be examined (EPA, 1989a). Currently, there are no plans in place to develop the Osmose site for any purpose other than its present industrial use. This conclusion is based on an Osmose Wood Preserving, Inc. corporate long-term development study. (Personal communication with Michael Rider, Osmose Wood Preserving, Inc.). Relative to future exposures, utility and construction workers represent a potentially exposed population. The extent of exposure would vary according to the activities of the workers and the location of their activities.

6.5.3 Media-Specific Potential Exposure Pathways

The purpose of this section is to identify potential complete exposure pathways which may be appropriate to address quantitatively. The following discussion of pathway selection is media specific. The scope of the discussion is to initially identify reasonable potential exposure routes and exposure points for the potentially exposed populations. Complete pathways are selected for quantitative evaluation unless there is justification to eliminate a pathway from detailed analysis (EPA, 1989a). Complete pathways are described below and summarized in Table 6-9.

Table 6-9

Complete Exposure Pathways

Medium	Exposure Point	Receptor	Exposure Routes
soil	biocell	worker	incidental ingestion; dermal absorption, inhalation of fugitive dust
soil	on-site(non-biocell)	worker	incidental ingestion; dermal absorption, inhalation of fugitive dust
soil	off-site	worker	incidental ingestion; dermal absorption, inhalation of fugitive dust
shallow groundwater	on- or off-site	worker	dermal absorption

6.5.3.1 Soil

PAHs are present in the soil in a specific area of the Osmose site, immediately south of the main building complex presumably as a result of historical small-scale spillage during tank filling and transfer. Utility and construction workers may contact compounds of potential concern in soil at several exposure points, both on and off the site. Potential contact with biocell soils is of particular interest as a subset of on-site soil and is treated as a separate exposure point. Exposure routes associated with the soil/worker pathway are dermal contact with contaminated soil, incidental ingestion of soil, and inhalation of fugitive dust from the soil.

6.5.3.2 Groundwater

Low concentrations of volatile organics (BTEX) and PAHs were detected in shallow groundwater wells (MW-9, MW-10, MW-11) immediately downgradient of the site. Utility workers may be in contact with compounds of potential concern in off-site shallow groundwater while servicing underground utility lines. The potential exposure route associated with the shallow groundwater is dermal contact.

Potential exposure to deep groundwater was not evaluated quantitatively in this assessment. Future impact of soil contaminants to groundwater is considered to be minimal because storage tanks have been removed from this location. Sampling of deep groundwater at well CW-1 has indicated levels of benzene and naphthalene that exceed their respective NYS ambient groundwater standard or guidance values; however, based on a well survey conducted for the area, there is no indication that deep groundwater is currently used, or will be used, in the vicinity of the site. Therefore, no complete exposure pathway is anticipated for the deep groundwater.

6.5.4 Quantification of Exposure

6.5.4.1 Exposure Point Concentrations (EPC)

Exposure point concentrations (EPCs) are calculated for each compound of potential concern in each medium at each representative exposure point location. EPCs are then input to intake models for each exposure route of interest. The Risk Assessment Guidance for Superfund, EPA (1989a) calls for the application of the arithmetic mean and the 95% upper confidence limit on the arithmetic mean. This procedure has been used to calculate the EPCs used in this assessment.

The Risk Assessment Guidance for Superfund (EPA, 1989a) recommends that nondetected values be considered with the detected results. A nondetected value means that the chemical was not present at or above the detection limit. The true concentration could be anywhere between zero and just below the detection limit. A sample quantitation limit (SQL) is the level at which a

chemical's presence and concentration can reliably and accurately be measured. There is a higher rate of false positive and false negative results in the concentration range between the detection limit and the sample quantitation limit than for results above the quantitation limit. If it is likely that the chemical is present in a sample below the SQL, then a proxy concentration equivalent to one-half the SQL is used (EPA, 1989a). Proxy concentrations equivalent to one-half the SQL value were used for off-site concentrations reported to be below the detection limit. Compound concentrations in the biocell samples that were reported as estimated (J qualifier) were used at their reported value. For those compound concentrations in the biocell reported as undetected (U qualifier) but with a SQL, proxy concentrations equivalent to one-half the SQL were used.

The results of the exposure point concentration calculations for each complete exposure pathway are discussed in the following sections. Estimated exposure point concentrations are summarized in Tables 6-10 and 6-11.

6.5.4.1.1 Soil

Exposure point concentrations were developed for each soil exposure point of interest using the following data points:

- Biocell: locations EC, SE, SW, NE, NW for June 1990 analyses;
- On-site (non-biocell): locations MW-8 (2-4'), CW-1 (6-8'), CW-1 (8-10'), and CW-2 (6-8') for October 1990 analyses;
- Off-site: locations MW-9 (4-6'), MW-10 (6-8'), and MW-11 (4-6') for October 1990 analyses.

The 95% upper confidence limit on the arithmetic mean was calculated for each of the three receptor locations. Results of the calculations are presented in Table 6-10.

There are no site-specific data available on PAH concentrations in air for the Osmose site. Therefore, exposure point concentrations of particle-entrained PAHs in ambient air were estimated by using a model which combines the concentration of each compound in the soil and the concentration of the particles in the air within the respirable size range of less than or equal to 10 microns (PM_{10}). The concentration of each compound in soil was assumed to be the same as the value used in calculating EPC values for direct exposure to soil. It was conservatively assumed that 100% of the PM_{10} fraction is composed of on-site soil particles. A maximum respirable particle concentration of 33 ug/m^3 used in the model is based on field measurements at several locations in the Buffalo area in a three year period (NY State Department of Health, 1991, personal communication).

Table 6-10
Exposure Point Concentrations
(mg/kg)
SOIL

BIOCELL

Chemical	EPC*
Noncarcinogens	
Acenaphthene	226.875
Anthracene	23.709
Dibenzofuran	152.445
Fluoranthene	58.000
Fluorene	60.000
2-Methylnaphthene	111.226
Naphthalene	62.614
Phenanthrene	117.955
Pyrene	40.449
Carcinogens	
Benzo(a)anthracene	10.936
Benzo(b)fluoranthene	6.321

ON-SITE

Chemical	EPC*
Noncarcinogens	
Acenaphthene	38.280
Anthracene	174.899
Benzo(g,h,i)perylene	12.313
Fluoranthene	43.000
Fluorene	27.858
1-Methylnaphthalene	14.325
2-Methylnaphthene	28.605
Naphthalene	73.441
Phenanthrene	62.000
Pyrene	117.079
Carcinogens	
Benzo(a)anthracene	16.439
Benzo(a)pyrene	17.005
Benzo(b)fluoranthene	13.293
Benzo(k)fluoranthene	7.224
Chrysene	14.429
Dibenzo(a,h)anthracene	3.497
Indeno(1,2,3-cd)pyrene	9.452

OFF-SITE

Chemical	EPC*
Noncarcinogens	
Benzo(g,h,i)perylene	0.006
Fluoranthene	0.011
Pyrene	0.028
Carcinogens	
Benzo(a)anthracene	0.005
Benzo(a)pyrene	0.006
Benzo(b)fluoranthene	0.007
Benzo(k)fluoranthene	0.003

GROUNDWATER

* Maximum detected value is used when the 95% Upper Confidence Limit exceeds maximum detected value

Table 6-11
Exposure Point Concentrations
Shallow Groundwater
(mg/L)

Chemical	EPC*
Noncarcinogens	
Benzene	0.0769
Toluene	0.0480
Ethylbenzene	0.0272
Exylene	0.0464
Acenophthene	0.0009
Anthacene	0.0003
Fluoranthene	0.0001
Fluorene	0.0001
1-Methylnophthalene	0.0010
2-Methylnophthalene	0.0044
Naphthalene	0.0035
Phenanthrene	0.0005
Pyrene	0.0003
Carcinogens	
Benzene	0.07690
Benzo(a)anthracene	0.00003
Benzo(a)pyrene	0.00005
Benzo(b)fluoranthene	0.00005
Benzo(k)fluoranthene	0.00003
Benzo(g,h,i)perylene	0.00006
Dibenzo(a,h)anthracene	0.00002
Indeno(1,2,3-cd)pyrene	0.00004

*Based on the 95% Upper Confidence Limit for samples collected 11/9/90 and 1/11/91; MW-8,-9,-10,-11, DW-1

6.5.4.1.2 Groundwater

Exposure point concentrations for compounds of potential concern were developed for potential dermal exposure to shallow groundwater by a utility repair man working both on-site and off-site. The data points used for this calculation of EPC included shallow monitoring wells (MW-8, MW-9, MW-10, MW-11) for the November 1990 and the January 1991 sampling events. Results of the calculations are presented in Table 6-11.

6.5.5 Intake Models

For the Osmose site baseline risk assessment, exposure intake models are used to estimate potential occupational (on-site construction worker) intakes of PAHs in the soil via dermal exposure, incidental ingestion, and inhalation. In addition, an exposure model was developed to estimate potential intake of VOCs and PAHs in shallow groundwater via dermal exposure. In calculating potential carcinogenic risk, the chronic intake is modeled as the lifetime average daily dose (LADD) and expressed in units of milligram of chemical per kilogram body weight per day (mg/kg/day).

The generic equation for estimating the LADD is as follows:

> ***Dose*** = *Chemical Concentration x Contact Rate x Exposure Frequency x Duration x 1/Body Weight x 1/Averaging Time.*

The chemical concentration is the average concentration of the chemical contacted over the exposure duration. The contact rate is the amount of contaminated medium contacted per unit time, such as milligrams of soil per day or cubic meters of air per day. The exposure frequency describes the incidence or how often the exposure occurs, generally in terms of days per year, while the exposure duration describes how long the exposure occurs, usually in terms of years. A body weight is used which is representative of the average body weight over the exposure duration. Finally, the estimated dose for potentially carcinogenic compounds is averaged over a lifetime. The exposure parameters used for the potential exposure scenarios applicable to the Osmose site are described in Tables 6-12 through 6-15 at the end of the Intake Models discussion.

For calculation of noncarcinogenic risk, the average intake is modeled as the average daily dose (ADD) and is expressed in units of milligrams of chemical per kilogram body weight per day (mg/kg/day). The generic equation used for estimating ADD is:

Dose = Chemical Concentration x Contact Rate x Exposure Frequency x Duration x 1/Body Weight

6.5.5.1 Common Intake Parameters

There are several parameters in the exposure models that are constant regardless of the pathway of interest. Since these parameters are used repetitively throughout the baseline risk assessment, these parameters are identified and the appropriate values are discussed here. Several of the parameters, such as body weights and averaging times, are well documented in the available literature and require no site-specific considerations. On the other hand, some parameters such as exposure frequency and exposure duration are site-specific and require professional judgment.

The value for body weight is the average body weight over the period of the exposure (EPA, 1989d). For pathways where intake rate:body weight ratios are relatively constant over a lifetime, it is generally not necessary to provide body weights for specific age groups. For an adult body weight value, the EPA Exposure Factors Handbook recommends an average (for men and women combined) of 70 kilograms (EPA, 1989b).

The averaging time selected for the exposure assessment depends on the type of toxic effect being assessed. For calculating potential carcinogenic risks, it is necessary to average the estimated cumulative dose over a lifetime (EPA, 1989b; EPA, 1988a) or LADD. Although 70 years has been widely used in the past, current data suggest that 75 years (=27,375 days) would be a more appropriate average value (EPA, 1989b). This is based on life expectancy data published by the U.S. Department of Commerce, which reported an average life expectancy for the total U.S. population of 74.7 years.

The exposure duration describes how long the potential exposure extends. The value for this parameter is the same for several pathways. For the occupational scenarios, the potential exposure duration is assumed to be 40 days during one year, which represents the approximate duration of a construction project. This is an overestimated value, since it is unlikely that any one worker will be on location for that length of time. For occupational exposure to shallow groundwater, the exposure duration is assumed to be 5 days, which approximates the duration of a utility repair on, or in close proximity to, the site. These scenarios also assume that the daily occupational exposure for a worker is 8 hours per day, which is the length of a standard working day. Again, this is a conservative estimate since it is unlikely that worker will be continuously exposed for the full 8 hours.

In summary, the above values for average body weights, potential exposure durations, and averaging times are common to all the exposure models developed in this assessment. The

appropriate values for the Osmose site baseline risk assessment have been identified here, precluding the need to discuss the parameters again in conjunction with specific exposure models. Pathway-specific variables utilized in the intake models are discussed in Sections 6.5.5.2 and 6.5.5.3.

6.5.5.2 Soil Exposure

Soil exposures include inhalation of fugitive dust, dermal contact, and incidental ingestion. The intake model parameters unique to these exposure routes are discussed below.

6.5.5.2.1 Inhalation of Dust

Potential exposures to contaminants of concern in air may occur through inhalation of the contaminant either in the vapor phase or adsorbed to entrained particulates. The tendency for a chemical to exist in either phase is a function of its physical and chemical properties. PAHs are relatively insoluble in water, adsorb strongly to soils, and most PAHs have low vapor pressure. Little data are available regarding volatilization rates for PAHs from soil. In general, volatilization is unlikely to be a significant transport process for PAHs (Callahan et al., 1979); therefore, inhalation of vapors from soil does not represent a significant exposure pathway. Inhalation exposure resulting from contaminated soil would be limited to potential inhalation of entrained particulates. Because the on-site area of concern is a paved parking lot, it is assumed that the only period of potential inhalation exposure would be during construction activities.

Assuming that some fraction of atmospherically entrained PAHs exist in the particulate phase, a critical issue with regard to potential exposure via inhalation is whether the particles to which the PAHs are adsorbed are within the respirable size range ≤10 microns (PM_{10} fraction). Insufficient site-specific information is available regarding the characteristics of PAH adsorption relative to particle size to make conclusions applicable to exposure modeling. In the absence of site-specific information, it is conservatively assumed that 100% of the respirable particle fraction (PM_{10}) is composed of on-site soil particles. It should be noted, however, that the 100% absorption value used in this assessment represents a worst case scenario for this pathway.

The model used to estimate occupational exposure in this assessment is based on the PAH concentrations found in the soil. A maximum respirable particle concentration used in the model of 33 ug/ m^3 is based on field measurements at several locations in the Buffalo area in a three year period (NY State Department of Health, 1991, personal communication). The U.S. EPA (1990) has estimated the inhalation rate for occupational exposure at 1.88 cubic meters per hour (15 m^3 per work day).

Animal studies suggest that PAHs are readily absorbed through the lungs (Kotin et al., 1969; Vainio et al, 1976). It is estimated that 75% of inhaled compounds remain within the body; the remaining 25% of the inhaled compound is assumed to be exhaled from the body and, therefore, is not available for absorption. It is also estimated that of the total amount of inhaled compound available for absorption, approximately 16% remains in the alveolar region of the lung and the remainder is swallowed in the GI tract.

6.5.5.2.2 Dermal Contact

In the absence of peer-reviewed, experimentally-verified research results regarding dermal absorption of PAHs in soil, the relatively comprehensive data base on the dermal absorption of tetrachlorodibenzodioxin (TCDD) is utilized as surrogate data in this analysis. The experimental data regarding dermal absorption of PAHs in pure solution are interpreted in light of research results which demonstrate that dermal absorption of TCDD in solvent is significantly greater than dermal absorption of TCDD bound to soil. The resulting absorption of PAHs is expressed as a relative absorption coefficient consistent with methods for using absorption data for risk assessment purposes (EPA, 1989d).

Based on the Poiger and Schlatter study (1980) on TCDD, the percent dose of TCDD found in the liver after dermal administration of TCDD in a soil/water paste matrix was 1.7%, compared to 14.8% found in the liver for pure TCDD dissolved in methanol. From these data, it is concluded that binding of TCDD to soil reduces the dermal absorption to 11.5% (1.7/14.8) of the pure compound absorption. The authors hypothesized that after the water from the water/soil paste in contact with the skin evaporated, TCDD was totally immobilized on the soil particles. Therefore, the derived soil inhibiting factor represents a conservative upper bound estimate for dermal contact with dry soil. The soil matrix inhibition factor of 11.5% is appropriate for PAHs.

The soil matrix inhibition factor of 11.5% was applied to the average absorption estimate for pure PAHs of 50.0% for carcinogens and non-carcinogens. This conservative value for absorption was selected following a review of research data. The range of percent dermal absorption for various carcinogenic PAH compounds tested in several species is 0.01-40% (Kao et al., 1985; Sanders et al., 1986; Heidelberger and Weiss, 1951) and 52% for non-carcinogenic PAHs in one study by Young et al. (1986). The absorption coefficient used in the dermal intake models is 0.058.

Soil Contact Rate

The soil contact rate, or the adherence factor, is a measure of the average amount of soil which adheres to a given skin surface area for a given unit time. Typical units are milligrams of soil per

square centimeter of skin per day. The amount of soil that may accumulate on the skin depends upon the body part in contact with the soil, the soil type and moisture, and the activity resulting in contact with the soil. Several different values have been either used by the EPA in risk assessment or recommended in risk assessment guidance documents:

- EPA, 1984, "Risk Analysis of TCDD Contaminated Soils": upper range estimate of 0.5 to 1.5 $mg/cm^2/day$.
- EPA, 1986a, "Development of Advisory Levels for Polychlorinated Biphenyls (PCBs) Cleanup": 1.0 $mg/cm^2/day$.
- EPA, 1988a, "Superfund Exposure Assessment Manual": Commercial potting soil = 1.45 $mg/cm^2/day$; clay mineral kaolin = 2.77 $mg/cm^2/day$.

The basis of reported soil adherence factors will be reviewed in order to select a value which, when combined with the other exposure values, results in a reasonable maximum estimate (RME) of exposure.

The values reported by EPA in the Superfund Exposure Assessment Manual (EPA, 1988a) are based on an unpublished 1979 memorandum of the Michigan Toxic Substance Control Commission. Because no further information could be obtained, these values are not considered appropriate.

Lepow et al. (1975) reported a soil contact rate of 0.5 $mg/cm^2/day$. This study measured soil accumulation on the hands of 22 children ages 2-6 that had been playing both outdoors and indoors. Soil from the hands of the children was collected by repeatedly pressing a preweighted adhesive strip to a single area of the palm of the hand and dividing the weight of the collected dirt by the surface area from which it was collected.

Roels et al. (1980) calculated a range of 0.42 $mg/cm^2/day$ to 1.78 $mg/cm^2/day$ as the soil accumulation rate on the palms of the dominant hands of 11 year old children who had previously been playing in a school yard. A dilute nitric acid solution was used to rinse the hand. The total amount of lead in the rinsate was determined and compared to the concentration of lead in the soil from the play ground in order to calculate the total amount of soil rinsed from the hand. As with Lepow et al. (1975), the total amount of soil was divided by the surface area of the rinsed hand to derive the range of contact rates. The average soil adherence rate was 0.9 $mg/cm^2/day$ (Sedman, 1989).

Que Hee et al. (1985) quantified the amount of soil that adheres to the palm of the hand of an adult for different soil particle sizes. The hand of an adult was applied to a petri dish containing a preweighted amount of dust. The hand was removed from the petri dish and nonadhering dust remaining in the dish was weighed to calculate the amount of dust that adhered to the hand. The average adherence rate was 31.0 mg per hand. For an assumed hand surface area of 160 cm^2, the corresponding soil adherence rate is 0.2 mg/cm^2.

In summary, three average soil accumulation rates based on empirical data are reported in the published literature: 0.5 mg/cm^2/day (Lepow et al., 1975); 0.9 mg/cm^2/day (Roels et al., 1980); and 0.2 mg/cm^2/day (Qee Hee et al., 1985). All three of these estimates are based on the amount of soil accumulation on the palms of hands. Other skin surfaces are not likely to be in contact with soil to the same extent as the palms of the hands. Based on the empirical data and best professional judgment, a reasonable maximum estimate of the soil contact rate for high contact surface areas (such as hands and feet) is 0.75 mg/cm^2/day. This figure is approximately equal to the average of the two highest of the three reported contact rates, i.e., 0.5 mg/cm^2/day and 0.9 mg/cm^2/day.

For the lower contact body surface areas, such as legs and arms, a reasonable maximum estimate of the soil contact rate is 0.4 mg/cm^2/day. This value is approximately equal to the average of the two lower of the three reported contact rates, i.e., 0.5 mg/cm^2/day and 0.2 mg/cm^2/day. These contact rates represent reasonable maximum estimates. It is conservative to assume that soil will accumulate at these rates for every exposure event resulting in potential contact with outdoor soil.

Soil/Skin Contact Area and Total Soil Accumulation

In estimating exposures to surface soil, it is necessary to combine the soil contact rate with the surface area of the skin in contact with the soil. Best professional judgement was used to categorize the surface area as a high or a low contact part and to identify the percentage of the body part in contact with the soil. The total estimated soil accumulation is calculated by multiplying the soil/skin contact area by the appropriate soil contact rate:

Total soil accumulation (mg/day) = exposed skin surface area (cm^2) x soil contact rate (mg/cm^2/day)

Total estimated soil accumulation for adults is calculated assuming that the surface area potentially in contact with soil consists of 100% of the hands and forearms.

Relative to the current assessment, exposed surface areas of the skin for workers performing outdoor activities was calculated for a worker not wearing gloves (exposure to hands and forearms). In this case total soil accumulation was calculated:

HANDS	FOREARMS	TOTAL SOIL ACCUMULATION
(840 cm^2 x 0.75 mg/cm^2/day)	+ (1140 cm^2 x 0.4 mg/cm^2/day)	= 1086 mg/day.

The magnitude of potentially exposed surface areas is overestimated because not all workers roll up their sleeves or otherwise expose their forearms.

In conclusion, the estimated total soil accumulation rate is a reasonable maximum estimate. It is likely that the accumulation rate and exposed surface areas on certain days will be lower than as characterized by the assumptions utilized in this analysis.

6.5.5.2.3 Incidental Ingestion

Soil Ingestion Rate

The objective of this section is to discuss factors relevant to soil ingestion for adults that occurs as a result of normal mouthing or unintentional hand-to-mouth activity. The current adult soil ingestion rate recommended by EPA (1989c) is 100 mg/day. Most adults do not intentionally ingest soil. Adult soil ingestion rates have been estimated based on studies of children.
Paustenbach (1989a) evaluated the methods and results of a variety of studies which estimate child soil ingestion rates and concluded that recent soil tracer studies provide the most accurate estimates, particularly Calabrese et al. (1989). In consideration of studies estimating child soil ingestion rates from 1977 to 1989, Paustenbach cited Calabrese et al. (1989) as the most rigorous study which provided a likely estimate of child soil ingestion rate. This range was reported to be 25-50 mg/day for children.

The EPA has developed age-specific soil ingestion rates based on the results provided by several researchers including Lepow et al. (1975), Roels et al. (1980), and Kimbrough et al. (1984). Generally, the adult ingestion rate has been estimated at approximately 50% that of children. More recent studies suggest the adult soil ingestion rate may be lower than 50%. Preliminary direct evidence that adult soil ingestion rates are significantly lower than that of children has been described in detail by Gradient (1989). This evaluation described urinary arsenic levels in Mill Creek Montana residents before and after relocation. It was found that children had 15 times higher arsenic intake levels than the adults. The excess levels of arsenic were attributed to differences to

intake of arsenic from soil ingestion, suggesting that the soil ingestion rate of older children and adults is much less than one-half that of the younger child (Gradient, 1989).
LaGoy (1987) estimated an average soil ingestion rate of 50 mg/day for adults with frequent hand-to-mouth contact (i.e., smokers) or those who are in direct contact with contaminated soil (i.e., gardeners). For the average adult, he estimated a soil ingestion rate of 25 mg/day.
For adults, Paustenbach (1989) evaluated ingestion of food contaminated with soil and incidental ingestion via poor personal hygiene as potential routes of soil ingestion exposure. He concluded adult soil ingestion rates are likely to range from 2-5 mg/day.

Based on consideration of the cited child soil ingestion rates and values for adults extrapolated from these studies of children, this assessment uses an adult soil ingestion rate of 25 mg/day. This value is based on 50% of the maximum child value cited in recent scientific literature, even though the empirical data suggests a smaller percentage may be appropriate. This conservative adult soil ingestion rate, in combination with the other exposure parameters, results in a reasonable maximum estimate of potential exposure.

6.5.5.3 Groundwater Exposure - Utility Repair Worker

Repair work to the underground utilities has occurred on site, and directly adjacent to the site on Ellicott Street; and this type of activity could occur in the future. Because the water table occurs at approximately 7 feet below grade on and adjacent to the site, it is reasonable to assume that a utility repair worker may encounter groundwater. The potential groundwater exposure pathway evaluated in this assessment is dermal contact. The exposure duration for this scenario is 8 hours per day for 5 days during a single repair event. It is considered unlikely that a specific individual would be exposed more frequently or for a longer duration at or near this site. Other variables specific to this exposure route are discussed below.

Dermal exposure is determined by considering the chemical concentration in an environmental medium that is contacted, the body surface area contacted, and the duration of exposure. In addition, the extent of potential toxicological impact to the receptor via dermal exposure is dependent on the ability of the compound of potential concern to permeate the skin surface and be absorbed into the blood.

It is important to recognize that the calculated exposure from dermal contact is actually an absorbed dose (i.e., intake), not the amount of chemical that contacts the skin (EPA, 1989a). Application of permeability constants (PC) in the exposure model accounts for this distinction. Permeability constants reflect the movement of the chemical across the skin and into the body (EPA, 1989a).

Dermal permeability constants for compounds of potential concern are estimated based on an equation derived by Brown and Rossi (1989) that relates the octanol-water partition coefficient (K_{ow}) to dermal permeability:

$$P_c = 0.1\ [K_{ow}^{0.75}/(120 + K_{ow}^{0.75})]$$

For this analysis, it was assumed that the exposed surface area consists of the hands and forearms only. Although there may be occasions when workers stand in the water, it is likely that their feet and legs will be protected. Therefore, it is reasonable to assume that the lower extremities are not exposed. According to the *Risk Assessment Guidance for Superfund* (EPA, 1989a), it is not necessary to use 95th percentile values for surface area to achieve reasonable maximum exposure. Rather, it is appropriate to select 50th percentile values, because surface area and body weight are strongly correlated, and 50th percentile values are most representative of the surface area of individuals of average weight (70 kg) (EPA, 1989a). Estimates of exposure remain conservative, because conservative assumptions are used to estimate dermal absorption (permeability constants), and frequency and duration (EPA, 1989a). The surface area for hands and forearms of an adult male (50th percentile values) are 0.099 and 0.131 m^2, respectively (EPA, 1989b).

6.5.5.4 Summary

The intake calculation models, with resulting estimates of Lifetime Average Daily Dose (LADD) and Average Daily Dose (ADD), for the exposure pathways included in the quantitative evaluation are shown in Tables 6-12 through 6-15.

6.6 Risk Characterization

Risk characterization is the final step of the baseline health risk assessment. Results of the toxicity and exposure assessments are combined to quantify potential carcinogenic and noncarcinogenic health effects. The risks are then combined across exposure pathways to estimate a cumulative potential risk for the receptor. In order to put these risk estimates into proper perspective, a discussion is also included on the assumptions and uncertainties inherent in the risk assessment methodology.

TABLE 6-12

CONSTRUCTION WORKER
INCIDENTAL INGESTION OF CHEMICALS IN SOIL

EQUATION

[1] ADD = CS * IR * CF * 1/BW
[2] LADD = CS * IR * CF * EF * ED * 1/BW * 1/AT

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*	*REFERENCE*
CS = Concentration In Soil (95% UCL)	mg/kg	See Below	
IR = Ingestion Rate	mg/day	25	
CF = Conversion Factor	kg/mg	1.00E-06	
EF = Exposure Frequency	days/year	40	
ED = Exposure Duration	years	1	
BW = Body Weight	kg	70	
AT = Averaging Time	days	27375	
ADD = Average Daily Dose	mg/kg-day	EQ[1]	
LADD = Lifetime Average Daily Dose	mg/kg-day	EQ[2]	

RESULTS *CHEMICAL*	*BIOCELL EXPOSURE POINT CONC. (mg/kg)*	*ON-SITE EXPOSURE POINT CONC. (mg/kg)*	*OFF-SITE EXPOSURE POINT CONC. (mg/kg)*	*BIOCELL* INTAKE	*ON-SITE* INTAKE	*OFF-SITE* INTAKE
NONCARCINOGENIC				ADD	ADD	ADD
Acenaphthene	226.875	38.28	-	8.10E-05	1.37E-05	-
Anthracene	23.709	174.899	-	8.47E-06	6.25E-05	-
Benzo(g,h,i)perylene	-	12.313	0.006	-	4.40E-06	2.14E-09
Dibenzofuran	152.445	-	-	5.44E-05	-	-
Fluoranthene	58	43.00	0.011	2.07E-05	1.54E-05	3.93E-09
Fluorene	60	27.858	-	2.14E-05	9.95E-06	-
1-Methylnaphthene	-	14.325	-	-	5.12E-06	-
2-Methylnaphthene	111.226	28.605	-	3.97E-05	1.02E-05	-
Naphthalene	62.614	73.441	-	2.24E-05	2.62E-05	-
Phenanthrene	117.955	62.00	-	4.21E-05	2.21E-05	-
Pyrene	40.449	117.079	0.028	1.44E-05	4.18E-05	1.00E-08
CARCINOGENIC				LADD	LADD	LADD
Benzo(a)anthracene	10.936	16.439	0.005	5.71E-09	8.58E-09	2.61E-12
Benzo(a)pyrene	-	17.005	0.006	-	8.87E-09	3.13E-12
Benzo(b)fluoranthene	6.321	13.293	0.007	3.30E-09	6.94E-09	3.65E-12
Benzo(k)fluoranthene	-	7.224	0.003	-	3.77E-09	1.57E-12
Chrysene	-	14.429	-	-	7.53E-09	-
Dibenzo(a,h)anthracene	-	3.497	-	-	1.82E-09	-
Indeno(1,2,3-cd)pyrene	-	9.452	-	-	4.93E-09	-

TABLE 6-13

CONSTRUCTION WORKER
DERMAL CONTACT WITH CHEMICALS IN SOIL

EQUATION

[1] ADD = CS * SA *AF * CF * 1/BW

[2] LADD = CS * SA *AF * CF * EF * ED * 1/BW * 1/AT

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*	*REFERENCE*
CS = Concentration in Soil (95% UCL)	mg/kg	See Below	
SA = Soil Accumulation	mg/day	1086	
AF = Absorption Factor	unitless	0.058	
CF = Conversion Factor	kg/mg	1.00E-06	
EF = Exposure Frequency	days/year	40	
ED = Exposure Duration	years	1	
BW = Body Weight	kg	70	
AT = Averaging Time	days	27375	
ADF = Health Criterion Adjustment Factor	unitless	See Below	
ADD = Average Daily Dose	mg/kg-day	EQ[1]	
LADD = Lifetime Average Daily Dose	mg/kg-day	EQ[2]	

RESULTS *CHEMICAL*	*BIOCELL EXPOSURE POINT CONC. (mg/kg)*	*ON-SITE EXPOSURE POINT CONC. (mg/kg)*	*OFF-SITE EXPOSURE POINT CONC. (mg/kg)*	*BIOCELL* INTAKE	*ON-SITE* INTAKE	*OFF-SITE* INTAKE
NONCARCINOGENIC				ADD	ADD	ADD
Acenaphthene	226.875	38.28	-	2.04E-04	3.44E-05	-
Anthracene	23.709	174.899	-	2.13E-05	1.57E-04	-
Benzo(g,h,i)perylene	-	12.313	0.006	-	1.11E-05	5.40E-09
Dibenzofuran	152.445	-	-	1.37E-04	-	-
Fluoranthene	58	43.00	0.011	5.22E-05	3.87E-05	9.90E-09
Fluorene	60	27.858	-	5.40E-05	2.51E-05	-
1-Methylnaphthene	-	14.325	-	-	1.29E-05	-
2-Methylnaphthene	111.226	28.605	-	1.00E-04	2.57E-05	-
Naphthalene	62.614	73.441	-	5.63E-05	6.61E-05	-
Phenanthrene	117.955	62.00	-	1.06E-04	5.58E-05	-
Pyrene	40.449	117.079	0.028	3.64E-05	1.05E-04	2.52E-08
CARCINOGENIC				LADD	LADD	LADD
Benzo(a)anthracene	10.936	16.439	0.005	1.44E-08	2.16E-08	6.57E-12
Benzo(a)pyrene	-	17.005	0.006	-	2.24E-08	7.89E-12
Benzo(b)fluoranthene	6.321	13.293	0.007	8.31E-09	1.75E-08	9.20E-12
Benzo(k)fluoranthene	-	7.224	0.003	-	9.50E-09	3.94E-12
Chrysene	-	14.429	-	-	1.90E-08	-
Dibenzo(a,h)anthracene	-	3.497	-	-	4.60E-09	-
Indeno(1,2,3-cd)pyrene	-	9.452	-	-	1.24E-08	-

TABLE 6-14

CONSTRUCTION WORKER
INHALATION OF FUGITIVE DUST

EQUATION

[1] ADD = CS * PM10 * IR * (FL + FS) * ED1 * CF * 1/BW
[2] LADDih = CS * PM10 * IR * FL * ED1 * ED2 * ED3 * CF * 1/BW * 1/AT
[3] LADDin = CS * PM10 * IR * FS * ED1 * ED2 * ED3 * CF * 1/BW * 1/AT

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*	*REFERENCE*
CS = Concentration in Soil (95% UCL)	mg/kg	See Below	
PM10 = Particulate	ug/m3	33	
IR = Inhalation Rate	m3/hr	1.88	
FL = Fraction Retained in Lung	unitless	0.125	
FS = Fraction Swallowed	unitless	0.625	
ED1 = Exposure Duration	hours/day	8	
ED2 = Exposure Duration	days	40	
ED3 = Exposure Duration	yr	1	
CF = Conversion Factor	kg/ug	1E-09	
BW = Body Weight	kg	70	
AT = Averaging Time	days	27375	
ADD = Average Daily Dose	mg/kg-day	EQ[1]	
LADDin = Lifetime Average Daily Dose (Ingestion)	mg/kg-day	EQ[3]	
LADDih = Lifetime Average Daily Dose (Inhalation)	mg/kg-day	EQ[2]	

RESULTS	*BIOCELL EXPOSURE POINT*	*ON-SITE EXPOSURE POINT*	*OFF-SITE EXPOSURE POINT*	*BIOCELL* INTAKE		*ON-SITE* INTAKE		*OFF-SITE* INTAKE	
CHEMICAL	*CONC. (mg/kg)*	*CONC. (mg/kg)*	*CONC. (mg/kg)*						
NONCARCINOGENIC				ADD		ADD		ADD	
Acenaphthene	226.875	38.28	-	1.21E-06		2.04E-07		-	
Anthracene	23.709	174.899	-	1.26E-07		9.30E-07		-	
Benzo(g,h,i)perylene	-	12.313	0.006	-		6.55E-08		3.19E-11	
Dibenzofuran	152.445	-	-	8.11E-07		-		-	
Fluoranthene	58	43.00	0.011	3.08E-07		2.29E-07		5.85E-11	
Fluorene	60	27.858	-	3.19E-07		1.48E-07		-	
1-Methylnaphthene	-	14.325	-	-		7.62E-08		-	
2-Methylnaphthene	111.226	28.605	-	5.91E-07		1.52E-07		-	
Naphthalene	62.614	73.441	-	3.33E-07		3.91E-07		-	
Phenanthrene	117.955	62.00	-	6.27E-07		3.30E-07		-	
Pyrene	40.449	117.079	0.028	2.15E-07		6.23E-07		1.49E-10	
				BIOCELL		ON-SITE		OFF-SITE	
CARCINOGENIC				LADDih	LADDin	LADDih	LADDin	LADDih	LADDin
Benzo(a)anthracene	10.936	16.439	0.005	1.42E-11	7.08E-11	2.13E-11	1.06E-10	6.48E-15	3.24E-14
Benzo(a)pyrene	-	17.005	0.006	-	-	2.20E-11	1.10E-10	7.77E-15	3.89E-14
Benzo(b)fluoranthene	6.321	13.293	0.007	8.19E-12	4.09E-11	1.72E-11	8.61E-11	9.07E-15	4.53E-14
Benzo(k)fluoranthene	-	7.224	0.003	-	-	9.36E-12	4.68E-11	3.89E-15	1.94E-14
Chrysene	-	14.429	-	-	-	1.87E-11	9.34E-11	-	-
Dibenzo(a,h)anthracene	-	3.497	-	-	-	4.53E-12	2.26E-11	-	-
Indeno(1,2,3-cd)pyrene	-	9.452	-	-	-	1.22E-11	6.12E-11	-	-

TABLE 6-15

UTILITY REPAIR WORKER
DERMAL CONTACT WITH GROUNDWATER

EQUATION

[1] MDD = CW * SA * PC * ET * CF * 1/BW
[2] LADD = CW * SA * PC * ET * EF * ED * CF * 1/BW * 1/AT

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*
CW = Concentration in Water	mg/l	See Below
SA = Skin Area Available for Contact	cm2	2300
PC =Permeability Constant	cm/hr	See Below
CF = Conversion Factor	1L/1000cm3	0.001
EF = Exposure Frequency	days/year	5
ED = Exposure Duration	years	1
ET = ExposureTime	hrs/day	8
BW = Body Weight	kg	70
AT = Averaging Time	days	27375
MDD = Maximum Daily Dose	mg/kg-day	EQ[1]
LADD = Lifetime Average Daily Dose	mg/kg-day	EQ[2]

RESULTS

Chemical	*Exposure Point Concentration (mg/l)*	*PC*	*INTAKE*
NONCARCINOGENIC			MDD
Benzene	0.0769	0.019467	3.94E-04
Toluene	0.0480	0.046455	5.86E-04
Ethylbenzene	0.0272	0.065754	4.70E-04
Xylene	0.0464	0.067671	8.25E-04
Acenaphthene	0.0009	0.093644	2.22E-05
Anthracene	0.0003	0.094772	7.47E-06
Fluoranthene	0.0001	0.098807	2.60E-06
Fluorene	0.0001	0.091917	2.42E-06
1-methynaphthalene	0.0010	0.073736	1.94E-05
2-methynaphthalene	0.0044	0.073736	8.53E-05
Naphthalene	0.0035	0.073736	6.78E-05
Phenanthrene	0.0005	0.094857	1.25E-05
Pyrene	0.0003	0.098787	7.79E-06
CARCINOGENIC			LADD
Benzene	0.0769	0.019467	7.19E-08
Benzo(a)anthracene	0.00003	0.099261	1.43E-10
Benzo(a)pyrene	0.00005	0.099647	2.39E-10
Benzo(b)fluoranthene	0.00005	0.099858	2.40E-10
Benzo(k)fluoranthene	0.00003	0.099911	1.44E-10
Benzo(g,h,l)perylene	0.00006	0.099955	2.88E-10
Dibenzo(a,h)anthracene	0.00002	0.099602	9.56E-11
Indeno(1,2,3-cd)pyrene	0.00004	0.099978	1.92E-10

Potential carcinogenic risks of the chemicals of concern are estimated from daily intakes and chemical-specific dose response information. The cancer potency factor applied by EPA for regulatory purposes is the upper 95 percent confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure. The low-dose carcinogenic risk equation used by EPA for estimating risks for regulatory purposes is:

Risk = Lifetime Average Daily Dose (LADD) * Cancer potency factor

As outlined in the EPA Guidance, total upperbound cancer risk for each exposure pathway is calculated by summing the substance-specific cancer risks (EPA, 1989a). In addition, for baseline risk assessments, potential cancer risks from various exposure pathways are assumed to be additive if the risks are for the same receptor and time period (EPA, 1989a). Since a construction worker could theoretically face the "reasonable maximum exposure" (RME) by all three pathways, it is appropriate to combine risks across exposure pathways at this site (EPA, 1989a). For the purposes of this assessment, it was assumed that the criterion of acceptable total risk to a receptor is 1×10^{-5}, which falls within the range of acceptable risk (1×10^{-4} to 1×10^{-6}) frequently cited by the U.S.EPA (See discussion in Section 6.6.4).

6.6.1 Carcinogenic Risk Estimates

The estimates for the potential carcinogenic risk evaluation for occupational exposure to soil and groundwater are itemized for individual chemicals by specific receptors and exposure pathways in Tables 6-16 through 6-19. Specific estimates for each indicator compound were generated for incidental ingestion of soil, dermal contact with soil, and inhalation of fugitive dust for hypothetical biocell, on-site (non-biocell), and off-site construction workers. In addition, a cumulative potential risk estimate was generated for each potential receptor by summing the total potential risk (incidental soil ingestion, dermal contact, and inhalation). Table 6-20 summarizes these cumulative cancer risk estimates. In the biocell soil exposure scenario, total upper-bound risk estimates less than the criterion of acceptable risk (1×10^{-5}) were calculated for all three exposure routes (i.e., they represent acceptable levels of risk). In addition, the cumulative risk estimate of 9.07×10^{-8} is considerably less than the criterion of acceptable risk. For the on-site (non-biocell) soil exposure scenario, total risk estimates less than 1×10^{-5} were calculated for all three exposure routes and for the cumulative risk. Likewise, in the off-site soil exposure scenario individual pathway risk and total risk estimates were considerably less than the criterion. The total risk estimate for the utility repair worker exposure to groundwater is approximately 1×10^{-6}, which is also considerably less than the criterion of acceptable risk.

Table 6-16. BIOCELL: CANCER RISK ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	*Chemical*	*LADD (mg/kg-day)*	*Cancer Potency (mg/kg-day)-1*	*Chemical Risk*	*Total Exposure Pathway Risk*
CONSTRUCTION WORKER INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Benzo(a)anthracene	5.71E-09	1.7	9.70E-09	
	Benzo(a)pyrene	-	-	-	
	Benzo(b)fluoranthene	3.30E-09	1.6	5.28E-09	
	Benzo(k)fluoranthene	-	-	-	
	Chrysene	-	-	-	
	Dibenzo(a,h)anthracene	-	-	-	
	Indeno(1,2,3-cd)pyrene	-	-	-	
					1.50E-08
CONSTRUCTION WORKER DERMAL CONTACT WITH CHEMICALS IN SOIL					
				ADF	
	Benzo(a)anthracene	1.44E-08	1.7	0.5	4.89E-08
	Benzo(a)pyrene	-	-	-	-
	Benzo(b)fluoranthene	8.31E-09	1.6	0.5	2.66E-08
	Benzo(k)fluoranthene	-	-	-	-
	Chrysene	-	-	-	-
	Dibenzo(a,h)anthracene	-	-	-	-
	Indeno(1,2,3-cd)pyrene	-	-	-	-
					7.55E-08
CONSTRUCTION WORKER INHALATION OF FUGITIVE DUST		LADDIh	LADDIn	CPF (Inh) (mg/kg-day)-1	CPF (oral) (mg/kg-day)-1	
	Benzo(a)anthracene	1.42E-11	7.08E-11	0.88	1.7	1.33E-10
	Benzo(a)pyrene	-	-	-	-	-
	Benzo(b)fluoranthene	8.19E-12	4.09E-11	0.85	1.6	7.24E-11
	Benzo(k)fluoranthene	-	-	-	-	-
	Chrysene	-	-	-	-	-
	Dibenzo(a,h)anthracene	-	-	-	-	-
	Indeno(1,2,3-cd)pyrene	-	-	-	-	-
						2.05E-10

RISK = LADD * CPF
LADD = Lifetime Average Daily Dose (mg/kg-day)
CPF = Cancer Potency Factor (mg/kg-day)-1
ADF = Adjustment Factor applied to CPF, (CPF/ADF)

Table 6-17. ON-SITE: CANCER RISK ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	*Chemical*	*LADD (mg/kg-day)*	*Cancer Potency (mg/kg-day)-1*	*Chemical Risk*	*Total Exposure Pathway Risk*
CONSTRUCTION WORKER					
INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Benzo(a)anthracene	8.58E-09	1.7	1.46E-08	
	Benzo(a)pyrene	8.87E-09	11.5	1.02E-07	
	Benzo(b)fluoranthene	6.94E-09	1.6	1.11E-08	
	Benzo(k)fluoranthene	3.77E-09	0.76	2.87E-09	
	Chrysene	7.53E-09	0.05	3.76E-10	
	Dibenzo(a,h)anthracene	1.82E-09	12.77	2.33E-08	
	Indeno(1,2,3-cd)pyrene	4.93E-09	2.67	1.32E-08	
					1.67E-07
CONSTRUCTION WORKER					
DERMAL CONTACT WITH CHEMICALS IN SOIL					
				ADF	
	Benzo(a)anthracene	2.16E-08	1.7	0.5	7.35E-08
	Benzo(a)pyrene	2.24E-08	11.5	0.5	5.14E-07
	Benzo(b)fluoranthene	1.75E-08	1.6	0.5	5.59E-08
	Benzo(k)fluoranthene	9.50E-09	0.76	0.5	1.44E-08
	Chrysene	1.90E-08	0.05	0.5	1.90E-09
	Dibenzo(a,h)anthracene	4.60E-09	12.77	0.5	1.17E-07
	Indeno(1,2,3-cd)pyrene	1.24E-08	2.67	0.5	6.64E-08
					8.44E-07

CONSTRUCTION WORKER INHALATION OF FUGITIVE DUST	LADDih	LADDin	CPF (Inh) (mg/kg-day)-1	CPF (oral) (mg/kg-day)-1		
Benzo(a)anthracene	2.13E-11	1.06E-10	0.88	1.7	2.00E-10	
Benzo(a)pyrene	2.20E-11	1.10E-10	6.1	11.5	1.40E-09	
Benzo(b)fluoranthene	1.72E-11	8.61E-11	0.85	1.6	1.52E-10	
Benzo(k)fluoranthene	9.36E-12	4.68E-11	0.4	0.76	3.93E-11	
Chrysene	1.87E-11	9.34E-11	0.03	0.05	5.23E-12	
Dibenzo(a,h)anthracene	4.53E-12	2.26E-11	6.8	12.77	3.20E-10	
Indeno(1,2,3-cd)pyrene	1.22E-11	6.12E-11	1.4	2.67	1.81E-10	
						2.30E-09

RISK = LADD * CPF
LADD = Lifetime Average Daily Dose (mg/kg-day)
CPF = Cancer Potency Factor (mg/kg-day)-1
ADF = Adjustment Factor applied to CPF, (CPF/ADF)

Table 6-18. OFF-SITE: CANCER RISK ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	*Chemical*	*LADD (mg/kg-day)*	*Cancer Potency (mg/kg-day)-1*	*Chemical Risk*	*Total Exposure Pathway Risk*
CONSTRUCTION WORKER INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Benzo(a)anthracene	2.61E-12	1.7	4.44E-12	
	Benzo(a)pyrene	3.13E-12	11.5	3.60E-11	
	Benzo(b)fluoranthene	3.65E-12	1.6	5.84E-12	
	Benzo(k)fluoranthene	1.57E-12	0.76	1.19E-12	
	Chrysene	-	-	-	
	Dibenzo(a,h)anthracene	-	-	-	
	Indeno(1,2,3-cd)pyrene	-	-	-	
					4.75E-11
CONSTRUCTION WORKER DERMAL CONTACT WITH CHEMICALS IN SOIL				ADF	
	Benzo(a)anthracene	6.57E-12	1.7	0.5	2.24E-11
	Benzo(a)pyrene	7.89E-12	11.5	0.5	1.81E-10
	Benzo(b)fluoranthene	9.20E-12	1.6	0.5	2.95E-11
	Benzo(k)fluoranthene	3.94E-12	0.76	0.5	6.00E-12
	Chrysene	-	-	-	-
	Dibenzo(a,h)anthracene	-	-	-	-
	Indeno(1,2,3-cd)pyrene	-	-	-	-
					2.39E-10

CONSTRUCTION WORKER INHALATION OF FUGITIVE DUST	LADDih	LADDin	CPF (inh) (mg/kg-day)-1	CPF (oral) (mg/kg-day)-1		
Benzo(a)anthracene	6.48E-15	3.24E-14	0.88	1.7	6.07E-14	
Benzo(a)pyrene	7.77E-15	3.89E-14	6.1	11.5	4.94E-13	
Benzo(b)fluoranthene	9.07E-15	4.53E-14	0.85	1.6	8.02E-14	
Benzo(k)fluoranthene	3.89E-15	1.94E-14	0.4	0.76	1.63E-14	
Chrysene	-	-	0.03	-	-	
Dibenzo(a,h)anthracene	-	-	6.8	-	-	
Indeno(1,2,3-cd)pyrene	-	-	1.4	-	-	
						6.51E-13

RISK = LADD * CPF
LADD = Lifetime Average Daily Dose (mg/kg-day)
CPF = Cancer Potency Factor (mg/kg-day)-1
ADF = Adjustment Factor applied to CPF, (CPF/ADF)

Table 6-19. UTILITY WORKER: CANCER RISK ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	*Chemical*	*LADD (mg/kg-day)*	*Cancer Potency (mg/kg-day)-1*	*AF*	*Chemical Risk*	*Total Exposure Pathway Risk*
UTILITY REPAIR WORKER						
DERMAL CONTACT WITH GROUNDWATER						
	Benzene	7.19E-08	0.029	1.0	2.08E-09	
	Benzo(a)anthracene	1.43E-10	1.7	0.5	4.86E-10	
	Benzo(a)pyrene	2.39E-10	11.5	0.5	5.5E-09	
	Benzo(b)fluoranthene	2.40E-10	1.6	0.5	7.67E-10	
	Benzo(k)fluoranthene	1.44E-10	0.76	0.5	2.19E-10	
	Benzo(g,h,l)perylene	2.88E-10	0.253	0.5	1.46E-10	
	Dibenzo(a,h)anthracene	9.56E-11	12.77	0.5	2.44E-09	
	Indeno(1,2,3-cd)pyrene	1.92E-10	2.67	0.5	1.03E-09	
						1.27E-08

RISK = LADD * CPF
LADD = Lifetime Average Daily Dose (mg/kg-day)
CPF = Cancer Potency Factor (mg/kg-day)-1
AF = Adjustment factor for reference dose (to correspond to absorbed dose)

Table 6-20: CUMULATIVE CANCER RISK ESTIMATES FOR INDIVIDUAL AND COMBINED RECEPTORS

Receptor	Exposure Pathway	Pathway Risk	Contribution to Total Risk	Cumulative Risk
Construction Worker, Biocell				9.07E-08
	Incidental Ingestion of Soil	1.50E-08	16.521%	
	Dermal Contact with Soil	7.55E-08	83.252%	
	Inhalation of Fugitive Dust	2.05E-10	0.226%	
Construction Worker, On-Site (Non-Biocell)				1.01E-06
	Incidental Ingestion of Soil	1.67E-07	16.521%	
	Dermal Contact with Soil	8.44E-07	83.252%	
	Inhalation of Fugitive Dust	2.30E-09	0.227%	
Construction Worker, Off-site				2.87E-10
	Incidental Ingestion of Soil	4.75E-11	16.521%	
	Dermal Contact with Soil	2.39E-10	83.252%	
	Inhalation of Fugitive Dust	6.51E-13	0.227%	
Utility Repair Worker				1.27E-08
	Dermal Contact With Groundwater	1.27E-08	100.000%	

In summary, under the conservative assumptions and parameter values used in this assessment, estimates of total risk for the hypothetical on-site biocell receptor, the on-site (non-biocell) receptor, the hypothetical off-site worker, as well as the hypothetical utility repair worker exposed to groundwater, are well below the criterion of acceptable risk (1×10^{-5}) by factors of approximately 10 to 10,000, representing a large "margin of safety" for any of these potential receptors.

6.6.2 Hazard Index Estimates

The potential for noncarcinogenic toxicity is not expressed as the probability of an individual suffering an adverse effect (EPA, 1989a). Rather noncarcinogenic effects are evaluated by comparing an exposure level over a specified time period with a reference dose derived for a similar exposure period. This ratio of exposure to toxicity is called a hazard quotient (EPA, 1989a). The hazard quotient assumes that there is a level of exposure (i.e., the RfD) below which adverse health effects are unlikely (EPA, 1989a). If the exposure levels exceed this threshold (ratio > 1), there is a greater likelihood for noncarcinogenic effects to occur (EPA, 1989a). It is important not to interpret the ratios as probabilities. In addition, the likelihood of adverse effects does not increase linearly as the RfD is approached or exceeded, because RfDs do not have equal accuracy or are not based on the same severity of toxic effects (EPA, 1989a).

To assess the potential for noncarcinogenic effects posed by more than one chemical, it is appropriate to calculate a hazard index which sums the hazard quotients. If the hazard index exceeds one, the likelihood of adverse health effects increases (EPA, 1989a).

In this baseline risk assessment, hazard quotients were calculated for each chemical; and hazard indices were estimated for each exposure pathway. To assess the overall potential for noncarcinogenic effects, hazard indices posed by the exposure pathways (dermal contact, incidental ingestion, and inhalation) were summed. The hazard index estimates for individual chemicals, by receptor and exposure pathway, are shown in Tables 6-21 through 6-24. In addition, total hazard index estimates by receptor are presented in Table 6-25. The total hazard index for the hypothetical on-site biocell worker is approximately 1.1×10^{-1} ; for the on-site (non-biocell) worker is approximately 4.6×10^{-2}; and for the off-site worker is approximately 1.8×10^{-6}. Likewise, the total hazard index for the hypothetical utility repair worker exposed to groundwater is approximately 6×10^{-2}. All of these values indicate little likelihood for adverse noncarcinogenic effects to occur to any of these hypothetical receptors.

Table 6-21. BIOCELL: HAZARD INDEX ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	Chemical	ADD (mg/kg-day)	ADI	Chemical Hazard Quotient	Exposure Pathway Hazard Index
CONSTRUCTION WORKER INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Acenaphthene	8.10E-05	0.06	1.35E-03	
	Anthracene	8.47E-06	0.3	2.82E-05	
	Benzo(g,h,i)perylene	-	-	-	
	Dibenzofuran	5.44E-05	0.004	1.36E-02	
	Fluoranthene	2.07E-05	0.04	5.18E-04	
	Fluorene	2.14E-05	0.04	5.36E-04	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	3.97E-05	0.004	9.93E-03	
	Naphthalene	2.24E-05	0.004	5.59E-03	
	Phenanthrene	4.21E-05	0.3	1.40E-04	
	Pyrene	1.44E-05	0.03	4.82E-04	
					3.22E-02
CONSTRUCTION WORKER DERMAL CONTACT WITH CHEMICALS IN SOIL					
	Acenaphthene	2.04E-04	0.06	3.40E-03	
	Anthracene	2.13E-05	0.3	7.11E-05	
	Benzo(g,h,i)perylene	-	-	-	
	Dibenzofuran	1.37E-04	0.004	3.43E-02	
	Fluoranthene	5.22E-05	0.04	1.30E-03	
	Fluorene	5.40E-05	0.04	1.35E-03	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	1.00E-04	0.004	2.50E-02	
	Naphthalene	5.63E-05	0.004	1.41E-02	
	Phenanthrene	1.06E-04	0.3	3.54E-04	
	Pyrene	3.64E-05	0.03	1.21E-03	
					8.11E-02
CONSTRUCTION WORKER INHALATION OF FUGITIVE DUST					
	Acenaphthene	1.21E-06	0.06	2.01E-05	
	Anthracene	1.26E-07	0.3	4.20E-07	
	Benzo(g,h,i)perylene	-	-	-	
	Dibenzofuran	8.11E-07	0.004	2.03E-04	
	Fluoranthene	3.08E-07	0.04	7.71E-06	
	Fluorene	3.19E-07	0.04	7.98E-06	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	5.91E-07	0.004	1.48E-04	
	Naphthalene	3.33E-07	0.004	8.32E-05	
	Phenanthrene	6.27E-07	0.3	2.09E-06	
	Pyrene	2.15E-07	0.03	7.17E-06	
					4.79E-04

Hazard Quotient = ADD / ADI
ADD = Average Daily Dose (mg/kg-day)
ADI = Acceptable Daily Intake (Reference Dose) (mg/kg-day)

Table 6-22. ON-SITE: HAZARD INDEX ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	Chemical	ADD (mg/kg-day)	ADI	Chemical Hazard Quotient	Exposure Pathway Hazard Index
CONSTRUCTION WORKER					
INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Acenaphthene	1.37E-05	0.06	2.28E-04	
	Anthracene	6.25E-05	0.3	2.08E-04	
	Benzo(g,h,i)perylene	4.40E-06	0.03	1.47E-04	
	Dibenzofuran	-	-	-	
	Fluoranthene	1.54E-05	0.04	3.84E-04	
	Fluorene	9.95E-06	0.04	2.49E-04	
	1-Methylnaphthene	5.12E-06	0.004	1.28E-03	
	2-Methylnaphthene	1.02E-05	0.004	2.55E-03	
	Naphthalene	2.62E-05	0.004	6.56E-03	
	Phenanthrene	2.21E-05	0.3	7.38E-05	
	Pyrene	4.18E-05	0.03	1.39E-03	
					1.31E-02
CONSTRUCTION WORKER					
DERMAL CONTACT WITH CHEMICALS IN SOIL					
	Acenaphthene	3.44E-05	0.06	5.74E-04	
	Anthracene	1.57E-04	0.3	5.25E-04	
	Benzo(g,h,i)perylene	1.11E-05	0.03	3.69E-04	
	Dibenzofuran	-	-	-	
	Fluoranthene	3.87E-05	0.04	9.67E-04	
	Fluorene	2.51E-05	0.04	6.27E-04	
	1-Methylnaphthene	1.29E-05	0.004	3.22E-03	
	2-Methylnaphthene	2.57E-05	0.004	6.43E-03	
	Naphthalene	6.61E-05	0.004	1.65E-02	
	Phenanthrene	5.58E-05	0.3	1.86E-04	
	Pyrene	1.05E-04	0.03	3.51E-03	
					3.29E-02
CONSTRUCTION WORKER					
INHALATION OF FUGITIVE DUST					
	Acenaphthene	2.04E-07	0.06	3.39E-06	
	Anthracene	9.30E-07	0.3	3.10E-06	
	Benzo(g,h,i)perylene	6.55E-08	0.03	2.18E-06	
	Dibenzofuran	-	-	-	
	Fluoranthene	2.29E-07	0.04	5.72E-06	
	Fluorene	1.48E-07	0.04	3.70E-06	
	1-Methylnaphthene	7.62E-08	0.004	1.90E-05	
	2-Methylnaphthene	1.52E-07	0.004	3.80E-05	
	Naphthalene	3.91E-07	0.004	9.76E-05	
	Phenanthrene	3.30E-07	0.3	1.10E-06	
	Pyrene	6.23E-07	0.03	2.08E-05	
					1.95E-04

Hazard Quotient = ADD / ADI
ADD = Average Daily Dose (mg/kg-day)
ADI = Acceptable Daily Intake (Reference Dose) (mg/kg-day)

Table 6-23. OFF-SITE: HAZARD INDEX ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	Chemical	ADD (mg/kg-day)	ADI	Chemical Hazard Quotient	Exposure Pathway Hazard Index
CONSTRUCTION WORKER INCIDENTAL INGESTION OF CHEMICALS IN SOIL					
	Acenaphthene	-	-	-	
	Anthracene	-	-	-	
	Benzo(g,h,i)perylene	2.14E-09	0.03	7.14E-08	
	Dibenzofuran	-	-	-	
	Fluoranthene	3.93E-09	0.04	9.82E-08	
	Fluorene	-	-	-	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	-	-	-	
	Naphthalene	-	-	-	
	Phenanthrene	-	-	-	
	Pyrene	1.00E-08	0.03	3.33E-07	
					5.03E-07
CONSTRUCTION WORKER DERMAL CONTACT WITH CHEMICALS IN SOIL					
	Acenaphthene	-	-	-	
	Anthracene	-	-	-	
	Benzo(g,h,i)perylene	5.40E-09	0.03	1.80E-07	
	Dibenzofuran	-	-	-	
	Fluoranthene	9.90E-09	0.04	2.47E-07	
	Fluorene	-	-	-	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	-	-	-	
	Naphthalene	-	-	-	
	Phenanthrene	-	-	-	
	Pyrene	2.52E-08	0.03	8.40E-07	
					1.27E-06
CONSTRUCTION WORKER INHALATION OF FUGITIVE DUST					
	Acenaphthene	-	-	-	
	Anthracene	-	-	-	
	Benzo(g,h,i)perylene	3.19E-11	0.03	1.06E-09	
	Dibenzofuran	-	-	-	
	Fluoranthene	5.85E-11	0.04	1.46E-09	
	Fluorene	-	-	-	
	1-Methylnaphthene	-	-	-	
	2-Methylnaphthene	-	-	-	
	Naphthalene	-	-	-	
	Phenanthrene	-	-	-	
	Pyrene	1.49E-10	0.03	4.96E-09	
					7.49E-09

Hazard Quotient = ADD / ADI
ADD = Average Daily Dose (mg/kg-day)
ADI = Acceptable Daily Intake (Reference Dose) (mg/kg-day)

Table 6-24. UTILITY WORKER: HAZARD INDEX ESTIMATES, FOR INDIVIDUAL CHEMICALS, BY RECEPTOR AND EXPOSURE PATHWAY

Receptor / Pathway	*Chemical*	*MDD (mg/kg-day)*	*ADI*	*AF*	*Chemical Hazard Quotient*	*Exposure Pathway Hazard Index*
UTILITY REPAIR WORKER						
DERMAL CONTACT WITH GROUNDWATER						
	Benzene	3.94E-04	0.047	1	8.37E-03	
	Toluene	5.86E-04	0.2	1	2.93E-03	
	Ethylbenzene	4.70E-04	0.1	1	4.70E-03	
	Xylene	8.25E-04	2	1	4.13E-04	
	Acenaphthene	2.22E-05	0.06	1	3.69E-04	
	Anthracene	7.47E-06	0.3	1	2.49E-05	
	Fluoranthene	2.60E-06	0.04	1	6.49E-05	
	Fluorene	2.42E-06	0.04	1	6.04E-05	
	1-methynaphthalene	1.94E-05	0.004	1	4.85E-03	
	2-methynaphthalene	8.53E-05	0.004	1	2.13E-02	
	Naphthalene	6.78E-05	0.004	1	1.70E-02	
	Phenanthrene	1.25E-05	0.3	1	4.16E-05	
	Pyrene	7.79E-06	0.03	1	2.60E-04	
						6.04E-02

Hazard Quotient = MDD / ADI
MDD = Maximum Daily Dose (mg/kg-day)
ADI = Acceptable Daily Intake (Reference Dose) (mg/kg-day)
AF = Adjustment factor for reference dose (to correspond to absorbed dose)

Table 6-25: TOTAL HAZARD INDEX ESTIMATES BY RECEPTOR

Receptor	*Exposure Pathway*	*Hazard Index*	*Contribution to Total Hazard Index*	*Total Hazard Index*
Construction Worker, Biocell				**1.14E-01**
	Incidental Ingestion of Soil	3.22E-02	28.293%	
	Dermal Contact with Soil	8.11E-02	71.285%	
	Inhalation of Fugitive Dust	4.79E-04	0.421%	
Construction Worker, On-Site (Non-Biocell)				**4.62E-02**
	Incidental Ingestion of Soil	1.31E-02	28.293%	
	Dermal Contact with Soil	3.29E-02	71.285%	
	Inhalation of Fugitive Dust	1.95E-04	0.421%	
Construction Worker, Off-site				**1.78E-06**
	Incidental Ingestion of Soil	5.03E-07	28.293%	
	Dermal Contact with Soil	1.27E-06	71.285%	
	Inhalation of Fugitive Dust	7.49E-09	0.421%	
Utility Repair Worker				**6.04E-02**
	Dermal Contact With Groundwater	6.04E-02	100.000%	

6.6.3 Uncertainties and Overestimation of Potential Risks

An important facet of the method and use of human health risk assessment concerns the recognition of uncertainties and limitations inherent in the process (EPA, 1989a) which arise in connection with dose-response models, animal to human extrapolation, chemical fate and transport,models of potential exposure, and site-specific receptor characteristics. From a regulatory perspective, these uncertainties and limitations are dealt with by developing and employing assumptions which typically overestimate the magnitude of many variables. When these variables are combined by the additive and multiplicative processes of risk assessment, potential risks are often overestimated.

Factors which contribute significantly to uncertainty and likely overestimation of potential risks associated with the Osmose site are discussed below.

- For the purpose of this assessment, it was conservatively assumed that the current measured concentrations of the chemicals remain constant over time. In actuality, natural degradation processes are likely to result in reduced concentrations over time. Furthermore, there is no known continuing source of contamination on site. It is conservative to assume that concentrations of compounds of potential concern will remain constant over time.

- For this assessment, sampling data reported at or below the detection limit were used in calculating exposure point concentrations. It was assumed that the values for BMDLs were one-half the detection limit. In actuality, the values may be less than the values used in this assessment.

- In this assessment, the 95% upper confidence limit of the arithmetic mean or the maximum detected value, whichever is lower, has been applied as the exposure point concentration. Theoretically, use of this statistic is intended to account for uncertainty in the representativeness of environmental sampling results. Considering the biased distribution of sampling locations at the Osmose site, the arithmetic mean itself is likely to be most representative of the reasonable maximum potential exposure point concentration. Therefore, use of the 95% UCL or maximum values for exposure point concentrations is conservative.

- In this assessment, intake models were developed for hypothetical exposures and, for the most part, utilized values for specific exposure parameters which are not based on research into lifestyle and work habits specific to workers at the Osmose site (e.g., exposure frequency, hygienic practice, outdoor activity patterns). In all cases, actual intakes are not likely to be greater than estimated, and they may be substantially reduced or altogether eliminated by actual personal practices.

6.6.4 Comparable And Acceptable Risk

EPA (1989a) requires risk estimates to be evaluated "in the context of decisions to be made about selection of remedies", including discussion of the site-specific potential cancer risks relative to the NCP range of 10^{-4} to 10^{-6}. This evaluation is appropriate and necessary for site-specific risk management decisions which must consider comparable and acceptable levels of risk.

Based on review of 132 federal regulatory agency decisions, Travis et al., (1987) reported that, when preregulatory risks were less than 1×10^{-6}, no action was taken. When risks exceeded 4×10^{-3}, action was always taken. In a subsequent study of post-regulatory risk levels established for public exposure to 36 chemicals, Travis and Hattemer-Frey (1988) found that 30% of regulatory actions were associated with public health risks greater than 1×10^{-4}.

Federal agencies have accepted risks greater than 10^{-4} for occupational exposures (i.e., small populations). For example, the U.S. Supreme Court has suggested that a lifetime occupational cancer risk of 1×10^{-3} be considered the benchmark for significant risk (Rodricks et al., 1987).

Ultimately, acceptable risk will be defined by the population experiencing the risk, as public opinion and priorities are reflected in regulatory policy. The U.S. Court of Appeals for the District of Columbia has suggested that risks considered acceptable for everyday activities may provide a basis for setting acceptable risk levels for regulatory purposes (Travis and Hattemer-Frey, 1988). Studies of voluntary risk indicate that risks on the order of 1×10^{-2} (car accident) are commonly accepted (Crouch and Wilson, 1982).

6.7 Development of Acceptable Soil Concentrations

The goal of the risk assessment process is to provide a framework which will assist in site-specific remedial decision-making as specified in EPA Guidance (1989b). One specific objective of the risk assessment process includes providing a basis for determining levels of chemicals which can remain on-site and still be adequately protective of public health. The use of risk assessment for determining public-health protective cleanup levels is practiced by federal and state regulatory agencies, and is especially critical when site conditions, technology, and/or economic factors must be considered in the decision-making process.

The quantitative process for determining clean-up levels which are protective of public health involves a reversal of the baseline risk assessment equation (Risk = Intake x Toxicity). Rather than developing a risk estimate for a given contaminant concentration in a particular environmental medium, a predetermined risk level is assigned and the risk equation is solved for contaminant

concentration. For sites which have unacceptable risk levels based on average exposure point concentrations, remediation of selected areas may be desirable in order to reduce site-wide risks to acceptable levels. A contaminant concentration may thus be developed which, when achieved, will result in acceptable public health risk levels. Post-remediation sampling and analysis thus targets this risk-specific concentration.

In this assessment, a residual chemical concentration in soil, termed an Acceptable Soil Concentration (ASC) was developed for PAHs. In determining residual levels of PAHs which would not pose a threat to public health, it was necessary to make certain assumptions regarding toxicity, exposure, and acceptability of risk. Assumptions which were employed in developing an ASC are documented in the following paragraphs and presented in Tables 6-26 and 6-27. Since carcinogenic and noncarcinogenic health effects were evaluated in the baseline risk assessment, separate ASCs were calculated for each of these health endpoints.

In calculating the carcinogenic ASC, it is necessary to select a limit of acceptable risk. Based on review of 132 federal regulatory agency decisions, Travis et al., (1987) reported that, when preregulatory risks were less than 1×10^{-6}, no action was taken. When risks exceeded 4×10^{-3}, action was always taken. In a subsequent study of post-regulatory risk levels established for public exposure to 36 chemicals, Travis and Hattemer-Frey (1988) found that 30% of regulatory actions were associated with public health risks greater than 1×10^{-4}. Federal agencies have accepted risks greater than 10^{-4} for occupational exposures (i.e., small populations). For example, the U.S. Supreme Court has suggested that a lifetime occupational cancer risk of 1×10^{-3} be considered the benchmark for significant risk (Rodricks et al., 1987). On the basis of this precedent, an acceptable risk level of one in one hundred thousand (1×10^{-5}) was selected for this assessment.

The ASC was developed to be protective of reasonably anticipated exposures to soil for identified receptors. Theoretically, all exposure routes would be included in the development of the ASC. However, since inhalation exposures calculated in the baseline risk assessment contributed insignificantly to total risk and hazard index, the dust inhalation exposure route was eliminated from ASC calculations. The ASC therefore reflects combined exposure for the occupational construction worker for the dermal contact and incidental ingestion pathways. Exposure model assumptions utilized in the baseline risk assessment intake were utilized to calculate each ASC; equations were solved for soil concentration.

From the list of PAHs detected in biocell, on-site and off-site soil, one carcinogenic PAH n(benzo(a)pyrene) and one noncarcinogenic PAH (naphthalene) were selected as representative

Table 6-26
Calculation of Acceptable Soil Concentration
Occupational Construction Worker: Combined Dermal Contact and Soil Ingestion Exposures
Carcinogenic

EQUATION

ASC = ARL * BW * AT / (CPF * EF * ED * CF (IR + (SA * AF)))

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*
ASC = Acceptable Soil Concentration	mg/kg	See Below
IR = Ingestion Rate	mg/day	25
SA = Soil Accumulation	mg/day	1086
AF = Absorption Factor	unitless	0.058
CF = Conversion Factor	kg/mg	0.000001
EF = Exposure Frequency	days/year	40
ED = Exposure Duration	years	1
BW = Body Weight	kg	70
AT = Averaging Time	days	27375
CPF = Cancer Potency Factor (Benzo(a)pyrene oral)	(mg/kg/day)-1	11.5
ARL = Acceptable Risk Level	unitless	0.00001 $=1\times10^{-5}$

RESULTS

	Concentration	*Units*
ASC =	473.45	mg/kg

Table 6-27
Calculation of Acceptable Soil Concentration
Occupational Construction Worker: Combined Dermal Contact and Soil Ingestion Exposures
Noncarcinogenic

EQUATION

ASC = ADI * BW / CF * (IR + (SA * AF))

SYMBOLS AND DESCRIPTIONS	*UNITS*	*VALUES*
ASC = Acceptable Soil Concentration	mg/kg	See Below
IR = Ingestion Rate	mg/day	25
SA = Soil Accumulation	mg/day	1086
AF = Absorption Factor	unitless	0.058
CF = Conversion Factor	kg/mg	0.000001
BW = Body Weight	kg	70
ADI = Acceptable Daily Intake (Napthalene oral)	(mg/kg/day)	0.004

RESULTS

	Concentration	*Units*
ASC =	3182.25	mg/kg

indicator compounds. These two PAHs have the most stringent available EPA health criteria (Cancer Potency Factor and Reference Dose) (HEAST, 1991). Consistent with the baseline risk assessment, the oral Cancer Potency Factor of 11.5 $(mg.kg/day)^{-1}$ was selected as the health criterion for benzo(a)pyrene; the RfD of 0.004 mg/kg/day for naphthalene was utilized as the Acceptable Dose (AD). Table 6-26 presents the combined intake models for dermal contact and incidental ingestion of soil at the Osmose site. Assuming an acceptable risk level of 1×10^{-5} (one in one hundred thousand), the calculation indicated that a total PAH concentration of 473 ppm, as 100% benzo(a)pyrene, is protective of human health at this risk level. In fact, at the Osmose site, analytical data indicate that B(a)P comprises on 21% of all carcinogenic PAHs (based on maximum values detected) and only 2.6% of all PAHs (see Table 6-4). An ASC of 473 ppm is, therefore, in all likelihood, protective of worker exposure at this site at the 1-in-a-million risk level.

Table 6-27 presents a similar analysis for non-carcinogenic health effects end points. The calculation indicates that a total PAH concentration of over 3000 ppm in soil on the Osmose site is health-protective. This calculation assumes that all PAHs detected are naphthalene, or a PAH with equivalent toxicity. In fact, Table 6-4 indicates that naphthalene comprises only 12.6% of all non-carcinogenic PAHs and only 11% of total PAHs. An ASC of 3000 ppm is, therefore, protective of site workers for any non-carcinogenic health effects endpoints.

6.8 Summary and Conclusions

This report applies accepted quantitative risk assessment methodology to evaluate compounds of potential concern detected in on-site soils and groundwater, and potential exposures to those compounds associated with hypothetical future exposure scenarios, in order to characterize baseline risks associated with a no-action alternative. The results of this baseline analysis have then been applied in conjunction with site-specific environmental conditions to derive risk based clean-up objectives for on-site soils.

In this report three different areas are addressed relative to soil conditions and potential exposure to compounds of concern in the soil. These areas include the bioremediation cell (biocell), on-site locations east and west of the biocell (on-site), and off-site locations along Ellicott Street adjacent to the site (off-site). These three areas were selected to reflect potential exposure events and exposure conditions corresponding to distinct locations. Relative to groundwater, exposure and risk evaluations were conducted based on data from shallow monitoring wells. Deep groundwater conditions were not addressed in the quantitative exposure and risk evaluations, because there is no indication of either current or future exposure to this groundwater.

Analytical data were generated for the Osmose site based on soil and groundwater samples collected between August 1990 and January 1991 from on-site areas immediately south of the existing building complex and from off-site areas immediately downgradient of the site along Ellicott Street.

Compounds of concern to be carried through the quantitative exposure and risk assessments include 18 different PAH compounds for the hypothetical occupational exposure to soil, in addition to 16 PAHs and BTEX compounds for hypothetical occupational exposure to shallow groundwater. In this assessment, EPA cancer potency factors (CPFs) and Reference Doses (RfDs) as reported in IRIS and HEAST, 1991 are utilized in the risk characterization step. Table 6-8 presents a summary of carcinogenic and noncarcinogenic hazard identification and dose-response for the compounds included in the quantitative evaluation.

In order to evaluate potential receptors on or near the Osmose site, it is necessary to determine the location of current populations relative to the site. The land use around the Osmose site is primarily residential, commercial, and vacant lots. Residential neighborhoods are located immediately adjacent to the site; however, access to the site is restricted. In addition, there is no evidence that occupational exposure currently exists on site. Overall, there is no evidence for exposure under current use conditions. Currently, there are no plans in place to develop the Osmose site for any purpose other than its present industrial use. Relative to future exposures, utility and construction workers represent a potentially exposed population. The extent of exposure would vary according to the activities of the workers and the location of their activities.

PAHs are present in the soil in a specific area of the Osmose site, immediately south of the main building complex, presumably as a result of historical small-scale spillage during tank filling and transfer. Utility and construction workers may contact compounds on concern in soil at several exposure points, both on and off the site. Potential contact with biocell soils is of particular interest as a subset of on-site soil and is treated as a separate exposure point. Exposure routes associated with the soil/worker pathway are dermal contact with contaminated soil, incidental ingestion of soil, and inhalation of fugitive dust from the soil. Low concentrations of volatile organics (BTEX) and PAHs were detected in shallow groundwater wells (MW-9, MW-10, MW-11) immediately downgradient of the site. Utility workers may be in contact with compounds of concern in off-site shallow groundwater while servicing underground utility lines. The potential exposure route associated with the shallow groundwater is dermal contact. The intake calculation models, with resulting estimates of Lifetime Average Daily Dose (LADD) and Average Daily Dose (ADD), for the exposure pathways included in the quantitative evaluation are shown in Tables 6-12 through 6-15.

Under the conservative assumptions and parameter values used in this assessment, estimates of total carcinogenic risk for the hypothetical on-site biocell receptor, the on-site (non-biocell) receptor, the hypothetical off-site worker, as well as the hypothetical utility repair worker exposed to groundwater, are well below the criterion of acceptable risk (1×10^{-5}) by factors of approximately 10 to 10,000, representing a large "margin of safety" for any of these potential receptors. Relative to evaluation of noncarcinogenic effects, hazard quotients were calculated for each chemical; and hazard indices were estimated for each exposure pathway. To assess the overall potential for noncarcinogenic effects, hazard indices posed by the exposure pathways (dermal contact, incidental ingestion, and inhalation) were summed. The hazard index estimates for individual chemicals, by receptor and exposure pathway, are shown in Tables 6-21 through 6-24. In addition, total hazard index estimates by receptor are presented in Table 6-25. The total hazard index for the hypothetical on-site biocell worker is approximately 1.1×10^{-1} ; for the on-site (non-biocell) worker is approximately 4.6×10^{-2}; and for the off-site worker is approximately 1.8×10^{-6}. Likewise, the total hazard index for the hypothetical utility repair worker exposed to groundwater is approximately 6×10^{-2}. All of these values indicate little likelihood for adverse noncarcinogenic effects to occur to any of these hypothetical receptors.

In this assessment, a residual chemical concentration in soil, termed an Acceptable Soil Concentration (ASC) was developed for PAHs. Since carcinogenic and noncarcinogenic health effects were evaluated in the baseline risk assessment, separate ASCs were calculated for each of these health endpoints. On the basis of decision-making precedent by federal regulatory agencies, an acceptable risk level of one in one hundred thousand (1×10^{-5}) was selected for this assessment. Because inhalation exposures calculated in the baseline risk assessment contributed insignificantly to total risk and hazard index, the dust inhalation exposure route was eliminated from ASC calculations. The ASC therefore reflects combined exposure for the occupational construction worker for the dermal contact and incidental ingestion pathways. Exposure model assumptions utilized in the baseline risk assessment intake were utilized to calculated each ASC; equations were solved for soil concentration. From the list of PAHs detected in biocell, on-site, and off-site soil, one carcinogenic PAH (benzo(a)pyrene) and one noncarcinogenic PAH (naphthalene) were selected as representative indicator compounds. These two PAHs have the most stringent available EPA health criteria (Cancer Potency Factor and Reference Dose) (HEAST, 1991). Assuming an acceptable risk level of 1×10^{-5} (one in one hundred thousand), the calculation indicated that a total PAH concentration of 473 ppm, as 100% benzo(a)pyrene, is protective of human health at this risk level. Relative to the ACL analysis for non-carcinogenic health effects end points, the calculation indicates that a total PAH concentration of over 3000 ppm in soil on the Osmose site is health-protective.

7.0 CONCLUSIONS

7.1 Remediation Goals and Objectives

The objective of this section is to present remediation goals and objectives for each specific media as described in the Risk Assessment (Section 6.0). These goals represent closure criteria that are protective of short and long term adverse health and environmental impacts. The following specific medias have remediation goals and objectives proposed:

- Soils contained within the biocell (Biocell Soils)
- On-site, non-biocell soils (On-site soils)
- Off-site, downgradient soils (Off-site soils), and
- On-and Off-site groundwater (Groundwater)
- Separate phase product

Separate ASCs were developed for carcinogenic and non-carcinogenic PAHs in soils. (refer to Risk Assessment, Section 6.0). From the list of PAHs detected in biocell, on-site, and off-site soils, the following ASCs were found to possess acceptable risks:

- Total Carcinogenic PAHs: 473 ppm
- Total Noncarcinogenic PAHs: 3182 ppm

The above ASC's were conservatively developed by assuming all PAHs possess the carcinogenic characteristics of benzo{a}pyrene and the noncarcinogenic hazard index for naphthalene.

7.1.1 Biocell Soils

Based upon the results of the Risk Assessment, Osmose proposes to operate the biocell until total PAH levels in the soils are at, or below 473 mg/kg. At the time when each individual composite soil sample from each of the 5 sampling locations possesses total PAH concentrations below 473 ppm, a petition for closure will be submitted.

7.1.2 On-site Soils

Only one location at the Osmose site contained adsorbed PAH levels in exceedance of the 473 ppm upper bound for acceptable risk as determined by the Risk Assessment. This location was in the area of the former coal bin (MW-8) from 2' - 4' below grade where 500.9 mg/kg total PAHs were found. A soil boring is proposed downgradient (east) of MW-8 to collect data which will provide

evidence that these PAHs are not associated with the former UST system (two discrete areas). Soil samples will be collected and analyzed using standard laboratory protocols.

7.1.3 Off-site Soils

Current data exists which shows total PAH levels in off-site soils ranges between non-detectable to 71.8 μg/kg. These levels are far below the 473 mg/kg level which represents the conservative upper bound for acceptable carcinogenic risk. No remedial action, therefore, is proposed for off-site soils.

7.1.4 Groundwater

The Risk Assessment addressed potential risks associated with exposure to PAHs in on- and off-site groundwater. The existing total carcinogenic risk estimate for groundwater is approximately 1×10^{-8}, which is considerabley less than the criterion for acceptable risk. Likewise, the total hazard index for noncarcinogenic risks is approximately 6×10^{-2} (far below unity) which represents acceptable risks.

No remedial action for on- or off-site groundwater is proposed. Quarterly monitoring and sampling of wells MW-8, MW-9, MW-10, MW-11 and cluster well CW-1 for PAH and BTEX analytes by standard lab protocols is proposed to monitor groundwater quality over time. One soil boring, completed as an FRP monitor well will be installed in the overburden west of the Osmose facility to monitor upgradient water quality. The boring will be placed so as to also provide additional soils information which will help delineate adsorbed PAH levels in the area surrounding MW-8. Details of monitor well location and installation will be provided under separate cover.

7.1.5 Separate Phase

Separte phase LNAPL and an intermittent DNAPL have been detected in on site PVC monitor wells MW-3, MW-5 and MW-7. Due to the intermittent nature, and typical thickness of the product layers(< 0.1'), manual gauging and bailing twice per week from the existing monitor wells is proposed. Recovered product will be stored in DOT approved 55 gallon drums until sufficient quantities exist for proper disposal. If the product layer(s) persist, an automatic product recovery system will be installed.

In addition, delineation of the separate phase product plume will be addressed by the installation of downgradient of existing monitor wells MW-3 and MW-5. The well will be completed in the shallow overburden and will be gauged on a twice weekly basis.

SOIL-

Samples Taken at Vapor Point Locations

Volatiles mg/kg	Detection Limit	8/25/90 VP-12 *
Toluene	11.0	4.0
Ethyl Benzene	1.9	5.0
Xylenes (total)	2.2	10.3
BTEX (total)	6.5	19.0

* All other vapor point analyses were non-detect.

Samples from Monitoring Well Locations

Volatiles mg/kg	Detection Limit	10/1/90 MW-8 2-4'	10/5/90 MW-9 10-12'	10/2/90 CW-1 6-8'	10/2/90 CW-1 8-10'	10/2/90 CW-1 30-32'
Toluene	0.25	-	-	-	0.25	-
Xylenes (total)	0.85	-	-	-	4.2	-
BTEX (total)	-	-	-	-	4.4	-
Misc. Aromatics (C8-C10)	5.0	49.0	4.4	55.0	170.0	5.5
Total Hydrocarbons	-	49.0	4.4	55.0	170.0	5.5

SOIL

Samples Taken at Vapor Point Locations

METALS mg/kg	Detection Limit a	8/25/90 VP-5	VP-8	VP-13	VP-15	VP-16	VP-17	VP-18
Arsenic	0.5	J 17	J 36	J 4.1	J 18	J 3.9	J 3	J 3.8
Cadmium	0.5	1.2	1.1	< 0.6	2.5	< 2.7	0.92	< 0.6
Chromium	1.0	32	19	14	33	14	11	14
Copper	2.5	41	64	52	73	30	27	53
Lead	10.0	J 810	J 610	J 200	J 820	J 310	J 410	J 260
Mercury	0.2	0.68	1.9	0.3	1.2	1.1	0.88	< 0.25
Nickel	4.0	0.68	18	22	21	< 22	12	21
Selenium	0.5	J 0.65	J 0.84	< 0.6	J 1.1	< 0.6	< 0.56	J 0.57
Zinc	2.0	380	270	140	860	380	450	170

Samples from Monitoring Well Locations

METALS mg/kg	Detection Limit	10/1/90 MW-8 2-4'	MW-8 16-18'	MW-9 4-6'	10/5/90 MW-9 10-12'	10/6/90 MW-10 10-12'	MW-10 6-8'
Arsenic	5	cs 10	+ 2.2	ds 6.6	s 2.6	s 1.1	c+ 16
Beryllium	0.5	4.2	< 0.34	< 0.35	< 0.33	< 0.37	0.43
Chromium	1	11	5	13	4.3	4.6	15
Copper	2.5	< 9.8	7	14	6	6.4	15
Lead	10	< 39	9.7	24	8.5	9.9	12
Nickel	4	< 16	5.3	17	4.2	5.2	19
Zinc	2	20	52	60	49	66	56

c Detection Limit Multiplier = 8.00.

d Detection Limit Multiplier = 8.94.

s The reported value was determined by the Method of Standard Addition.

\+ The correlation coefficient for the Method of Standard Addition is less than 0.995.

a Test Methods for Evaluating Solid Waste, SW-846, Third Edition, Revision), US EPA November 1986; digestion by EPA Method 3050 (ICP and Furnace). Results are reported on a dry weight basis. Various multipliers have also been used.

Soil PAH Concentrations

Off-Site Soil*

Analyte ug/kg	Detection Limit	SB-1 4-6' 10/15/90	MW-9 4-6' 10/5/90	MW-9 10-12' 10/5/90	MW-9 30-32' 10/5/90	MW-10 6-8' 10/15/90	MW-10 10-12' 10/15/90	MW-11 4-6' 10/16/90	MW-11 10-12' 10/16/90
Acenaphthene	60	< 71	< 71	< 68	< 73	< 71	< 67	< 70	< 68
Anthracene	22	< 26	< 26	< 25	< 27	< 26	< 25	< 26	< 25
Benzo(a)anthracene	0.43	2.7	1.7	< 0.49	< 0.52	3.8	1.7	4.9	< 0.49
Benzo(a)pyrene	0.77	4.1	1.7	< 0.87	< 0.93	4.6	< 0.88	6.2	< 0.88
Benzo(b)fluoranthene	0.6	3.7	1.7	< 0.68	< 0.73	4.9	2.2	6.6	< 0.68
Benzo(g,h,i)perylene	2.5	< 3	< 3	< 2.8	< 3	< 3	< 1.1	6.3	< 2.8
Benzo(k)fluoranthene	0.57	1.5	0.88	< 0.64	< 0.69	2	< 0.64	2.9	< 0.65
Chrysene	5	< 6	< 6	< 5.6	< 6.1	< 6	< 5.6	< 5.8	< 5.7
Dibenzo(a,h)anthracene	1	< 1.2	< 1.2	< 1.1	< 1.2	< 1.2		< 1.2	< 1.1
Fluoranthene	7	< 8.3	< 8.3	< 7.9	< 8.5	< 8.3	< 7.8	11	< 8
Fluorene	7	< 8.3	< 8.3	< 7.9	< 8.5	< 8.3	< 7.8	< 8.1	< 8.1
Indeno(1,2,3-cd)pyrene	1.4	< 1.7	< 1.7	< 1.6	< 1.7	< 1.7	2.8	5.9	< 1.6
1-Methylnaphthalene	60	< 71	< 71	< 68	< 73	< 71	< 67	< 70	< 68
2-Methylnaphthene	60	< 71	< 71	< 68	< 73	< 71	< 67	< 70	< 68
Naphthalene	60	< 71	< 71	< 68	< 73	< 71	< 67	< 70	< 68
Phenanthrene	21	< 25	< 25	< 24	< 25	< 25	< 24	< 24	< 24
Pyrene	9	14	< 11	< 10	< 11	27	16	28	< 10
Detection Limit Multiplier		1.19	1.19	1.13	1.21	1.19	1.12	1.16	1.14

On-Site Soil *

Analyte ug/kg	Detection Limit	MW-8 2-4' 10/1/90	MW-8 16-18' 10/1/90	CW-1 6-8' 10/2/90	CW-1 8-10' 10/2/90	CW-1 30-32' 10/2/90	CW-1 62-64' 10/3/90	CW-2 6-8' 10/15/90
Acenaphthene	60	3200	< 73	8000	40000	< 68	300	1700
Anthracene	22	180000	< 27	27000	63000	57	720	27000
Benzo(a)anthracene	0.43	17000	1.8	1400	4700	4.4	53	980
Benzo(a)pyrene	0.77	18000	1.6	9.3	97	3.1	21	450
Benzo(b)fluoranthene	0.6	14000	1.5	490	1600	3.1	20	530
Benzo(g,h,i)perylene	2.5	13000	< 3	260	991	< 2.8	11	280
Benzo(k)fluoranthene	0.57	7600	< 0.69	290	980	1.5	11	290
Chrysene	5	15000	< 6.1	1100	3700	< 5.6	47	1100
Dibenzo(a,h)anthracene	1	3700	< 1.2	20	120	< 1.1	1.6	53
Fluoranthene	7	43000	< 8.5	9000	28000	21	320	5000
Fluorene	7	3200	< 8.5	6700	29000	12	260	1300
Indeno(1,2,3-cd)pyrene	1.4	10000	< 1.7	49	360	< 1.6	3.6	88
1-Methylnaphthalene	60	1200	< 73	2600	15000	< 68	82	350
2-Methylnaphthene	60	4000	< 73	4100	30000	< 68	160	820
Naphthalene	60	12000	< 73	8400	77000	< 68	160	2100
Phenanthrene	21	36000	< 25	22000	62000	36	670	6500
Pyrene	9	120000	< 11	15000	41000	44	490	10000
Detection Limit Multiplier		11.4	1.21	1.20	1.16	1.13	1.11	1.00

The detection limit multiplier indicates the adjustment made to the data and detection limits as a result of dilutions and percent solids.

* Only samples taken at depths less than 10 feet are carried through the exposure assessment

GROUNDWATER DATA: VOLATILES

Volatiles ug/l	Detection Limit	EPA Method 602 11/9/90 CW-1*		D-1		MW-8		MW-9		MW-10		MW-11	
Benzene	0.2	B	15.0	b	0.7	b	0.2		150.0	b	0.2	B	0.7
Ethyl Benzene	0.8		1.6	<	0.8	<	0.5		76.0	<	0.5	<	0.5
Toluene	0.5		4.9	<	0.5	<	0.8		9	<	0.8	<	0.8
Xylenes (total)	1.7		12	<	1.7	<	1.7		66	<	1.7	<	1.7
BTEX (total)	-		34		0.7	b	0.2		300	b	0.2	b	0.7
Misc. Aliphatics (C4-C12)	15		70	<	15	<	15	<	15	<	15	<	15
Misc. Aromatics (C8-C10)	10		140	<	10	<	10		470	<	10	<	10
Total Hydrocarbons	-		240		0.7	b	0.2		770	b	0.2	b	0.7
				Dup. of MW-8									

Volatiles ug/l	PQL ug/l	EPA Method 8240 11/9/90 CW-1*	
Acetone	10.0	J	7.7
Benzene	5.0		10.0
Ethyl Benzene	5.0	U	5.0
Toluene	5.0	J	4.1
Xylenes (total)	5.0		6.0
BTEX (total)			

Volatiles ug/l	Detection Limit	EPA Method 8020 1/11/91 CW-1*		1/11/91 MW-11		1/10/91 MW-9		1/10/91 DW-1		1/14/91 MW-10		1/14/91 MW-8	
Benzene	0.2	X	49.0		0.2	X	81.0	X	82.0	U	0.2	U	0.2
Ethyl Benzene	0.8		2.7	U	0.8		14.0		14.0	U	0.5	U	0.5
Toluene	0.5		8.5	U	0.5	X	90.0	X	100.0	U	0.8	U	0.8
Xylenes (total)	1.7		25.0	U	1.7	X	74.0	X	76.0	U	1.7	U	1.7
BTEX (total)	-	X	85.0		0.2	X	260.0	X	270.0				
Misc. Aliphatics (C4-C12)	15		68.0	U	15.0	U	15.0	U	15.0	U	15.0	U	15.0
Misc. Aromatics (C8-C10)	10		400.0		51.0		380.0		390.0	U	10.0	U	10.0
Total Hydrocarbons	-	X	550.0		51.0	X	640.0	X	660.0				
								Dup of MW-9					

X - Estimated concentration. Exceeded the calibrated linear range of the instrument.

U - Analyzed for but not detected

b - See Nonconformance Section 1.2

B - Indicates that the analyte was found in the blank as well as a sample. It indicates possible/probable blank contamination and warns the data user to take appropriate action.

J - Estimated concentration

* CW-1 is a deep well. Only results from the shallow wells were carried through the exposure scenarios.

GROUNDWATER DATA: TOTAL METALS

METALS ug/l	Detection Limit	11/9/90 MW-8	CW-1*	D-1	MW-11
Arsenic	5.0	< 5.0	< 5.0	6.3	< 5.0
Cadmium	5.0	< 5.0	6.7	< 5.0	< 5.0
Chromium	10.0	< 10.0	19.0	< 10.0	< 10
Lead	5.0	< 5.0	< 5.0	< 5.0	6.9
Zinc (c)	20.0	44	140.0	34.0 Dup of MW-11	39

METALS ug/l	Detection Limit	1/10/91 DW-1	MW-9	1/11/91 CW-1*	MW-11	1/14/91 MW-10	MW-8
Arsenic	5.0	B 4.9	< 4.5	< 4.5	< 4.5	< 4.5	B 8.0
Cadmium	5.0	< 5.0	< 5.0	< 5.0	< 5.0	< 5.0	< 5.0
Chromium	10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10.0	< 10
Lead	5.0	10.8	10.0	< 1.5	8.2	B 1.8	8.3
Zinc (c)	20.0	80.3 Dup of MW-9	104.0	< 20.0	51.6	20.4	41.1

B - Indicates that the analyte was found in the blank as well as a sample. It indicates possible/probable blank contamination and warns the data user to take appropriate action.

* CW-1 is a deep well. Only results from the shallow wells were carried through the exposure scenarios.

GROUNDWATER DATA: POLYNUCLEAR AROMATIC HYDROCARBONS (PAHs)

PAHs ug/l	Detection Limit	EPA Method 610 11/9/90 CW-1*	D-1	MW-11	MW-8	MW-9	MW-10
Naphthalene	1.8	51	< 1.8	< 1.8	< 1.8	< 2	< 1.8
1-Methylnaphthalene	1.8	4	< 1.8	< 1.8	< 1.8	< 2	< 1.8
2-Methylnaphthene	1.8	5.9	< 1.8	< 1.8	4.6	< 2	< 1.8
Acenaphthene	1.8	3.6	< 1.8	< 1.8	< 1.8	< 2	< 1.8
Fluorene	0.21	0.96	< 0.21	< 0.21	< 0.21	< 0.23	< 0.21
Phenanthrene	0.64	1.9	< 0.64	1.1	< 0.64	< 0.71	< 0.64
Anthracene	0.66	5.6	< 0.66	< 0.65	< 0.66	< 0.73	< 0.66
Fluoranthene	0.21	0.66	0.29	< 0.21	< 0.21	< 0.23	< 0.21
Pyrene	0.27	1.5	0.55	0.29	0.29	< 0.3	< 0.27
Benzo(a)anthracene	0.013	0.16	0.08	0.04	< 0.013	< 0.014	< 0.013
Benzo(b)fluoranthene	0.018	0.22	0.098	0.049	0.06	< 0.02	< 0.018
Benzo(k)fluoranthene	0.017	0.11	0.051	0.026	0.029	< 0.019	< 0.017
Benzo(a)pyrene	0.023	0.22	0.12	0.054	0.061	< 0.026	< 0.023
Dibenzo(a,h)anthracene	0.03	0.054	< 0.03	< 0.03	< 0.03	< 0.033	< 0.03
Benzo(g,h.i)perylene	0.076	0.23	0.12	< 0.076	< 0.076	< 0.084	< 0.076
Indeno(1,2,3-cd)pyrene	0.043	0.16	0.088	< 0.043	< 0.043	< 0.048	< 0.043
			Dup-MW-11				

PAHs ug/l	Detection Limit	EPA Method 8310 1/10/91 MW-9	DW-1*	1/14/91 MW-10	MW-8	1/11/91 CW-1*	MW-11
Naphthalene	1.8	7.8	6.6	U 1.8	U 1.8	160	U 1.8
1-Methylnaphthalene	1.8						
2-Methylnaphthene	1.8						
Acenaphthene	1.8	U 1.8	U 1.8	U 1.8	U 1.8	5.9	U 1.8
Fluorene	0.21	U 0.21	U 0.21	U 0.21	U 0.21	1.3	U 0.21
Phenanthrene	0.64	U 0.64	U 0.64	U 0.64	U 0.64	1.3	U 0.64
Anthracene	0.66	U 0.66	U 0.66	U 0.66	U 0.66	U 0.66	U 0.66
Fluoranthene	0.21	U 0.21	U 0.21	U 0.21	U 0.21	0.36	U 0.21
Pyrene	0.27	U 0.27	U 0.27	U 0.27	U 0.27	0.54	U 0.27
Benzo(a)anthracene	0.013	U 0.013	U 0.013	0.026	U 0.013	0.068	U 0.013
Benzo(b)fluoranthene	0.018	U 0.018	U 0.018	0.03	U 0.018	0.05	U 0.018
Benzo(k)fluoranthene	0.017	U 0.017	U 0.017	U 0.017	U 0.017	0.028	U 0.017
Benzo(a)pyrene	0.023	U 0.023	U 0.023	0.027	U 0.023	0.047	U 0.023
Dibenzo(a,h)anthracene	0.03	U 0.03	U 0.03	U 0.03	U 0.03	U 0.03	U 0.03
Benzo(g,h.i)perylene	0.076	U 0.076	U 0.076	U 0.076	U 0.076	U 0.076	U 0.076
Indeno(1,2,3-cd)pyrene	0.043	U 0.043	U 0.043	U 0.043	U 0.043	U 0.043	U 0.043
			Dup-MW-9				

U - Analyzed for but not detected

* CW-1 is a deep well. Only results from the shallow wells were carried through the exposure scenarios.

GROUNDWATER - BASE/NEUTRALS & ACIDS

Semi-Volatiles ug/l	EPA Method 8270 PQL		11/13/90 CW-1*
Naphthalene	10		35.0
2-Methylnaphthalene	10	J	3.3
Acenaphthene	10	J	3.1
Dibenzofuran	10	J	1.1

GROUNDWATER - PURGEABLE HALOCARBONS

Purgeable Halocarbons ug/l	Detection Limit		1/11/91 CW-1*		1/11/91 MW-11
1,2-Dichloroethane	0.68	Z	3.4	<	0.68
1,1,1-Trichloroethane	0.53	Z	0.91		0.84
Detection Limit Multiplier			1		1

Z = Estimated concentrations. Surrogate recovery could not be accurately quatitated.

* CW-1 is a deep well. Only results from the shallow wells were carried through the exposure scenarios.

POLYNUCLEAR AROMATIC HYDROCARBONS

1.0 INTRODUCTION

Polynuclear Aromatic Hydrocarbons (PAHs) are a class of organic chemicals which consist of carbon and hydrogen. The structure of these chemicals incorporates two or more fused benzene rings in linear, angular or cluster arrangements. PAHs in the environment are a result of both natural and anthropogenic sources. Most of the direct releases of PAHs into the environment are to the atmosphere. Incomplete combustion or uncontrolled emissions of PAHs from residential burning of wood results in the largest release of PAHs to the atmosphere (IARC, 1983). PAHs can enter surface water through atmospheric deposition, from discharges of industrial effluents and municipal wastewater, and from improper disposal of used motor oil. Depending on the source and impacted media in the environment, human exposure routes may include inhalation, ingestion and dermal contact (ATSDR, 1989c).

1.1 Physical and Chemical Properties

PAHs have a high affinity for organic matter and generally exhibit a low water solubility. Water solubility of PAHs decreases, and affinity for organic material increase with higher molecular weight. When present in soil or sediments, PAHs tend to remain bound to the soil particles and dissolve slowly into ground water or the perched zone.

Henry's Law Constant, the ratio of a chemical in air and in water at equilibrium, indicates the ability of a chemical to volatilize. The low molecular weight PAHs have Henry's Law constants in the range of 10^{-3} to 10^{-5} atm-m^3/mol; medium molecular weight PAHs are in the 10^{-6} range and high molecular weight PAHs have values in the range of 10^{-5} to 10^{-6}. Compounds with values less than 10^{-5} exhibit a limited volatilization while those compounds with a value of 10^{-3} to 10^{-5} are associated with significant volatilization. It is estimated that high molecular weight PAHs have atmospheric half-lives of approximately 100 hours; low molecular weight PAHs have atmospheric half-lives of around 18 hours. However, half-life values for individual PAH compounds are highly variable and depend on environmental conditions (ATSDR, 1989c).

Physical and chemical properties of selected PAHs are presented in Tables 1 through 14.

TABLE 1

Physical and Chemical Properties of Anthracene

Property	Value	Reference
Molecular formula	$C_{14}H_{10}$	IARC,1983
Molecular weight	178.2	ATSDR,1989c
Appearance	Pure: colorless solid violet fluorescence	ATSDR,1989c
Melting point	218° C	ATSDR,1989c
Boiling point	342° C	ATSDR,1989c
Solubility		ATSDR,1989c
water	insoluble	
organic solvents	benzene, carbon disulfide, chloroform, ether, ethanol methanol, toluene	
Vapor pressure	$1.7E^{-06}$ mmH_g @ 25° C	ATSDR,1989c
Henry's Law constant	$8.6\ E^{-06}$	ATSDR,1989c
Density	1.25 @ 27/4° C	IARC,1983
Partition coefficients	log K_{ow} = 4.45 K_{oc} = 1.4 E^{+04}	ATSDR,1989c
Flashpoint	250° F	ATSDR,1989c

TABLE 2

Physical and Chemical Properties of Benzo[a]Anthracene

Property	Value	Reference
Molecular formula	$C_{18}H_{12}$	IARC, 1989
Molecular weight	228.29	ATSDR, 1988a
Appearance	yellow-blue fluorescence	ATSDR, 1988a
Melting point	158-159° C	ATSDR, 1988a
Boiling point	400° C	ATSDR, 1988a
Solubility		ATSDR, 1989c
water	9-14 mg/L	
organic solvents	hot ethanol and acetic acid acetone and diethyl ether benzene	
Vapor pressure	2.2 E^{-08} mm Hg @ 20° C	ATSDR, 1988a
Henry's Law constant	1 x E^{-06} atm-m^3/mol	ATSDR, 1988a
Density	1.274 @ 20° C	ATSDR, 1988a
Partition Coefficients	K_{ow} = 4.1 E^{+05} K_{oc} = 2 x E^{+05}	ATSDR, 1988a

TABLE 3

Physical and Chemical Properties of Benzo[b]Fluoranthene

Property	Value	Reference
Molecular formula	$C_{20}H_{12}$	IARC,1983
Molecular weight	252.3	IARC,1983
Appearance	colorless solid	ATSDR,1988c
Melting point	168.3° C	IARC,1983
Solubility water organic solvents	14 µg/L benzene, acetone	ATSDR,1988c
Vapor pressure	1 E^{-11} to 1 E^{-08} mm Hg @ 20° C	ATSDR,1988c
Henry's Law constant	1.22 E^{-05} atm-m^3/mol	ATSDR,1988c
Partition coefficients	K_{ow} = 1 E^{+06}	ATSDR, 1988c

TABLE 4

Physical and Chemical Properties of
Benzo[k]Fluoranthene

Property	Value	Reference
Molecular formula	$C_{20}H_{12}$	IARC, 1983
Molecular weight	252.3	IARC, 1983
Appearance	pale-yellow solid	IARC, 1983
Melting point	215.7° C	ATSDR, 1989c
Boiling point	480° C	ATSDR, 1989c
Solubility water organic solvents	 insoluble soluble in benzene, acetic acid, ethanol	ATSDR, 1989c
Vapor pressure	5 E^{-07} mm Hg @ 20° C	ATSDR, 1989c
Henry's Law constant	3.87 E^{-06} atm-m^3/mol	ATSDR, 1989c
Partition coefficient	K_{ow} = 1.15 E^{+06}	ATSDR, 1989c

TABLE 5

Physical and Chemical Properties of Benzo[a]Pyrene

Property	Value	Reference
Molecular formula	$C_{20}H_{12}$	IARC,1983
Molecular weight	252.3	IARC,1983
Appearance	pale-yellow	ATSDR,1989a
Melting point	179-179.3° C	ATSDR,1989a
Boiling point	495° C	ATSDR,1989a
Solubility water organic solvents	 3.8 E^{-06} g/L sparingly soluble in methanol, ethanol; soluble in benzene, toluene and ether	ATSDR,1989a
Vapor pressure	5.6 E^{-09} mm Hg	ATSDR,1989a
Henry's Law constant	4.9 E^{-07} atm-m^3/mol	ATSDR,1989a
Density	1.351	ATSDR,1989a
Partition coefficients	K_{ow} = 1.55 E^{+06} K_{oc} = 5.5 E^{+06}	ATSDR,1989a

TABLE 6

Physical and Chemical Properties of Chrysene

Property	Value	Reference
Molecular formula	$C_{18}H_{12}$	IARC,1983
Molecular weight	228.3	IARC,1983
Appearance	colorless with blue fluorescence	ATSDR,1988b
Melting point	255-256° C	ATSDR,1988b
Boiling point	448° C	ATSDR,1988b
Solubility		ATSDR,1988b
water	1.5-2.2 µg/L	
organic solvents	slightly soluble in acetone, carbon disulfide, diethyl ether, ethanol, soluble in benzene	
Vapor pressure	6.3 E^{-09} mm Hg @ 20° C	ATSDR,1988b
Henry's Law constant	1.05 E^{-06} atm-m^3/mol	ATSDR,1988b
Density	1.274	ATSDR,1988b
Partition coefficients	K_{ow} = 4.1 E^{+05} K_{oc} = 2.0 E^{+05}	ATSDR,1988b

TABLE 7

Physical and Chemical Properties of Dibenzo[a,h]Anthracene

Property	Value	Reference
Molecular formula	$C_{22}H_{14}$	IARC,1983
Molecular weight	278.4	ATSDR,1988d
Appearance	Colorless	ATSDR,1988d
Melting point	262° C	Eller, 1984
Boiling point	269-270° C	ATSDR,1988d
Solubility		ATSDR,1988d
water	0.5 µg/L	
organic solvents	slightly soluble in ethyl alcohol; soluble in acetone, benzene, toluene and xylene.	Weast, 1988
Vapor pressure	1 E^{-10} mm Hg @ 20° C.	ATSDR,1988d
Henry's Law constant	7.3 E^{-8} atm-m^3/mol	ATSDR,1988d
Density	1.282	ATSDR,1988d
Partition coefficients	K_{ow} = 6.9 E^{+06} K_{oc} = 3.3 E^{+08}	ATSDR,1988d

TABLE 8

Physical and Chemical Properties of Fluoranthene

Property	Value	Reference
Molecular formula	$C_{16}H_{10}$	IARC,1983
Molecular weight	202.26	IARC,1983
Appearance	pale yellow	ATSDR,1989c
Melting point	111° C	ATSDR,1989c
Boiling point	375° C	ATSDR,1989c
Solubility water organic solvents	0.20-0.26 mg/L @ 25° C alcohol, ether, benzene, acetic acid	IARC,1983
Vapor pressure	0.01 mm HG @ 20° C	ATSDR,1989c
Henry's Law constant	6.5 E^{-06} atm -m^3/mol	ATSDR,1989c
Density	1.252 @ 4° C	ATSDR,1989c
Partition Coefficients	log K_{ow}= 4.90 log K_{oc}= 4.58	ATSDR,1989c

TABLE 9

Physical and Chemical Properties of Fluorene

Property	Value	Reference
Molecular formula	$C_{13}H_{10}$	IARC, 1983
Molecular weight	166.2	IARC, 1983
Appearance	white flakes	IARC, 1983
Melting point	116-117° C	IARC, 1983
Boiling point	295° C	IARC, 1983
Solubility		IARC, 1983
water	1.68-1.98 mg/L	
organic solvents	acetone, benzene, carbon tetrachloride, ethanol	
Vapor pressure	10 mm Hg @ 146° C	IARC, 1983
Henry's Law constant	6.4 E^{-06} atm -m^3/mol	ATSDR,1989c
Density		
Partition Coefficients	K_{ow} 1.5 E^{+04} K_{oc} 7.3 E^{+03}	ATSDR,1989c

TABLE 10

Physical and Chemical Properties of
Indeno[1,2,3-cd]pyrene

Property	Value	Reference
Molecular formula	$C_{22}H_{12}$	IARC,1983
Molecular weight	276.3	Eller, 1984
Appearance	Yellow to greenish-yellow fluorescence	ATSDR,1989c
Melting point	163.6° C	IARC, 1983
Boiling point	530° C	ATSDR,1989c
Solubility		ATSDR,1989c
water	Insoluble in water	
organic solvents	Soluble	IARC, 1983
Vapor pressure	1 E^{-10} mm Hg @ 20° C	Mabey, 1982
Henry's Law constant	6.95 E^{-08} atm-m^3/mol	ATSDR,1989c
Partition coefficients	K_{ow} = 3.8 E^{+06} K_{oc} = 1.6 E^{+06}	Mabey, 1982

TABLE 11

Physical and Chemical Properties of 2-Methylnaphthalene

Property	Value	Reference
Molecular formula	$C_{11}H_{10}$	ATSDR, 1989b
Molecular weight	142.2	ATSDR, 1989b
Appearance	solid @ 25° C	ATSDR, 1989b
Melting point	34.6° C	ATSDR, 1989b
Boiling point	241° C	ATSDR, 1989b
Solubility		ATSDR, 1989b
water	insoluble @ 20° C	
organic solvents	soluble in ethanol ether, benzene	
Density	1.0058 @ 20° C	ATSDR, 1989b

TABLE 12

Physical and Chemical Properties of Naphthalene

Property	Value	Reference
Molecular formula	$C_{10}H_8$	ATSDR,1989b
Molecular weight	128.16	ATSDR,1989b
Appearance	white solid	ATSDR,1989b
Melting point	80.5° C	ATSDR,1989b
Boiling point	218° C	ATSDR,1989b
Solubility		ATSDR,1989b
water	31.7 mg/L @ 20° C	
organic solvents	benzene, ethanol,	
ether, acetone		
Vapor Pressure	0.087 mmHg @ 25° C	ATSDR,1989b
Henry's Law constant	4.6×10^{-4} atm-m^3/mol	ATSDR,1989b
Density	1.145 @ 20° C	ATSDR,1989b
Partition coefficients	$K_{ow} = 1.9\ E^{+03}$ $K_{oc} = 9.3\ E^{+02}$	ATSDR,1989b
Bioconcentration Factors	40-300	ATSDR,1989b
Rainbow trout, bluegill sunfish		

TABLE 13

Physical and Chemical Properties of Phenanthrene

Property	Reference	Value
Molecular formula	$C_{14}H_{10}$	IARC,1983
Molecular weight	178.2	IARC,1983
Appearance	colorless crystals	ATSDR,1989c
Melting point	100° C	ATSDR,1989c
Boiling point	340° C	ATSDR,1989c
Solubility		ATSDR,1989c
water	1.6 mg/L	
organic solvents	benzene, carbon disulfide carbon tetrachloride	
Vapor pressure	9.6 E^{-06} torr @ 25° C	ATSDR,1989c
Henry's Law constant	2.26 E^{-04}	ATSDR,1989c
Density	0.900 @ 4° C	ATSDR,1989c
Partition coefficients	K_{ow} = 2.8 E^{+04} K_{oc} = 1.4 E^{+04}	ATSDR,1989c

TABLE 14

Physical and Chemical Properties of Pyrene

Property	Value	Reference
Molecular formula	$C_{16}H_{10}$	IARC,1983
Molecular weight	202.3	IARC,1983
Appearance	pale yellow	ATSDR,1989c
Melting point	156° C	ATSDR,1989c
Boiling point	385° C	IARC,1983
Solubility		ATSDR,1989c
water	insoluble	
organic solvents	benzene, carbon disulfide, diethyl ether, ethanol	
Vapor pressure	2.5 E^{-06} mm Hg @ 25° C	ATSDR,1989c
Henry's Law constant	5.1 E^{-06}	ATSDR,1989c
Density	1.271 @ 23° C	ATSDR,1989c
Partition coefficients	K_{ow} = 7.6 E^{+04} K_{oc} = 3.8 E^{+04}	ATSDR,1989c

1.2 Environmental Fate

PAHs may be present in air, water, sediment and soil. In each of these media, environmental fate processes are dependent upon the inherent chemical properties associated with each PAH. Because there are limited data regarding the environmental fate and transport of specific PAHs, this section will consider the behavior of PAHs as a group.

In the atmosphere, PAHs are either sorbed to particulates or exist in the gaseous phase. They are subject to short and long distance transport and are removed by both wet and dry deposition. The size of particulate material to which a PAH is sorbed determines the atmospheric residence time and transport distance. In urban environments, PAHs are typically sorbed to soot particles of submicron diameter and, therefore, may be subject to long range transport. In contrast, PAHs sorbed to large particles tend to deposit shortly after release to the atmosphere.

PAHs in the atmosphere can react with ozone, nitrogen oxides, sulfur dioxide and peroxyacetylnitrate. Reaction products may include diones, nitro- and dinitro- PAH and sulfonic acids. The photooxidation of PAHs may result in the formation of mutagenic compounds including quinones, phenols and dihydrodiols. The rate of oxidation is dependent upon the physical and chemical properties of the adsorbent. The atmospheric half-lives of these products are usually less than 30 days.

Degradation of PAHs in soil occurs primarily via microbial decomposition. The rate and extent of this process is characterized by several environmental factors. These factors include the presence of microbial populations, soil pH, temperature, oxygen concentration, soil type, moisture, nutrients and substrate metabolites.

In water, PAHs can volatilize, photodegrade, oxidize, biodegrade, bind to particulates or accumulate in aquatic organisms (ATSDR, 1989c). In aquatic systems, PAHs tend to bind strongly to sediment or particles that are suspended in the water column. PAHs with higher molecular weights exhibit increasing affinity to soil and sediment. In sediments, PAHs can biodegrade or accumulate in aquatic organisms.

The most important processes resulting in the degradation of PAH in water are photooixidation , chemical oxidation and biodegradation by aquatic microorganisms. Many factors influence the rate and extent of photodegradation; including water depth, turbidity, and temperature. Under aerobic conditions, PAH can be metabolized by microbes such as aquatic bacteria and fungi, whereas under anaerobic conditions degradation is extremely slow. Other removal processes from water include: volatilization to the atmosphere, binding to particulates or sediments, or accumulation or sorption onto aquatic biota.

Henry's Law states that the ratio of the chemical in air and water are at equilibrium and indicates the ability

of a chemical to volatilize. The low molecular weight PAH's have Henry's Law constants in the range of 10^{-3} to 10^{-5} atm-m^3/mole; medium molecular weight in the 10^{-6} range, and high molecular weight PAH have values in the range of 10^{-5} to 10^{-8}. Compounds with values less than 10^{-5} volatilize to a limited extent, while those compounds with a value of 10^{-3} to 10^{-5} are associated with significant volatilization. It is estimated that high molecular weight PAH's have atmospheric half-lives around 100 hours and low molecular wight PAH half-lives are around 18 hours. However, the half-life values are highly variable and depend on the environmental conditions (ATSDR, 1989c).

1.3 Toxicity to Aquatic Species

Polycyclic aromatic hydrocarbons are ubiquitous chemicals that are normally not considered to be acutely toxic to aquatic organisms because they are only sparingly soluble in water. In general, PAH concentrations that are acutely toxic to aquatic organisms are several orders of magnitude higher than concentrations found in even the most heavily polluted waters, excepting circumstances of oil spills (Neff, 1979). The LC_{50} values reported by Eisler (1987) for several species of aquatic organisms demonstrate this trend (Table 15). Sediments from heavily polluted areas, however, may contain PAH concentrations similar to those which are acutely toxic, but their limited bioavailability in sediments apparently renders them substantially less toxic than PAHs in aqueous solution (Neff, 1979).

PAHs vary substantially in their toxicity to aquatic organisms. Acute toxicity to aquatic organisms has been demonstrated primarily for the small PAHs (up to three rings) and only at relatively high concentrations (Neff, 1979; Korn et al., 1979; Cairns and Nebeker, 1982; DeGraeve et al., 1982; Eastmond et al., 1984; Edmisten and Bantle, 1982; Giddings, 1979; Govers et al., 1984; Lee and Nicol, 1978a; Lee and Nicol, 1978b; Sabourin, 1982; Soto et al., 1975; Holcombe et al., 1983). Acute toxicity appears to increase with molecular weight within that group; however, when the molecular weight reaches that of the three-ring compounds, an aqueous concentration equivalent to the solubility in water is required to elicit acute toxicity (LC_{50}) (Neff, 1979). The acute toxicity of PAHs with a higher molecular weight has been demonstrated for some compounds, but only by using a carrier solvent at concentrations above the solubility of the PAH in water. At concentrations less than the aqueous solubility limit, the higher molecular weight, less soluble, compounds have not been shown to be acutely toxic (Neff, 1979). However, most of these studies were carried out under gold fluorescent lights to avoid photodegradation of the parent compound, and possible photoinduced toxicity was not observed. For this reason, most work on PAH effects has focused on chronic toxicity, since many of the homologous series are potentially mutagenic and carcinogenic to organisms, including fish (Schultz and Schultz, 1982; Black, 1983a; Black, 1983b; Baumann et al., 1982).

Table 15

LC_{50} VALUES FOR PAHs IN AQUATIC ORGANISMS

Species	PAH	Concentration (mg/L)*
Sandworm (*Neanthes arenaceodentata*)	Benzo(a)pyrene	>1.0
	Chrysene	>1.0
	Fluoranthene	0.5
	Fluorene	1.0
	Naphthalene	3.8
	Dimethylnaphthalenes	2.6
	Trimethylnaphthalenes	2.0
	Phenanthrene	0.6
Grass shrimp (*Paleomonetes pugio*)	Fluorene	0.3
	Naphthalene	2.4
	2-Methylnaphthalene	1.1
Amphipod (*Gammarus pseudolimnaeus*)	Fluorene	0.6
Amphipod (*Elasmopus pectenicrus*)	Naphthalene	2.7
Dungeness crab (*Cancer magister*)	Naphthalene	2.0
	1-Methylnaphthalene	1.9
	2-Methylnaphthalene	1.3
Mosquito Fish (*Gambusia affinis*)	Naphthalene	150.0
Sheepshead minnow (*Cyprinodon variegatus*)	Fluorene	1.7
Coho salmon, fry (*Oncorhyncus kisutch*)	Naphthalene	3.2
Rainbow trout (*Salmo gairdneri*)	Fluorene	0.8
Bluegill sunfish (*Leopomis macrochirus*)	fluoranthene	3.98
Cladoceran (*Daphnia magna*)	fluoranthene	325

* Assay type not reported
Sources: Eisler, 1987; EPA, 1980

Sensitivity to phototoxicity also may vary within a single species. In some preliminary screening studies with *Daphnia magna*, time to toxicity was examined for 3-methylcholanthrene (3-MC), B[a]P, dimethylbenz[a]anthracene (DMBA) and anthracene in paired experiments. DMBA was more phototoxic than B[a]P, while B[a]P was more or equally as toxic as 3-MC, and both were more toxic than anthracene (Landrum et al., 1987; Leversee, 1984).

Compounds with carcinogenic potential typically require enzymatic transformation to the active intermediate metabolites by means of the mixed function oxidase (MFO) system (Knutzen, 1987). Consequently, the presence of a MFO system appears to be a prerequisite for an organism to develop cancer from PAH exposure. It follows that organisms with high MFO activity should be most susceptible, and that the lack of this enzyme system should confer protection against cancer. The highest MFO activity has been found in fish, whereas the activity in mussels and snails typically is very low (Knutzen, 1987). Although neoplasms have been observed in mussels chronically exposed to petroleum, the most serious effect from PAHs will be accumulation in these organisms. PAH concentration in mussels and snails may reach three orders of magnitude higher concentrations than the normal levels (Knutzen, 1987).

Huggett et al. (1987) discussed the distribution of abnormalities in fish in relation to sediment contamination levels in the Elizabeth River in Virginia. Fishery surveys were conducted during October, November and December of 1983 at 11 stations along the river. Depressions in biomass, total numbers of individuals and abundance of selected species occurred at the more contaminated stations. There was also a great increase in the rate of structural abnormalities. Eleven percent of hogchokers (*Trinectes maculatus*) and 30% of toadfishes (*Opsanus tau*) collected in the most contaminated areas showed fin erosion. The incidence of cataracts in spot (*Leiostomus xanthurus*), croaker (*Micropogonias undulatus*) and weakfish (*Cynoscion regalis*) was 10, 18 and 21%, respectively, in the contaminated zone. PAH residues of 60 ug/g (60 mg/kg) were attained from oysters at the most contaminated station after a nine-week exposure period. Fish collected from this area showed the highest incidences of abnormalities.

In many cases, aquatic organisms from PAH contaminated environments exhibit a higher incidence of tumors and hyperplastic diseases than those from nonpolluted environments. Neoplasms in several species of fish have been produced experimentally with 3-methylcholanthrene, acetylaminofluorene, B[a]P and 7,12-dimethylbenzo(a)anthracene, with tumors evident within 3 to 12 months after exposure (Couch and Harshbarger, 1985; Hendricks et al., 1985). Under laboratory conditions, liver neoplasms were induced in two species of minnows (*Poeciliopsis spp.*) by repeated short-term exposures (6 hours once a week for 5 weeks) to an aqueous suspension of 5 mg/L 7,12-dimethylbenzo(a)anthracene. About 44% of the fish surviving this first treatment developed hepatocellular neoplasms within six to nine months after exposure (Schultz and Schultz, 1982). Eastern mudminnows (*Umbra pygmaea*) which were kept in water containing

up to 700 ug/l PAH for 11 days showed increased frequencies of chromosomal aberrations in gills: 30% vs. 8% in controls (Prein et al., 1978).

The state of Florida has set its surface quality standards based on U.S. EPA water quality criteria. The Florida standard for Class I (potable surface water) is 0.0028 ug/L. This is based on the EPA lifetime cancer risk of 10^{-6}, and accounts for both human ingestion of water and ingestion of aquatic organisms. The Florida standard for Class II and III waters is 0.0311 ug/L, and is based on EPA estimates made for consumption of aquatic organisms only, again assuming a lifetime cancer risk of 10^{-6}. U.S. EPA (1980b) concluded that there is insufficient data to regulate individual PAHs, and therefore total PAHs are the subject of the standards. The EPA standards are based on the assumption that each compound is as potent as B[a]P and the carcinogenic effect of the compounds is proportional to the sum of their concentrations (U.S. EPA, 1980b). Since they are based on EPA criteria, the state of Florida standards also refer to total PAHs. Florida provides additional standards for two individual PAHs based on other data, such as organoleptic data which suggests that certain concentrations of PAHs may taint water or fish flesh. The fluoranthene standard is 42 ug/L for Class I and 54 ug/L for Class II and III, while the acenaphthene standard is 20 ug/L for Class I, Class II and III (Chapter 17-302, FAC; proposed 1990).

1.4 Toxicokinetics

Factors which influence the toxicokinetics of PAHs include solubility, particulate adsorption and biotransformation. The oral and inhalation uptake of PAHs is well studied in experimental animals,but no human studies are available. Human dermal studies have demonstrated evidence of PAH absorption, but due to high variability in urinary metabolites, no absorption rates have been estimated. The distribution and metabolism of several PAHs has been well studied in animals and is highly dependent upon the specific chemical properties of the PAH. The urinary elimination of PAH is generally rapid but may vary according to the route of administration and the absorbed dose.

1.4.1 Absorption

Absorption of PAHs has been studied predominantly in rodents. PAHs can be taken into the body via inhalation, ingestion, or skin contact, although the compounds typically are poorly absorbed from the gastrointestinal tract (Eisler, 1987). PAHs are readily absorbed following inhalation exposure to PAH vapors or PAHs attached to dust and other particles. Due to the production of PAH's through combustion processes, exposure to PAHs in soil may occur in areas where coal, wood, gasoline and other products historically have been burned. The following are descriptive summaries of the available studies for each absorption route: inhalation, ingestion and dermal.

1.4.1.1 Animal Studies

Absorption Following Inhalation

Many animal studies are available regarding the absorption of B(a)P following inhalation exposure, though the specific route of administration was of variable relevance. Sun et al. (1982) administered B(a)P vapor at a concentration of 0.6 ug/L or B(a)P adsorbed on Ga_2O_3 particles (at a concentration of 1 ug/L). After 30 minutes of exposure, the fraction deposited in the lung was approximately 20% for Ga_2O_3 and approximately 10% for the pure hydrocarbon aerosol. B(a)P excretion was monitored for over two weeks, at which time nearly all the B(a)P had been recovered, indicating complete absorption and elimination of the initially instilled hydrocarbon. Significant differences in the clearance of Ga_2O_3 adsorbed and pure B(a)P suggested that a substantial amount of B(a)P coated on Ga_2O_3 particles was removed from the lungs by mucociliary clearance and subsequent ingestion. The B(a)P retained by the lungs was removed by absorption into the blood stream. The association of B(a)P with the particles increased the deposition of B(a)P in the lung and increased the relative amount of B(a)P that was cleared by mucociliary action and subsequently ingested. Hence, there was an increase in the absorption of B(a)P in the alimentary tract, which increased the dose of B(a)P and its metabolites to the stomach, liver and kidneys relative to that observed for pure B(a)P vapor.

The size of the particles on which B(a)P is adsorbed affects the pulmonary absorption and elimination of this chemical. For example, the elimination of B(a)P from the lungs was studied following intratracheal administration of pure B(a)P crystals in comparison with B(a)P coated on carbon particles in two size ranges (0.1-1.0 um and 15-30 um; Cresia et al., 1976). Whereas, 50% of the pure B(a)P crystals was eliminated from the lungs within 1.5 hours and 95% within 24 hours, the B(a)P adsorbed to the small carbon particles took 36 hours to clear 50% of the initial dose. Pulmonary elution was slower with the larger carbon particle size (approximately 4-5 days).

Intratracheal administration of B(a)P (0.001 mg/kg) to rats resulted in rapid absorption. Concentrations in the liver reached a maximum of 21% of the administered dose within 10 minutes of installation. Presence of B(a)P and its metabolites in other tissues and the bile was also indicative of its absorption. Similar results were also reported for guinea pigs and hamsters following intratracheal exposure (Weyand and Bevan, 1986; Weyand and Bevan, 1987; Weyand and Bevan, 1988).

Nasal instillation of B(a)P (0.13 mg/kg) to hamsters resulted in the metabolism of B(a)P in the nasal cavity. A large fraction of the metabolites was recovered from the epithelial surface, indicating that B(a)P was first absorbed in the mucosa, metabolized, and returned to the mucus (Dahl et al., 1985).

Monkeys and dogs received nasal instillation of B(a)P at doses of 0.16-0.21 mg/kg. B(a)P metabolites were

detected in the nasal cavity, but only to a limited extent in the blood and excreta of either species during the 48 hours after exposure. These results indicate that there was either little or a very slow rate of direct transfer of B(a)P or its metabolites into the blood by this route (Petridou-Fisher et al., 1988).

Approximately 50% of the B(a)P that was instilled intratracheally in hamsters was metabolized in the nasal tissues. The metabolites produced in the hamster nose included tetrols, 9,10-dihydroldiols, 4,5-dihydrodiols, 7,8-dihydrodiols, quinones, 3-phenols, and 9-phenols. A prevalence of quinone production was not observed in hamsters as it was in rats (Dahl et al., 1985). *In vitro* metabolism of B(a)P in the ethmoid turbinates of dogs resulted in a prevalence of phenol metabolites. However, small quantities of quinones and dihydrodiols also were identified (Bond et al., 1988).

The absorption and elimination of B(a)P from rat and mouse lungs are very rapid. Eighty-five percent of a single intratracheal instillation of 2.5 mg/kg B(a)P was cleared from the lungs of a mouse after 24 hours (Schnizlein et al., 1987). In the rat lung, 40% of a B(a)P dose was cleared within five minutes, and >94% was cleared within six hours (Weyand and Bevan, 1986). In the latter study, a large fraction of the administered dose was excreted in the bile. In general, the rate of B(a)P excretion into bile declined as the dose increased. Excreted metabolites included thioether (62.5%), glucuronide (22.8%) and sulfate (7.4%) conjugates, as well as free B(a)P (9.8%). Significant species differences in pulmonary absorption are apparent, based on the fact that only 10% of an intratracheal dose of B(a)P was excreted in the urine and feces of dogs and monkeys after 48 hours (Petridou-Fisher et al., 1988).

The effect of dose on the pulmonary clearance of B(a)P in the rat was studied by intratracheal instillation of [^{14}C]-B(a)P (16, 90, and 6400 µg of hydrocarbon). Clearance was determined to be biphasic with a fast component (half-life ≤ 1 day) and a slow component (half-life ≥ 1 day). As dose increased (16-6400 µg B(a)P), an increased percentage (from 89 to 99.76%) was cleared with a half-life ≤ 1 day and a decreased percentage was cleared (from 11 to 0.24%) with a half-life ≥ 1 day (Medinsky and Kampcik, 1985). The slower component half-life is clearly subject to saturation at the high dose levels.

Absorption Following Ingestion

Gastrointestinal absorption of B(a)P has been studied in the rat. B(a)P was administered to rats by gavage (0.04 umol, 0.4 umol, 4.0 umol.). Total excretion of the dose in the feces averaged 74-79% at 48 hours vs. 85% at 168 hours following administration (indicating approximately 15-26% absorption). Only 1-3% of the administered dose was excreted in the urine. The amount of parent compound which was excreted decreased as the dose increased (Hecht et al., 1974). B(a)P was absorbed in Sprague-Dawley rats following oral administration and was detected in the liver, lung and kidney (Yamazaki et al., 1987).

Oral absorption of benzo(a)anthracene in rats was reported to be rapid and efficient. Levels of

benzo(a)anthracene in the blood, liver, and brain reached a maximum within 1-2 hours after administration (Modica et al., 1982).

Intestinal absorption of chrysene was not quantified, but the extent of absorption in rats was dependent on the oral dose of chrysene and the vehicle of administration. Approximately 25-41% of the chrysene dose (indicating approximately 59-75% absorption) was recovered in the feces within 72 hours after administration in olive oil (Modica et al., 1982). Chang (1943) reported an excretion of 79% of the chrysene dose (21% absorption) in the feces following dietary (500 mg/kg) and gavage (200 mg/kg) administration.

Administration of dibenzanthracene (DBA) in the diet (250 mg) or by stomach tube (200 mg) resulted in greater than 90% of the dose being excreted (indicating approximately 10% absorption) in the feces of white rats (Chang, 1943). As with chrysene, absorption of DBA was not quantified directly.

Chang (1943) studied the intestinal absorption of PAHs administered to rats by gastric intubation in starch solution. The data indicated that different agents were absorbed to different extents and that no more than 50% of the dose given of the carcinogenic substances tested was absorbed. The percent of material found in the feces relative to the amount administered was chrysene, 85%; dibenz(a,h)anthracene, 95%; benzo(a)pyrene, 57%; 3-methylcholanthrene, 68% (maximum 43% absorption). In contrast, the non-carcinogenic agent, phenanthrene, was almost completely absorbed.

In rats which were administered radiolabeled naphthalene, the amount of label recovered in 24 hours was 77 to 93% in urine and 6 to 7% in feces, indicating over 90% absorption (Bakke, 1985). In rats, Summer et al. (1979) found a dose-dependent increase in urinary mercapturic acid excretion following gavage doses of naphthalene approximately 39, 32, and 26% of each dose, respectively, with 24 hours.

3-MC or B(a)P was given in the diet of lactating rats, rabbits and ewes and the excretion of materials via the milk was determined (West and Horton, 1976). In rats, 0.19% of the total dose was excreted via the milk within four hours. In rabbits, only 0.003% was excreted in 24 hours and in ewes, 0.01% was excreted by seven days. Almost all of the material fed to sheep was recovered from the feces, indicating very little absorption via the intestine.

In general, PAHs absorption following the ingestion of contaminated food or drinking water depends on the vehicle of administration. The extent of PAHs absorption is enhanced when they are solubilized in a vehicle that is itself readily absorbed, such as oils.

Absorption Following Dermal Contact

Evidence regarding PAH distribution in animals following dermal exposure is limited. Although the compounds may penetrate the skin, very little is distributed to tissues by this route of exposure. Only 1.3 % of an applied dose of anthracene (9.3 $\mu g/cm^2$) was detected in tissues of rats at six days after administration (Yang et al., 1986). Animal studies with dimethylbenzanthracene (DMBA) suggest that absorption and distribution of many PAHs compounds through the skin is not extensive (ATSDR, 1990).

Percutaneous absorption of B(a)P in mice was reported after monitoring the appearance of B(a)P in excreta and at the site of application. Disappearance of the applied dose from the application site was 6% and 40% at 1 and 24 hours following administration, respectively. Within seven days after exposure, 93% of the applied dose was recovered in the feces (Sanders et al., 1986).

The percutaneous absorption of anthracene in rats (9.3 $\mu g/cm^2$) resulted in approximately 52% of the dose being absorbed in a dose-dependent manner within 6 days. Diffusion of anthracene through the skin (*stratum corneum*) was dependent upon the amount of anthracene on the skin surface as well as the surface area to which the anthracene was applied (Yang et al., 1986).

Metabolism of chrysene at relatively high rates in mouse skin provides evidence of its dermal uptake (Hodgson, 1983; Weston et al., 1985). DBA also was absorbed dermally, but not to the extent reported for B(a)P. Sanders et al. (1986) applied DBA (5.4, 56, 515 $\mu g/cm^2$) and B(a)P (1.25-125 $\mu g/cm^2$) to the shaved nuchal area. The presence of PAHs in the skin at the application site, in excreta, and in exhaled air were monitored. At 24 hours after the maximum dose of DBA was applied, 67.2% of the dose was recovered from the application site, 25.4% from the body tissues, and 7.4% from excreta (approximately 30% absorption). Under similar conditions with B(a)P, 17.4% 36.6%, and 46.8% of the dose was recovered from the application site, tissue, and excreta, respectively (approximately 80% absorption). The amount which was absorbed did not increase linearly with the dose due to an apparent saturation of the uptake process. The authors suggested that the difference in dermal uptake among the PAHs may be attributable to the lower rate of DBA metabolism relative to B(a)P and decreased rate of metabolite transfer (Sanders et al., 1986).

Monitoring the removal of compounds from the epidermis is indicative of the compound's dermal absorption. The half-life of B(a)P and its metabolites in the epidermis was approximately 2 hours (Melikian et al., 1987). Recovery of B(a)P was 99-100% throughout the period of the experiment (8 hours), indicating the volatilization of B(a)P from the skin was not a confounding factor (Melikian et al., 1987). In contrast, removal of one of its metabolites, 7,8-dihydroxy-9,10-epoxy-7,8,9,10-tetrahydro-B(a)P (anti-BPDE), from the epidermis was slower, suggesting that the *stratum corneum*, the outermost layer of skin which consists

of several layers of inactive, keratinized cells surrounded by extracellular lipids, may act as a reservoir that can retain and slowly release topically applied lipophilic substances such as B(a)P, but which is penetrated rapidly by more polar metabolites.

Thus, PAHs may be absorbed through the skin of animals to a variable extent, and contact with soil or water contaminated with PAHs may result in systemic exposure to these compounds, based on the limited available information.

1.4.1.2 Human Studies

Absorption Following Inhalation

No quantitative studies were found regarding the absorption of PAHs in humans following inhalation exposure. However, absorption of PAHs following inhalation can be inferred from the presence of urinary metabolites of PAHs in workers following exposure to these compounds in an aluminum plant (Beecher and Bjorseth, 1983). The high concentration of PAHs in the occupational setting did not correspond to the amount of PAHs deposited, metabolized and excreted in the urine in this study. The authors suggested that PAHs which are adsorbed to airborne particulate matter may not be bioavailable, and that the dose-uptake relationship may not be linear over the PAH concentration range.

Absorption Following Ingestion

No quantitative studies were found regarding the absorption of PAHs in humans following oral exposure.

Absorption Following Dermal Contact

PAHs may be absorbed through the skin of humans. Application of 2% crude coal tar to the skin of humans for eight hour periods on two consecutive days yielded evidence of PAH absorption (Storer et al., 1984). Phenanthrene, anthracene, pyrene, and fluoranthene were detected in the blood, but B(a)P was not detected; thus, absorption of PAHs in crude coal tar was variable and dependent on the chemical species. This variability in blood concentration was attributed to differences in the rate of percutaneous absorption, rapid tissue deposition after absorption, or metabolic conjugation with subsequent rapid urinary excretion. An *in vitro* study using human skin found that the extent of permeation after 24 hours was established as 3% of an applied dose of ^{14}C-B(a)P applied at 10 ug/cm^2 (Kao et al., 1985).

The relative rate of dermal penetration of B(a)A painted on the skin of mice was determined to be similar to that of benzo(a)pyrene (Bock and Burnham, 1960). The concentrations in the skin, as detected by fluorometry, reached a maximum 2 hours after topical application of a 1% solution of the hydrocarbons. The permeation rate for B(a)P in the mouse for a 24 hour exposure has been reported as 10% (Kao et al., 1985) and 40% (Sanders et al., 1986) of an applied dose of 10 µg/cm^2 and 1.25 to 125 µg/cm^2 of [^{14}C]-B(a)P, respectively. Evidence of dermal absorption in animals is found in the carcinogenicity studies

summarized in IARC (1973).

The skin penetration of an applied dose of (^{14}C) B(a)P (10 µg/cm^2) was determined in several mammalian species under *in vitro* conditions (Kao et al., 1985). Dorsal skin from marmoset, guinea pig, rabbit, rat, and mouse were used for the permeation experiments. The mouse showed the highest permeation at 10% (24 hours), followed by the rat, rabbit, and marmoset (1 to 3%); the guinea pig exhibited the lowest permeation at 0.1%.

The dermal uptake of DB(a,h)A was studied in mice (Heidelberger and Weiss, 1951). In these investigations, a single application of ^{14}C-DB(a,h)A (0.2 µmol) dissolved in benzene was applied to the shaved skin of the sacral region of mice. The sites of application were then dissected and fractionated. An average of 8% of the applied dose was absorbed after 2 days. This rate of dermal absorption was significantly lower than that determined for B(a)P following application of an equivalent dose (Heidelberger and Weiss, 1951).

1.4.2 Distribution and Retention

Distribution of B[a]P in the rat following inhalation indicates that the highest concentrations occur in the lungs, liver, kidney and gastrointestinal tract. B[a]P was concentrated in the protein fractions of the liver, lungs and kidney of orally dosed rats. Significant biotransformation of PAHs occurs in the liver, lung and kidney. The liver plays the major role in biotransformation of PAHs. The biotransformation may lead to the formation of more reactive metabolites (ATSDR, 1989a).

Orally administered PAHs may cross the placenta. PAHs (1.53-1.6 ug/g)were detected in the fetuses of pregnant rats administered an oral dose of 200 mg/kg of a PAH mixture (ATSDR, 1989a).

1.4.3 Metabolism

In mammals, the cytochrome mixed-function oxidase (MFO) system, a portion of which is represented by aryl hydrocarbon hydroxylase (AHH), is responsible for initiating the metabolism of various lipophilic organic compounds, including PAHs. The relevant effect of this system is to convert poorly water soluble, lipophilic materials into more water soluble congeners and thereby increase the rate of excretion (Eisler, 1987; Williams and Burson, 1985).

The activity of this enzyme system is readily induced by exposure to PAHs and other chemicals and is found in most mammalian tissues, although predominantly in the liver. The MFO system is involved in the metabolism of endogenous substances (e.g., steroids) and the detoxification of many xenobiotics. Paradoxically, however, some PAHs are transformed by this system to intermediate metabolites which have been identified as more toxic, mutagenic, teratogenic, or carcinogenic agents than the parent compound

(U.S. EPA, 1980a; Eisler, 1987). Metabolic activation by the MFO system appears to be a necessary prerequisite for PAH-induced carcinogenesis and mutagenesis (Neff,1979).

The MFO system, specifically AHH, can convert PAHs to various oxygenated/hydroxylated derivatives including phenols, quinones, dihydrodiols, and epoxides. These oxygenated metabolites may be converted further to less toxic products such as water soluble conjugates of glutathione, glucoronides and sulfates. As noted previously, the MFO system may also activate PAHs to produce carcinogenic metabolites (Eisler, 1987; DiGiovanni, 1989; Yang, 1988).

Since PAHs are composed of aromatic rings with little else to metabolize, hydroxylation by the MFO system is the first step in the biological action of PAH metabolism to more water soluble forms that can be readily excreted. In the process, highly electrophilic and unstable arene oxides, epoxides in particular, may by generated. Arene epoxides can bind covalently to cellular macromolecules such as DNA, RNA and proteins. Covalent interaction with DNA appears to be critical to the initiation of PAH-induced carcinogenesis. However, the simple or initial epoxide metabolites are not the ultimate carcinogens (Williams and Burson, 1985; DiGiovanni, 1989).

Another component of the drug metabolizing enzyme system, epoxide hydrolase, can transform arene epoxides to dihydrodiols, which are precursors of biologically active diol epoxides. These secondary diol epoxides have been shown to be more potently mutagenic and carcinogenic than the primary metabolites because they form DNA adducts which are more resistant to DNA-repair processes (Williams and Burson, 1985; Eisler, 1987; Jerina et al., 1986).

In particular, the "bay region" diol epoxides (i.e., epoxides formed at the juncture of two angularly fused rings) (Mohammad, 1984), have been implicated as reactive products in PAH carcinogenesis (Eisler, 1987; Jerina et al., 1986). PAHs that possess a bay region that is metabolized to a diol epoxide derivative are very reactive. Carcinogenesis studies *in vivo* and mutagenesis and transformation assays *in vitro* indicate that the biologic effects of the parent compound can be mimicked by treating the respective animal or cell line with metabolites of PAH-containing diol epoxides in the bay region (Zedeck, 1980).

1.4.4 Excretion

Elimination is generally rapid following all routes of exposure to PAHs. A single intratracheal instillation of 2.5 mg/kg in the lung resulted in clearance of 85% of the administered dose after 24 hours. Rats eliminate a large fraction of the administered dose in the bile following inhalation exposure. After 6 hours, 53% was excreted into the intestine and intestinal contents of rats without a cannula, and 74% in rats with a cannula (ATSDR, 1989c). Rats dosed orally with chrysene excreted 90% in the feces (ATSDR, 1989c).

1.8 Toxicity of Selected PAHs

In the following sections, chemical-specific discussions for the PAHs of interest are presented, including physical and chemical properties, genotoxicity, animal toxicity, and human toxicity. PAHs classified as carcinogens are presented first, followed by PAHs which are classified as noncarginogens or have not been classified as to carcinogenicity. In addition, Table 16 presents carcinogenic weight-of-evidence determinations by IARC and EPA, as available; Table 17 lists Federal and State of Florida Regulations, Standards and Guidelines for PAHs as a compound class, and for individual PAHs, as available.

2.0 BENZ[a]ANTHRACENE

Benzo[a]thracene (B[a]A) is not produced or used commercially. It is formed during incomplete combustion and is a major component of the total PAHs found in the environment (ATSDR, 1988a). Physical and chemical properties of B[a]P are listed in Table 2.

2.1 Genotoxicity

B[a]A has been examined for potential genotoxic effects in a variety of short-term bioassays. The metabolism of B[a]A is an essential event in producing genotoxic effects in both *in vitro* and *in vivo* biological test systems. B[a]A tested positive for genotoxicity in the host-mediated gene mutation assay with *Salmonella typhimurium* strain TA1535 (Simmon et al., 1979; Poirier and de Serres, 1979). Rosenkranz and Poirier (1979) reported no genotoxic response with the microsomal-mediated Ames assay. Results were also negative for DNA damage in *E. coli* and mutations in *Saccharomyces cerevisiae* (Rosenkranz and Poirier, 1979; Simmon, 1979). B[a]A exhibited mutagenic potential (Barfknecht et al., 1982; Rocchi et al., 1980) and produced DNA damage in cultured animal and human cells (Martin et al., 1978).

2.2 Animal Toxicity

Oral absorption of B[a]A in rats was reported to be rapid and efficient. Levels of benzo(a)anthracene in the blood, liver, and brain reached a maximum within 1-2 hours after administration (Modica et al., 1982).

Orally administered benzo(a)anthracene is distributed rapidly and widely in the rat (Bartosek et al., 1984). Maximum concentrations in well-perfused tissues, like the liver, blood and brain, were achieved within 1-2 hours after administration. Maximum levels in lesser perfused tissues, like adipose and mammary tissue, were reached in 3-4 hours. At 72 hours after oral administration, B[a]A had the greatest affinity for adipose tissue, sequentially followed by mammary gland, brain, liver, and blood.

The relative rate of dermal penetration of B[a]A painted on the skin of mice was determined to be similar to that of benzo[a]pyrene (Bock and Burnham, 1960). The concentrations in the skin, as detected by

fluorometry, reached a maximum 2 hours after topical application of a 1% solution of B[a]A. The permeation rate for benzo[a]pyrene in the mouse for a 24-hour exposure has been reported as 10% (Kao et al., 1985) and 40% (Sanders et al., 1986) of an applied dose of 10 $\mu g/cm^2$ and 1.25 to 125 $\mu g/cm^2$ of [^{14}C]benzo[a]pyrene, respectively. Evidence of dermal absorption in animals is found in the carcinogenicity studies summarized in IARC (1973).

The metabolism of B[a]A in human and animal systems apparently proceeds via the biotransformation pathways established for benzo[a]pyrene (Cooper et al., 1983, Levin et al., 1982, Sims 1982, Thakker et al., 1982).

The induction of preneoplastic hepatocytes, known as GGT foci, in animals has been correlated with cancer promotion. A one day intragastric administration of 200 mg/kg of benzo(a)anthracene to partially hepatectomized rats followed by a diet containing 2-acetylaminofluorene and carbon tetrachloride induced GGT foci (Tsuda and Farber, 1980).

The ability to induce aldehyde dehydrogenase (ADH) in animals has also been correlated with carcinogenic potency. Rats intragastrically administered 100 mg/kg/day of benzo(a)anthracene for four days exhibited cytosolic ADH induction (Torronen et al., 1981). Exposure to benzo(a)anthracene also increased the relative liver weights by 19%, (Torronen et al., 1981).

Lymphoid effects have been observed in mice following subchronic weekly subcutaneous injections of benzo(a)anthracene for 40 weeks (Hoch-Ligeti, 1941). This treatment resulted in gross changes in the lymphoid system including an increase in stem cells, an accumulation of iron, reduced lymphoid cells and dilated lymph sinuses. Spleen weight in treated mice was significantly lower than that observed in controls (Hoch-Ligeti, 1941).

Many, but not all, 4, 5 and 6 ring PAH compounds exhibit carcinogenic activity, but only a few unsubstituted hydrocarbons with 7 rings or greater are tumorigenic or carcinogenic (Neff, 1979; U.S. EPA, 1980b; Dipple, 1985). The unsubstituted PAHs with less than four condensed rings that have been tested have not shown tumorigenic activity. Of the six possible arrangements with four benzene rings, only two of these compounds are active: benzo[c]phenanthrene and benzo[a]anthracene.

Certain PAHs, including benzo(a)anthracene, have been shown to be carcinogenic in animals following exposure by the oral route. Mice acutely administered 2 mg benzo[a]anthracene by gavage for two days exhibited increased incidences (80% and 85%) of hepatomas and pulmonary adenomas after 568 days of observation (Klein, 1963). No malignant tumors were observed in this study. However, a single gavage administration of 0.5 mg benzo[a]anthracene produced no tumors in mice after 68 weeks. Multiple gavage

administration resulted in the occurrence of forestomach papillomas in 7% of the animals compared to none in the controls (Bock and King, 1959).

Two subchronic studies in which B[a]A was administered by gavage provide evidence of its carcinogenic potential. Mice that received intermittent doses of 1.5 mg/kg/day B[a]A for 5 weeks (Klein, 1963) or for unspecified intermediate lengths of time (Bock and King, 1959) exhibited significantly elevated incidences of hepatomas and lung adenomas following up to 60 days of observation (Klein, 1963). Neither of these studies were adequately reported; they did not include complete histopathology, adequate treatment durations, large enough sample sizes or statistical analysis. In addition, the authors did not report whether B[a]A produced malignant tumors. Although these studies are inconclusive because of methodological limitations, they do provide some qualitative evidence for the potential carcinogenicity of B[a]A by the oral route.

Results of tumor initiation/promotion studies indicate that benzo(a)anthracene is a complete carcinogen (ATSDR, 1989c).

Benzo(a)anthracene applied to the shaved backs of Swiss mice was reported to suppress sebaceous glands (Bock and Mund, 1958). However, controls were not employed; therefore, it is not possible to determine if the effects seen were due to the solvent and/or the application procedures.

Benz(a)anthracene and its 5 metabolically possible transdihydrodiols were tested for carcinogenicity in newborn Swiss-Webster mice (Wislocki et al., 1978) and for skin tumor-initiating activity in mice and mutagenicity in Chinese hamster V-79 cells (Slaga et al., 1978). In each case, the trans-3,4-dihydroxy-3,4-dihydrobenz(a)anthracene was the most active derivative compared to the parent substance or to any of the other possible derivatives. Also, the corresponding diol-epoxide, trans-3α, 4β-dihydoxy-1α, 2α-epoxy-1,2,3,4-tetrahydrobenz(a)anthracene was found to be a more effective tumor initiator than was the 3,4-dihydrodiolbenz(a)anthracene (Slaga et al., 1978). All of these results suppport the hypothesis that the bay-region diol-epoxide derivatives of benz(a)anthracenes are carcinogens. The 3,4-dihydrodiol derivative of 7,12-dimethylbenz(a)anthracene was a more potent initiator of skin tumors in mice than the parent substance, 7,12-dimethylbenz(a)anthracene (Slaga et al., 1979).

IARC (1983) has classified B[a]A as a Group 2A carcinogen. A 2A ranking indicates that there is limited evidence of carcinogenicity to humans. EPA has classified B[a]A as a B2 carcinogen (probable human carcinogen).

2.3 Human Toxicity

No studies regarding human toxicity were located in the literature.

3.0 BENZO[b]FLUORANTHENE

Benzo[b]fluoranthene (B[b]F) is a colorless solid at room temperature (IARC, 1983). B[b]F is not produced or used commercially; it occurs as a product of conbustion (IARC, 1983). Physical and chemical properties of B[b]F are listed in Table 3.

3.1 Genotoxicity

The genotoxicity of B[b]F has been evaluated in *in vitro* studies. Mutagenic activity was indicated in an investigation involving *Salmonella typhimurium* in the presence of an exogenous rat-liver extract (LaVoie et al., 1979). However, negative results were obtained from other similar studies (Hermann, 1981; Mossanda et al., 1979). The data in these studies are inadequate to support a positive or negative determination for B[b]F mutagenicity (ATSDR, 1988c).

3.2 Animal Toxicity

The metabolism of B[b]F has been investigated *in vitro* using hepatic S9 preparations (Amin et al., 1982). The general biotransformation pathways established for benzo[a]pyrene are also active on B[b]F (Cooper et al., 1983, Levin et al., 1982, Grover, 1986).

Many, but not all, 4, 5 and 6 ring PAH compounds exhibit carcinogenic activity, but only a few unsubstituted hydrocarbons with 7 rings or greater are tumorigenic or carcinogenic (Neff, 1979; U.S. EPA, 1980b; Dipple, 1985). The group of unsubstituted 5 and 6 ring PAH, which include B[b]F, are clearly the most potent of the series.

Benzo(b)fluoranthene has been shown to be carcinogenic to animals by the dermal route (ATSDR, 1989c; U.S. EPA, 1980b). Papillomas and carcinomas were observed by Wynder and Hoffman, (1959b) after the dermal application of B[b]F to mice. Habs et al. (1980) also reported that dermal application of B[b]F produced a significant carcinogenic response.

No studies on the carcinogenicity of B[b]F in animals following inhalation exposure were found in the available literature. However, B[b]F has been shown to cause respiratory tract tumors in rats following intratracheal instillation (Deutsch-Wenzel et al., 1983). In this experiment, B[b]F was prepared in solution with trioctanoin and molten beeswax and injected into the left lobe of the lungs of female Osborne-Mendel rats. The mixture congealed into a pellet from which the test compound diffused over time into the surrounding tissue. Doses of 0,0.1,0.3, or 1.0 mg B[b]F were administered, eliciting 0/35, 1/35, 3/35, or 13/35 lung tumor-bearing animals per group, respectively. Tumors were epidermoid carcinomas or pleomorphic sarcomas. This experiment indicates that B[b]F is a moderately active respiratory tract carcinogen.

IARC (1983) has classified B[b]F in Group 2A (limited evidence of carcinogenicity to humans). EPA has classified B[b]F as a group B2 carcinogen (probable human carcinogen).

3.3 Human Toxicity

Detectable levels of PAHs, including B[b]F, were reported in the lung and adjoining tissue of patients with bronchial carcinoma in concentrations ranging from 0.9 ng/g to 15,000 ng/g (Tomingas et al., 1976). No other studies regarding human toxicity of B[b]F were located in the literature.

4.0 BENZO[k]FLUORANTHENE

Benzo(k)fluoroanthene (B[k]F) is a pale yellow solid at room temperature. There is no production or known use of this compound. It occurs ubiquitously as a product of incomplete combustion (IARC, 1983). Physical and chemical properties of B[k]F are listed in Table 4.

4.1 Genotoxicity

Conflicting results have been reported for genotoxic effects of B[k]F (ATSDR, 1989c). Weyand et al., (1987) reported positive results for DNA binding in a mouse skin test system.

4.2 Animal Toxicity

Chronic dermal application of benzo(k)fluoranthene to Swiss mice resulted in no tumors, but skin papillomas were observed in 10% of animals treated with a higher concentration of B[k]F (Wynder and Hoffmann, 1959b). In another study, no significant increase in tumor incidence was observed in NMRI mice painted with up to 9.2 ug benzo(k)fluoranthene for a lifetime; no effect on mortality was noted (Habs et al., 1980).

A dose-related increase in tumor incidence was observed in mice receiveing 30-1000 µg B[k]F followed by TPA promotion (ATSDR, 1990). However, in the absence of a promoter, B[k]F did not induce tumors and is, therefore, not considered a complete carcinogen (IARC, 1983).

IARC (1983) has classified B[k]F in Group 2B, sufficient evidence of carcinogenicity in animals.

4.3 Human Toxicity

Autopsies performed on cancer-free patients found total PAH levels ranging from 11 to 2,700 ppt (parts per trillion; ng/kg) in fat samples. Several PAHs were detected, including B[k]F (Obana et al., 1981).

5.0 BENZO[a]PYRENE

Benzo[a]pyrene, B[a]P, is the most well studied of the several hundred chemically related compounds belonging to the general class of PAHs (IARC, 1983). Environmental sources of B[a]P are both natural and man made. B[a]P occurs ubiquitously in products of incomplete combustion. It is found in mainstream and sidestream cigarette smoke, in vehicle exhaust, and in some cooked foods. There is no commercial production or use of this compound (IARC, 1983). Physical and chemical properties of B[a]P are listed in Table 5.

5.1 Genotoxicity

There is sufficient evidence from short-term *in vivo* and *in vitro* genetic toxicology tests to demonstrate that B[a]P is a genotoxic agent when metabolically activiated. This evidence indicates that B[a]P interacts with mammalian gonads and germ cell DNA and induces such end points as unscheduled DNA synthesis (Sega, 1979), chromosomal aberrations (Basler and Rohrborn, 1978), and morphological abnormalities (Wyrobek et al., 1981). However, B[a]P is present as a component of the total content of PAHs in the environment. How interactions among various PAHs affect their potential for human genotoxicity is uncertain.

Positive mutagenic activity has been reported in the mouse spot test (Davidson and Dawson, 1977) and the somatic mutation and sex-linked recessive lethal mutation assays with *Drosophila melanogaster* (Fahmy and Fahmy, 1980; Nguyen et al., 1979; Vogel et al., 1983). However, negative results have been reported in similar studies with *Drosophila* (Valencia and Houtchens, 1981; Zijlstra and Vogel, 1984). Mixed results have been reported for aneuploidy studies with *Drosophila melanogaster* via feeding (Vogel et al., 1983; Valencia et al., 1984; Fabian and Matoltsy, 1946).

B[a]P requires metabolic activiation in order to exert its mutagenic and carcinogenic effects. The initial steps in the proposed mechanism of action of B[a]P-induced carcinogenesis involve metabolic formation of bay-region diol epoxides followed by covalent interaction of these reactive metabolites with DNA (Conney, 1982).

In some recent experiments, it has been determined that certain derivatives of B[a]P can be mutagenic without being metabolically activated (Pitts et al., 1978). Atmospheric particulate matter was mutagenic without further metabolic activation and it was suggested that PAHs in the atmosphere could react with gaseous agents to result in formation of directly-acting mutagenic substances. PAHs were exposed to nitrogen dioxide and nitric acid, ozone or peroxyacetyl nitrate. In each case, derivatives of B[a]P were formed that were mutagenic in the Ames assay without requiring metabolic activation.

EPA has concluded that B[a]P is an animal carcinogen and has classified it in Group B2, a probable human

carcinogen. IARC (1983) has classified B[a]P in Group 2B.

5.2 Animal Toxicity

The biological fate and mechanisms of absorption of inhaled B[a]P adsorbed on particles were studied in the rat (Sun et al., 1982). A [^{3}H]-benzo[a]pyrene radiolabeled concentration of 0.6 μg/L adsorbed on ultrafine Ga_2O_3 particles (diam ~0.1 μm/L) was administered to rats as an aerosol. A parallel study was conducted with a pure [^{3}H]-B[a]P aerosol (no carrier) at a concentration of 1 μg/L. Total exposure time for both groups was 30 minutes. The amount of aerosol particles deposited in the lung after termination of exposure was ~20% for Ga_2O_3 (corresponding to 3% [^{3}H]-B[a]P) and ~10% for the pure hydrocarbon aerosol. These values represent the percentage of the total inhaled mass that was deposited in the lungs. The excretion of hydrocarbon was monitored for over 2 weeks at which time a nearly quantitative recovery of radioalabel was obtained, indicating complete absorption of the initially deposited hydrocarbon. Consistent with administration of B[a]P by other routes, inhaled hydrocarbon was excreted predominantly in the feces (94% for B[a]P on Ga_2O_3 particles and 86% for the pure aerosol). Significant differences in the clearance times of Ga_2O_3 adsorbed and pure B[a]P strongly suggested that a substantial amount of B[a]P coated on Ga_2O_3 particles was cleared from the lungs by mucociliary clearance and subsequent ingestion. The pure B[a]P aerosol particles retained by the lungs were cleared by absorption into the blood stream. Particle association of B[a]P not only increased respiratory tract clearance, but also increased the effective dose of this compound as reflected by higher tissue concentrations relative to the pure aerosol exposure experiments. Similar observations have been reported by other workers (Creasia et al., 1976; Tornquist et al., 1985).

The gastrointesinal absorption of B[a]P was studied in the rat. ^{14}C-labeled B[a]P (0.04 μmol, 0.4 μmol and 4.0μmol), dissolved in peanut oil, was administered to rats by gavage (Hecht et al., 1979). Absorption of hydrocarbon was determined by measuring radioactivity in feces and urine. Total excretion of label in feces averaged 74% to 79% from 0 to 48 hours and 85% from 0 to 168 hours; excretion in urine was significantly less (1% to 3% of administered dose). The role of metabolism in the excretion of B[a]P was briefly explored. The amount of unchanged B[a]P excreted decreased as dose increased (13%, 7.8%, and 5.6% respectively) for the three doses studied.

The percutaneous absorption of ^{14}C-B[a]P was studied in adult Swiss Webster mice (Sanders et al., 1986). Absorption was measured by analyzing radioactivity in excreta (feces and urine) and by analysis of residual label at the site of application. Dissapearance of radiolabel from the application site was rapid: 6% (of an applied dose) in 1 hour and 40% in 24 hours. After 7 days, 93% of the radioactivity was recovered in excreta, mostly in the feces.

The skin penetration of an applied dose of [^{14}C] B[a]P (10 μg/cm^2) was determined in several mammalian

species under *in vitro* conditions (Kao et al., 1985). Dorsal skin from marmoset, guinea pig, rabbit, rat, and mouse were used for the permeation experiments. The mouse showed the highest permeation at 10% (24 hours), followed by the rat, rabbit, and marmoset (1% to 3%); the guinea pig exhibited the lowest permeation at 0.1%. The authors (Kao et al., 1985) suggested that first-pass cutaneous metabolism was an important factor in determining the extent of B[a]P penetration through the skin. They consider that, in addition to diffusion, metabolic pathways play a decisive role in the percutaneous absorption of B[a]P.

B[a]P which was orally administered to rats at a dosage of 4 μg/kg was distributed primarily to the protein fractions of the liver, lung and kidney, with concentrations gradually increasing with time (Yamazaki et al., 1987). In contrast, the lipid fractions of these tissues accounted for 70% of the administered dose at three hours, but subsequently decreased rapidly. The nucleic acid fraction maintained approximately 10% of the administered dose throughout the experiment. The authors concluded that protein binding of B[a]P in the lung and kidney may contribute to the cytotoxicity, mutagenicity and carcinogenicity of B[a]P and its metabolites, since these organs have low metabolic activity, while the liver has high detoxification potential and can expedite the excretion of these toxic products.

The metabolism of B[a]P is complex and includes the formation of a proposed carcinogen, B[a]P 7,8-diol-9,10-epoxide. The formation of other reactive metabolites of B[a]P generated under specific situations (i.e., free-radical intermediates) has also been demonstrated, although these pathways have not been shown to be relevant to the *in vivo* toxicity of B[a]P.

Metabolism of B[a]P is a prerequisite for hepatobiliary excretion and elimination through the feces, regardless of the route of administration. The rate-determining step in the biliary excretion of B[a]P administered intravenously has been shown to be metabolism and not biliary transport (Schlede et al., 1970). Because of the "first-pass" metabolism in the liver, orally administered B[a]P would be expected to show an enhanced rate of excretion relative to other administration routes.

B[a]P may be fatal to mice following ingestion, and death in animals has been reported following parenteral (non-oral) exposure to a number of PAH. Lethality and decreased longevity have been reported in "nonresponsive" strains of mice following subchronic oral exposure to 120 mg/kg body weight B[a]P and in "responsive" mice following a single intraperitoneal dose of 500 mg/kg body weight B[a]P (Robinson et al., 1975). No LD_{50} values have been reported for experimental animals exposed by the oral or dermal routes of exposure, nor have LC_{50} values been reported for experimental animals exposed to B[a]P by inhalation. The acute lethality of B[a]P has been investigated following intraperitoneal injection. The LD_{50} for B[a]P administered intraperitoneally to mice is 250 mg/kg body weight (Gerarde, 1960; Salamone, 1981).

Acute intragastric administration of 50 or 150 mg/kg/day B[a]P resulted in suppressed carboxylesterase activity in the intestinal mucosa (Nousianen et al., 1984). In the same study B[a]P was also a moderate inducer of hepatic carboxylesterase activity in rats intragastrically administered 50 mg/kg/day for 4 days. Enzyme alteration in the absence of other signs of gastrointestinal toxicity was not considered an adverse health effect, but may precede the onset of more serious effects. Given the selectivity of PAHs for rapidly proliferating tissues such as gastrointestinal mucosa, oral exposure to PAHs at higher doses could lead to adverse gastrointestinal effects (ATSDR, 1989c).

The results of two oral studies in mice (Mackenzie and Angevine, 1981; Rigdon and Neal, 1965) and one in rats (Rigdon and Rennels, 1964) indicate that B[a]P induces reproductive toxicity in animals. The incidence and severity of these effects depends on the strain, method of administration and dose levels used. In a two-generation study, B[a]P administered by gavage to pregnant CD-1 mice decreased the percentage of pregnant females at parturition and produced a high incidence of sterility in the progeny (Mackenzie and Angevine, 1981). In contrast, benzo(a)pyrene administered in the diet caused no adverse effects of pregnancy in female rates (Rigdon and Neal, 1965), but reduced the incidence of pregnancy in female rats (Rigdon and Rennels, 1964). Based on these studies, the LOAEL for B[a]P-induced reproductive toxicity in parental mice was 160 mg/kg/day and the LOAEL for these effects in the progeny of exposed animals was 10 mg/kg/day (Mackenzie and Angevine, 1981). No NOAEL was identified.

Three animal studies were reviewed that evaluated the developmental effects of B[a]P on inbred strains of rats and mice. The data from these studies indicate that prenatal exposure to B[a]P produced reduced mean pup weight during postnatal development and a high incidence of sterility in the F1 progeny of mice (Mackenzie and Angevine, 1981). Using aromatic hydrocarbon (Ah)-responsive and non-responsive strains of mice, the increased incidence of stillborns, resorptions and malformations observed were directly related to the maternal and/or embryonal genotype (Legraverend et al., 1984). In rats, effects were reported following B[a]P treatment during gestation (Sheveleva, 1978).

There are reports of immunotoxicity of PAHs following dermal, intraperitoneal and subcutaneous injection in animals. The carcinogenic PAHs as a group have an immunosuppressive effect; in general, the degree of immunosuppression is correlated with carcinogenic potency (ATSDR, 1989c). B[a]P markedly inhibits the immune system in mice, especially T-cell dependent antibody production by lymphocytes exposed either *in vivo* or *in vitro* (Blanton et al., 1986, Lyte and Bick, 1985). B[a]P exerts an inhibitory effect on antibody production through alterations in the normal functioning of macrophages, T-cells and B-cells (Blanton et al., 1988). In contrast B[a]P has no effect on most cellular immune responses prior to the appearance of tumors (Dean et al., 1983), although B[a]P exposure does inhibit interleukin-2 dependent proliferation (Myers et al., 1988).

Immunotoxicity of B[a]P following intraperitoneal and subcutaneous injection has been studied. B[a]P-induced immune suppression was reported in male B6CF1 mice (Lyte and Bick, 1985) and in the offspring of C3H/Anf mice treated intraperitoneally with B[a]P (Urso and Gengozian, 1980). Subcutaneous injections of B[a]P in female B6C3F1 mice produced a dose-related suppression of antibody production to both T-cell-independent and T-cell-dependent antigens (White and Holsapple, 1984). Reports concerning the immunotoxicity of B[a]P following inhalation, oral, or dermal exposure could not be found in the available literature.

Benzo(a)pyrene is active as a tumor initiator using initiation/promotion protocols. Topical application of a single initiation dose of B[a]P to the backs of mice followed by promotion with TPA or croton oil resulted in an enhanced incidence (80-92%) of skin papillomas (Cavalieri et al. 1988b, see Table 2-3; Hoffmann and Wynder 1966). Ten doses of B[a]P (0.1 mg/dose) topically applied to the backs of Swiss mice followed by promotion with croton oil (for 20 weeks) resulted in the development of skin tumors (Hoffmann et al. 1972).

The induction of preneoplastic hepatocytes, known as GGT foci, in animals has been correlated with cancer promotion. A one day intragastric administration of 200 mg/kg of B[a]P to partially hepatectomized rats followed by a diet containing 2-acetylaminofluorene and carbon tetrachloride induced GGT foci (Tsuda and Farber, 1980).

Toxic effects of B[a]P in animals depends greatly on the inducibility of the enzyme aryl hydrocarbon hydroxylase, or the genetic constitution of the species (activity of Ah locus). Oral administration of 120 mg/kg body weight B[a]P per day in the diet produced aplastic anemia and death within four weeks in poorly inducible mouse strains, whereas the poorly inducible group developed bone marrow cell irregularities. However, highly inducible mouse strains (those which experienced enzyme activation) remained health for at least six months (IARC, 1983). The difference in toxic response is a result of a more efficient detoxification mechanism in highly inducible mice than in less inducible mice.

Rats intragastrically administered 100 mg/kg/day of B[a]P for 4 days exhibited cytosolic ADH induction (Torronen et al., 1981). Exposure to B[a]P also increased the relative liver weights by 27% (Torronen et al., 1981). However, intragastric administration of 51.4 mg/kg/day B[a]P to partially hepatectomized rats had no effect on the extent of liver regeneration (Gerschbein, 1975).

B[a]P is a moderately potent experimental skin carcinogen, and it is often used as positive control in bioassays of other agents. B[a]P was first reported to induce skin tumors in mice in 1933 (Cook et al., 1933; Cook, 1933), although mixtures of PAHs that include B[a]P such as coal tar were shown to be dermal carcinogens in animals as early as 1918 (Yamagiwa and Ichikawa, 1918). B[a]P is active both as a

"complete" carcinogen and as initiator using initiation/promotion protocols. In its role as a positive control, B[a]P is usually administered at a single dose level, so that quantitative evaluation of dose-response relationships is not possible.

Subchronic (19-20 weeks) topical application of a B[a]P solution to the backs of mice resulted in a dose-related development of skin papillomas and squamous cell carcinomas (Cavalieri et al. 1988b; Shubik and Porta 1957). In mice, the tumorigenic dose of B[a]P is influenced by the solvent used for delivery. Graded concentrations of B[a]P dissolved in decalin or a solution of n-dodecane and decalin were topically administered to the backs of mice for 50 weeks (Bingham and Falk, 1969). Use of the n-dodecane and decalin solvent mixture significantly enhanced the potency of B[a]P at lower doses in comparison with decalin alone. Malignant tumors appeared in 21% of the animals at 0.00002% (0.0054 mg/kg/day) B[a]P in dodecane and decalin solvent. In contrast, a 42% skin tumor incidence was not observed until 0.02% (4.8 mg/kg/day) of B[a]P in decalin alone was applied. The method of application was not specified, sample sizes were small and no decalin solvent controls were included; however decalin is not considered to be carcinogenic. In this same study, subchronic (50 week) dermal application of B[a]P dissolved in the co-carcinogens 1-dodecanol or 1-phenyldodecane produced skin tumors in animals exposed to 0.05% B[a]P in either solvent. The tumor incidence varied depending on the solvent concentration; however, the latency period was reduced only when 1-dodecanol was the solvent (Bingham and Falk, 1969).

Mammary tumors have also been observed following intermediate duration oral exposure to B[a]P in rats. Eight weekly oral doses of 6.25 mg B[a]P (12.5 mg/kg) administered to rats resulted in a 67% increase in the incidence of mammary tumors in female rats after 90 weeks of observation (McCormick, 1981). A 30% incidence in these tumors was observed in the control animals.

Studies in experimental animals have demonstrated the ability of B[a]P to induce skin tumors following long-term dermal exposure. Mice receiving doses of 1.7 µg/day and above applied to their skin developed an excess of skin tumors following long-term exposure (Habs et al., 1980).

5.3 Human Toxicity

Dermal absorption of B[a]P through human skin (leg skin) was determined under *in vitro* conditions (Kao et al., 1985). The extent of permeation after 24 hours was established as 3% of an applied dose of [^{14}C] benzo[a]pyrene (10 µg/cm^2).

Autopsies performed on cancer-free patients found total PAH levels ranging from 11 to 2,700 ppt (parts per trillion; ng/kg) in fat samples. Several PAHs were detected, including B[a]P (Obana et al., 1981).

Detectable levels of PAHs were reported in the lung and adjoining tissue of patients with bronchial

carcinoma in concentrations ranging from 0.9 ng/g to 15,000 ng/g (Tomingas et al., 1976). Of the PAHs detected, B[a]P was found in the highest concentrations in samples taken from carcinomas.

No studies have been able to conclusively demonstrate carcinogenicity of B[a]P in humans. Epidemiologic studies, however, have shown an increased mortality due to lung cancer from inhalation exposure to coke oven emissions, roofing-tar emissions, and cigarette smoke. Skin cancer has also been reported among workers exposed dermally to shale oils; scrotal cancer has been reported among chimney sweeps (ATSDR, 1989a). These mixtures contain many potentially carcinogenic PAHs including tumor promoters, initiators, and cocarcinogens such as coal tar pitch and creosote. B[a]P is known to be present in these mixtures, but due to the complexity of the mixture and presence of other carcinogens, the percent contribution of B[a]P ot the toxic effects observed has not been quantified.

EPA has concluded that B[a]P is an animal carcinogen and has classified it in Group B2, a probable human carcinogen. IARC (1983) has classified B[a]P in Group 2B.

6.0 CHRYSENE

Chrysene is formed from the incomplete combustion of fossil fuels or other organic matter. It is a component of coal tar pitch which is used in industry as a binder for electrodes, and of creosote which is used to preserve wood (ATSDR, 1988b). Physical and chemical properties of chrysene are listed in Table 6.

6.1 Genotoxicity

The genotoxicity of chrysene has been extensively studied. As with other PAH compounds, genotoxic action is dependent upon metabolic activation, either exogenously supplied or endogenously present (ATSDR, 1988b).

The 1,2-dihydrodiol, a metabolic product of chrysene, is active as a tumor initiator on mouse skin. The 1,2-diol and 1,2 diol-3,4-epoxide are mutagenic in bacterial and mammalian cells (IARC, 1983). Additionally, the 1,2-diol-3,4-epoxide has been shown to form DNA adducts in hamster cells treated with chrysene (IARC, 1983).

In the presence of an exogenous metabolic system, chrysene was mutagenic to *Salmonella typhimurium*. In one study of mice and hamsters, chrysene induced sister chromatid exchange and chromosomal aberrations. Several other studies, however, have produced conflicting results regarding mutagenicity in mammalian cells (ATSDR, 1989c).

6.2 Animal Toxicity

The LD_{50} for mice administered chrysene intraperitoneally was found to be >320 mg/kg body weight (ATSDR, 1988b).

In a study by Modica et al. (1982), intestinal absorption of chrysene was not quantified, but its extent of absorption in rats was dependent on the oral dose and the vehicle of administration. Approximately 25-41% of the chrysene dose (22.8 mg/kg in olive oil) was recovered in the feces within 72 hours after administration (Modica et al., 1982). Chang (1943) reported an excretion of 79% of the chrysene dose in the feces following dietary (500 mg/kg) and gavage (200 mg/kg) administration.

Metabolism of chrysene at relatively high rates in mouse skin provides evidence of its dermal uptake (Hodgson, 1983; Weston et al., 1985).

Orally administered chrysene is distributed rapidly and widely in the rat (Bartosek et al., 1984). Maximum concentrations in well-perfused tissues, like the liver, blood and brain, were achieved within 1-2 hours after administration. Maximum levels in lesser well-perfused tissues, like adipose and mammary tissue, were reached in 3-4 hours.

The extent of liver regeneration, which is indicative of the ability to induce a proliferative response, has been examined following acute oral exposure to various PAHs. Partially hepatectomized rats fed a diet containing 514 mg/kg/day chrysene exhibited equivocal results; in one trial a significant increase in liver regeneration was noted, while in another trial no increase in liver regeneration on liver-to-body weight ratio was observed (Gershbein, 1975).

Initiating doses of chrysene followed by promotion with TPA or croton resin induced a dose-related papilloma incidence in mice (Levin et al., 1978; Slaga et al., 1980; Van Duuren et al., 1966; Wood et al., 1979). Ten daily treatments of chrysene to Swiss mice followed by TPA promotion (for 20 weeks), resulted in an enhanced incidence of papillomas and carcinomas (61%) compared to the control group (Hecht et al., 1974; Scribner, 1973).

Chrysene has been classified by EPA in Group B2, a probable human carcinogen based on animal studies (HEAST, 1990).

6.3 Human Toxicity

No studies regarding the toxicity of chrysene to humans were located in the literature.

7.0 DIBENZ[a,h]ANTHRACENE

Dibenz[a,h]anthracene (DB[a,h]A) is not produced or used commercially. It is a colorless solid at room temperature and has a melting point of 269-270°C. It is formed as a result of incomplete combustion, and it found in cigarette smoke and coal tar. Physical and chemical properties of DB[a,h]A are listed in Table 7.

7.1 Genotoxicity

The genotoxicity of DB[a,h]A has been demonstrated in various *in vitro* genetic assays. DB[a,h]A was positive in *in vitro* differential survival assays measuring DNA damage that used DNA-repair-proficient/deficient strains of bacteria. Unscheduled DNA synthesis was observed in both human epithelial and HeLa cell cultures, but none were noted in rodent cell cultures. DB[a,h]A was mutagenic in *Salmonella typhimurium* and cultured mammalian cells in the presence of an exogenous metabolic activation system. Generally, it was positive in rodent cell transformation assays. Only one study showed that it was mutagenic to a human epithelial-like cell line (ATSDR, 1987c).

There is sufficient evidence, predominantly from *in vitro* assays, to indicate that DB[a,h]A is a genotoxic agent when metabolically activated to the dihydrodiol and oxide forms. However, the absence of information on the ability of DB[a,h]A to reach and interact with mammalian germ cells makes it difficult to state whether this chemical is genotoxic in humans (ATSDR, 1987e).

7.2 Animal Toxicity

Administration of DB[a,h]A in the diet (250 mg) or by stomach tube (200 mg) resulted in greater than 90% of the dose being excreted in the feces of white rats (Chang, 1943); absorption was not quantified.

Sanders et al. (1986) applied DB[a,h]A (5.4, 56, 515 $\mu g/cm^2$) to the shaved backs of mice. The presence of PAHs in the skin at the application site, in excreta, and in exhaled air were monitored. At 24 hours after the maximum dose of DB[a,h]A was applied, 67.2% of the dose was recovered from the application site, 25.4% from body tissues, and 7.4% from excreta. The amount absorbed did not increase linearly with the dose due to an apparent saturation of the uptake process.

DB[a,h]A and several other PAHs, orally administered to rats, were widely distributed to several tissues (Daniel et al., 1967). Maximum tissue concentrations were not reached until 10 hours after administration, with highest tissue concentrations were in the liver and kidneys, followed by adrenal glands, ovaries, blood, and fat. Soon after administration, large quantities of DB[a,h]A were found in the liver and kidneys. The elimination rate from these organs was rapid. At 3-4 days after administration, the PAH compounds were

distributed only in the adrenal glands, mesenteric lymph nodes, ovaries and fat, where they were detected for several months after treatment. Generally all of the administered PAHs behaved similarly (Daniel et al., 1967). Thus, following oral intubation, these PAHs were absorbed into the lymph, distributed via the blood, and concentrated in liver and kidney from which they were excreted via bile and urine; Small amounts were retained in only a few tissues for long periods of time. Since tumors in rats are induced following single doses of orally administered PAHs, the amount of PAHs absorbed from the intestinal tract may be sufficient to exert its biological effects.

DB[a,h]A undergoes metabolic transformations in animals to reactive intermediates responsible for its toxicity. No information on biotransformation of BD[a,h]A in humans is available. In animals, DB[a,h]A is metabolized to a bay-region 3,4-dihydrodiol-1,2-epoxide derivative which is thought to be responsible for its genotoxic and carcinogenic activity.

Dibenzo(a,h)anthracene is a symmetrical hydrocarbon and possesses two bay regions. Testing of the bay-region 3,4-dihydrodiol derivative along with other dihydrodiol derivatives for tumor-initiating activity on mouse skin and for tumorigenicity in newborn mice led to the conclusion that the bay-region diol-epoxide derivative of this symmetrical polycyclic aromatic hydrocarbon is carcinogenic (Buening et al., 1979). In view of all the data presented, the theory that the carcinogenic activity of most, if not all, polycyclic aromatic hydrocarbons are due to the metabolically-derived bay-region diol-epoxide derivatives appears very sound. Also supportive of this theory is the finding that K-region epoxides are less tumorigenic than the parent compound (Grover et al., 1975).

DB[a,h]A injected subcutaneously weekly for 40 weeks and pyrene incorporated in the diet were associated with pale, soft and enlarged livers that showed evidence of fatty degeneration and iron deposition (Hoch-Ligeti, 1941; White and White, 1939).

Acute topical application of various PAHs has been reported to suppress or destroy sebaceous glands in mouse skin (Bock and Mund, 1958). DB[a,h]A applied to the shaved backs of Swiss mice was reported to suppress sebaceous glands (Bock and Mund, 1958). However, controls were not employed; therefore, it is not possible to determine if the effects seen were due to the solvent and/or the application procedures.

The immunosuppressive effects of DB[a,h]A were studied in both AHH-inducible mice (C57B1/6) and AHH-noninducible mice (DB[a,h]A/2N) by intraperitoneal and oral administration (Lubet et al., 1984). Immunosuppression occurred in both strains following intraperitoneal administration and was more pronounced in the C57B1/6 mice than in the DB[a,h]A/2N mice. However, the DB[a,h]A/2N mice were more susceptible to immunosuppression following oral administration. These results suggest that PAHs are rapidly metabolized and excreted following oral administration in AHH-inducible mice, whereas in

noninducible mice the PAHs are absorbed and distributed to target organs. Based on these results, the authors concluded that AHH inducibility plays an important role in the immunosuppressive activity of PAHs.

DB[a,h]A at a daily dose of 5 mg given subcutaneously from the first day of pregnancy, resulted in fetal death and resorption, and may also have affected the subsequent fertility of the dams (Wolfe and Bryan, 1939).

DB[a,h]A has demonstrated tumor initiation activity using a standard initiation/promotion protocal (Slaga et al., 1980). DB[a,h]A has been reported to initiate skin development in a dose-response relationship at doses as low as 0.02 µg administered once (Klein, 1960) or 0.028 µg followed by promotion with TPA (for 25 weeks) (Buening et al., 1979).

Many, but not all, 4, 5 and 6 ring PAH compounds exhibit carcinogenic activity, but only a few unsubstituted hydrocarbons with 7 rings or greater are tumorigenic or carcinogenic (Neff, 1979; U.S. EPA, 1980b; Dipple, 1985). The unsubstituted 5 and 6 ring PAHs, which include DB[a,h]A, are the most potent of the series.

The induction of preneoplastic hepatocytes, known as GGT foci, in animals has been correlated with cancer promotion. A one day intragastric administration of 200 mg/kg of DB[a,h]A to partially hepatectomized rats followed by a diet containing 2-acetylaminofluorene and carbon tetrachloride induced GGT foci (Tsuda and Farber, 1980).

The extent of liver regeneration, which is indicative of the ability to induce a proliferative response, has been examined following acute oral exposure to various PAHs. Partially hepatectomized rats were fed diets containing various PAH for 10 days. Diets containing 51.4 mg/kg/day DB[a,h]A produced no increase in the liver-to-body weight ratio (Gershbein, 1975).

Several subchronic studies that investigated the carcinogenicity of DB[a,h]A in animals following oral exposure via the diet or drinking water were located. Mammary carcinoma was observed in 5% of the female BALB/c mice dosed with 0.5% DB[a,h]A after 15 weeks, however, no control group was included (Biancifiori and Caschera, 1962). Mice (strain unspecified) receiving DB[a,h]A in the diet for five to seven months developed forestomach tumors in 32% of the animals surviving at one year (Larinow and Soboleva, 1938). None of these studies was adequately reported; they did not perform appropriate histopathologic evaluations, treatment or study durations and sample size were inadequate.

Forestomach papillomas were found in mice after a single oral dose of DB[a,h]A (Berenblum and Haran, 1955). In other studies, mice that received DB[a,h]A emulsions developed lung adenomas and papillomas and squamous cell carcinomas of the forestomach (Lorenz and Steward, 1948; Snell and Stewart, 1962;

Snell and Stewart, 1963).

There is experimental evidence that DB[a,h]A can cause tumors in mice following oral administration, lung tumors in rats and hamsters following intratracheal instillation, and skin cancer following dermal application (Kennaway and Heiger, 1930). DB[a,h]A was the first chemically pure substance shown to induce cancer.

As part of a study of the carcinogenicity of tobacco and its constituents Wynder and Hoffman, (1959) several PAHs, including DB[a,h]A, were tested as carcinogens on mouse skin. Groups of 20 female Swiss mice received concentrations of 0.001, 0.01, or 0.1% DB[a,h]A dissolved in acetone three times a week throughout their lifetimes. No solvent control groups were reported; however, since no papillomas or carcinomas were obtained for several of the PAHs tested, a solvent control group would most likely have been negative as well. Incidences of papillomas and carcinomas at the site of application were dose related at the two lowest doses. The decrease in tumor rate at the highest dose tested probably reflects DB[a,h]A's toxicity and the resulting decreased survival observed. Reductions in tumor latency period were also found to be dose related. The lowest concentration at which DB[a,h]A elicited tumors was 0.001%, which is approximately equal to a dose of 2.9×10^{-2} mg/kg (1.2×10^{-2} mg/kg/day).

DB[a,h]A is classified as a probable carcinogen by EPA (Group B2), and in Group 2B by IARC (1983).

7.3 Human Toxicity

No studies regarding the toxicity of DB[a,h]A were located.

8.0 INDENO[1,2,3-cd]PYRENE

Indeno[1,2,3-cd]pyrene (IndP) exists as a yellow to greenish yellow solid at room temperature. It occurs with other PAHs in the environment from anthropogenic sources and from incomplete combustion. Physical and chemical properties of IndP are listed in Table 10.

8.1 Genotoxicity

IndP tested positive for gene mutation in activated test systems with bacteria (*S. tymphinurium*) (ATSDR, 1989c).

8.2 Animal Toxicity

Results of tumor initiation/promotion studies indicate that IndP is carcinogenic in rats and mice following dermal exposure (ATSDR, 1989c). However, IndP is not a complete carcinogen.

Chronic dermal application of IndP in dioxane to mice did not produce an increased incidence of skin tumors. However, when acetone was used as the solvent, a dose-related increase in tumor incidence was observed after 9 months (Hoffman and Wynder, 1966). Indeno(1,2,3-cd)pyrene was observed to have tumor initiating activity at repeated doses of 250 µg (10 applications), followed by promotion with croton oil (Hoffmann and Wynder, 1966). A pronounced dose-response relationship has been exhibited in an initiation-promotion bioassay when TPA was employed as the promoting agent (Rice et al., 1985). Chronic topical application of up to 9.2 µg of IndP in acetone to the backs of mice for a lifetime resulted in no tumor induction (Habs et al., 1980). Thus, the expression of IndP-induced carcinogenicity appears to vary with the solvent employed for delivery.

IndP has been classified by EPA in Group B2, a probable human carcinogen, based on sufficient evidence from animal studies (EPA, 1991).

8.3 Human Toxicity

No studies regarding human toxicity of IndP were located in the literature.

9.0 ANTHRACENE

Anthracene is present in coal tar, gasoline, and cigarette smoke. Anthracene was also commercially produced in the U.S. until 1983 for use in dyes (IARC, 1983). Physical and chemical properties of anthracene are listed in Table 1.

9.1 Genotoxicity

The majority of mutagenicity test results for anthracene are negative, although positive results have been reported in at least one *in vitro* test. Anthracene was mutagenic in *Salmonella typhimurium*, and positive results were obtained in several *in vitro* mammalian cell systems (ATSDR, 1989c).

Anthracene is generally considered inactive as a tumor initiating agent (ATSDR, 1990).

9.2 Animal Toxicity

The percutaneous absorption of anthracene in rats (9.3 ug/cm^2) resulted in approximatley 52% of the dose being absorbed in a dose-dependent manner. Diffusion of anthracene through the skin (stratum corneum) depended on the amount of anthracene on the skin surface (Yang et al., 1986).

Acute intragastric administration of 100 mg/kg/day of anthracene to rats resulted in a 13% increase in carboxylesterase activity of the intestinal mucosa (Nousiainen et al., 1984). Enzyme alteration in the absence of other signs of gastrointestinal toxicity was not considered an adverse health effect, but may precede the onset of more serious effects. Given the selectivity of PAHs for rapidly proliferating tissues such as gastrointestinal mucosa, oral exposure to PAHs at higher doses could lead to adverse gastrointestinal effects (ATSDR, 1989c).

The ability to induce aldehyde dehydrogenase (ADH) in animals has been correlated with carcinogenic potential. Rats intragastrically administered 100 mg/kg/day of anthracene for four days exhibited cytosolic ADH induction (Torronen et al., 1981). However, the authors concluded that anthracene is a poor ADH inducer (Torronen et al., 1981).

Rats intragastrically administered 100 mg/kg/day anthracene for 4 days did not exhibit induction of hepatic carboxylesterase activity (Nousianen et al., 1984). A single injection of anthracene had no adverse effect on the kidneys of mice (Shubik and Porta, 1957).

The extent of liver regeneration, which is indicative of the ability to induce a proliferative response, also has been examined following acute oral exposure to various PAHs. Partially hepatectomized rats were fed diets containing various PAHs for 10 days. Administration of 514 mg/kg/day anthracene had no

effect on the extent of liver regeneration. Diets containing 180 mg/kg/day anthracene produced no increase in the liver-to-body weight ratio (Gershbein, 1975).

Lymphoid effects have been observed in mice following subchronic weekly subcutaneous injections of anthracene for 40 weeks (Hoch-Ligeti, 1941). This treatment resulted in gross changes in the lymphoid system including an increase in stem cells, an accumulation of iron, reduced lymphoid cells and dilated lymph sinuses. Spleen weight in treated mice was significantly lower than that observed in controls (Hoch-Ligeti, 1941).

Chronic oral administration of a total dose of 4.5 gram anthracene in the diet to BD1 or B111 rats for 78 weeks did not produce tumors (Druckrey and Schmahl, 1955). Thus, the results of this single study suggest that anthracene is noncarcinogenic in animals following chronic oral exposure.

Anthracene was tested for carcinogenicity in mice by dermal application and in the mouse skin initiation-promotion assay. The results did not demonstrate a carcinogenic effect or a cancer initiating activity (IARC, 1983). Anthracene was tested for carcinogenicity in rats by oral, subcutaneous, intraperitoneal, and intrapulmonary administration, and in rabbits by implantation into the brain or eyes (IARC, 1983). These studies, either produced no evidence of carcinogenicity or were inadequate for evaluating carcinogenic properties.

The intraperitoneal LD_{50} for the mouse is greater than 430 mg/kg body weight (Salamone, 1981). The ID_{50} or skin irritant activity, for the mouse is 6.6×10^{-4} mm/ear (IARC, 1983).

EPA has placed anthracene in Group D, not classified as to carcinogenicity.

9.3 Human Toxicity

Autopsies performed on cancer-free patients found total PAH levels ranging from 11 to 2,700 ppt (parts per trillion; ng/kg) in fat samples. Several PAHs were detected, including anthracene (Obana et al., 1981).

PAHs may be absorbed through the skin of humans. Application of 2% crude coal tar to the skin of humans for eight hour periods on two consecutive days yielded evidence of PAH absorption (Storer et al., 1984). Anthracene was detected in the blood, but absorption of PAHs in crude coal tar was variable and dependent on the chemical species.

Anthracene has been associated with gastrointestinal toxicity in humans. Humans who consumed laxatives containing anthracene for prolonged periods (anthracene concentration not specified) were found to have

an increased incidence (73.4%) of melanosis of the colon and rectum in comparison to patients (26.6%) who did not consume the anthracene-containing laxatives (Badiali et al., 1985). Given the selectivity of PAHs for rapidly proliferating tissues, such as gastrointestinal mucosa, oral exposure to PAHs by humans may result in adverse gastrointestinal effects (ATSDR, 1989c).

10.0 FLUORANTHENE

Fluoranthene is a pale yellow solid at room temperature. Fluoranthene is present in crude oil, coal tar, gasoline, and cigarette smoke (IARC, 1983). Physical and chemical properties of fluoranthene are listed in Table 8.

10.1 Genotoxicity

Conflicting results have been reported for genotoxic effects of fluoranthene (ATSDR, 1989c). Fluoranthene is among the PAHs generally considered inactive as tumor initiating agents (ATSDR, 1989c).

10.2 Animal Toxicity

The oral LD_{50} for the rat is 2,000 mg/kg; the dermal LD_{50} for rabbits is 3,180 mg/kg (IARC, 1983). When added at a concentration of 1 µmol/ml in dimethyl sulfoxide to mouse ascites sarcoma cells in culture, the growth rate was inhibited 38% (IARC, 1983). Following incubation of fluoranthene with a rat-liver preparation, the 2,3-dihydrodiol metabolite was detected and tested for mutagenicity. It was found to be mutagenic in *Salmonella typhimurium* in the presence of an exogenous metabolic system and positive for mutagenicity *in vitro* in human lymphoblastoid cells (IARC, 1983). In two carcinogenicity tests by skin application to mice, fluoranthene did not produce a tumorigenic response. However, fluoranthene administered to mice by skin application together with B[a]P yielded twice as many tumors as the control group which was administered B(a)P alone (IARC,1983). These data suggest that fluoranthene is an incomplete carcinogen, but which may be capable of synergistic effects (e.g., tumor development) when combined with B(a)P.

IARC (1983) has classified fluoranthene in Group 3; not classifiable as to its carcinogenicity to humans. EPA has placed fluoranthene in Group D (EPA, 1991).

10.3 Human Toxicity

PAHs are absorbed through the skin of humans. Application of 2% crude coal tar to the skin of humans for eight hour periods on two consecutive days yielded evidence of PAH dermal absorption (Storer et al., 1984). Fluoranthene was detected in the blood, but dermal absorption of PAHs from crude coal tar was variable and dependent on the chemical species.

Genotoxic effects in human cells have been reported for fluoranthene. This PAH was reported to be mutagenic in human lymphoblasts in an *in vitro* test system with an exogenous metabolic activation system (Barfknecht et al., 1982); however, negative results were obtained in a second test without metabolic activation (Rocchi et al., 1980; Crespi et al., 1985).

Detectable levels of PAHs, which include fluoranthene, were reported in the lung and adjoining tissue of patients with bronchial carcinoma in concentrations ranging from 0.9 ng/g to 15,000 ng/g (Tomingas et al., 1976).

11.0 FLUORENE

Fluorene occurs in the environment as a result of incomplete combustion; it is also found in fossil fuels. It has been detected in coal tar (up to 1.6%), in mainstream cigarette smoke, and in vehicle exhaust (IARC, 1983). Physical and chemical properties of fluorene are listed in Table 9.

11.1 Genotoxicity

Fluorene tested negative for gene mutation in the Ames assay, with and without activation (ATSDR, 1989c). Results of *in vitro* tests in mammalian cells for DNA damage (unscheduled systhesis) were also negative (IARC, 1983).

11.2 Animal Toxicity

A single injection of fluorene had no adverse effect on the kidneys of mice (Shubik and Porta, 1957).

Fluorene is among the PAHs generally considered inactive as tumor initiating agents (ATSDR, 1989c).

The extent of liver regeneration, which is indicative of the ability to induce a proliferative response, has been examined following acute oral exposure to various PAHs. Partially hepatectomized rats were fed diets containing various PAHs for 10 days. Administration of 180 mg/kg/day of fluorene resulted in a statistically significant increase in the extent of liver regeneration (Gershbein, 1975).

Subchronic dietary administration of fluorene to rats for six months at a concentration approximately equivalent to 8.6 mg/kg/day produced increased incidences of squamous cell carcinoma of the kidney and uterus (9% and 9%, respectively). However, control animals exhibited a 5% increase in the incidence of kidney adenoma, 11% increase in the incidence of pituitary adenoma, and 5% increase in the incidence of granulocytic leukemia (Morris et al., 1960). The presence of tumors in control animals render these results difficult to interpret.

EPA has placed fluorene in Group D, not classified as to carcinogencity (EPA, 1991).

11.3 Human Toxicity

No studies regarding toxicity of fluorene in humans were located in the literature.

12.0 2-METHYLNAPHTHALENE

2-Methylnaphthalene is a PAH which is structurally similar to naphthalene, and has been identified in the waste water of coking operations and textile processing plants. It is often used as a component in slow-release insecticides, mole repellents, and in combination with the production of naphthalene (Clayton &

Clayton, 1981). Physical and chemical properties of 2-methylnaphthalene are listed Table 11.

12.1 Genotoxicity

No studies were located regarding the genotoxicity of 2-methylnaphthalene.

12.2 Animal Toxicity

Few toxicological and chemical data are available for this compound. In a study by Griffin et al. (1981), mice were administered a single intraperitoneal dose of 1,000 mg/kg 2-methylnaphthalene which resulted in 20-40% mortality. In this same study, a single intraperitoneal injection of 100 mg/kg produced slight exfoliation of the bronchial epithelium and 400 mg/kg resulted in marked to complete exfoliation of bronchial epithelium (ATSDR, 1989b). These results suggest that respiratory effects may be of concern.

When either naphthalene or 2-Methylnaphthalene was applied dermally in combination with B[a]P, there was an inhibitory effect on the induction of skin tumors in female mice (Schmeltz et al., 1978). These investigators also reported that a mixture containing naphthalene (0.02%), 2-methylnaphthalene (0.02%) and 10 other methylated and ethylated naphthalenes (each at 0.02%) also appeared to inhibit the development of B[a]P-induced skin tumors. The authors suggested that it is likely that certain naphthalenes compete with B[a]P for the same enzyme sites, resulting in alteration of the B[a]P metabolic pathway and decreased production of the active B[a]P metabolite. Dermal application of the naphthalene mixture did not induce tumors in the absence of B[a]P. The results of these studies were not analyzed statistically.

2-methylnaphthalene has not been evaluated by EPA for carcinogenicity.

12.3 Human Toxicity

Very little information is available on human health effects of 2-methlnaphthalene. Clinical effects are based on ingestion and inhalation exposure to mothballs. Effects include headache, restlessness, lethargy, convulsions, coma, nausea, vomiting, hepatocellular injury and hemoglobinuria (IRIS, 1989). No exposure concentration or duration of exposure associated with these effects was reported. 2-Methylnaphthalene is not a skin irritant or photosensitizer (Clayton & Clayton, 1981).

13.0 NAPHTHALENE

Naphthalene is a white solid with the odor of mothballs. It is derived from petroleum cracking, coke oven emissions, and the carbonization of bituminous coal (Clayton & Clayton, 1981). It is flammable in both solid and liquid form. Naphthalene is used extensively as a raw material and as an intermediate in the chemical, plastics, and dye industries. It is also used as an intermediate in the production of insecticides, fungicides, lacquers, varnishes, and as a moth repellant. As a medicinal agent, it has been applied as an antiseptic, anthelminthic, and dusting powder in skin diseases (Clayton & Clayton, 1981). Physical and chemical properties of naphthalene are listed in Table 12.

13.1 Genotoxicity

Naphthalene has tested negative in *in vitro* studies, including several strains of bacteria and in various mammalian test systems (ATSDR, 1989b).

13.2 Animal Toxicity

In rats administered radio-labeled naphthalene, the amount of label recovered in 24 hours was 77 to 93% in urine and 6 to 7% in feces (Bakke, 1985). In rats, Summer et al.(1979) found a dose-dependent increase in urinary mercapturic acid excretion following gavage doses of naphthalene at 30, 75 and 200 mg/kg, corresponding to the elimination of approximately 39, 32, and 26% of each dose, respectively, within 24 hours.

The metabolism of naphthalene is complex. While there are a few reports which have clearly demonstrated the presence of a variety of metabolites following the oral administration of naphthalene to various animal species, much of the information regarding naphthalene metabolism has come from studies using intraperitoneal administration and *in vitro* assays. Key metabolites in humans and other species are 2-naphthoquinones, which have been shown to cause hemolysis (Mackell et al., 1951); 1,2-naphthoquinones, which have been implicated in cataract formation (Rees and Pirie, 1967); and 3-glutathione adducts (arising from naphthalene-1,2-oxides), which are believed to be involved in pulmonary toxicity following intraperitoneal administration (Buckpitt et al., 1984).

In nonhuman primate studies, Rozman et al. (1982) reported that rhesus monkeys given naphthalene at oral doses up to 200 mg/kg did not excrete naphthalene as premercapturic or mercapturic acid conjugates in urine or feces. In a similar study, Summer et al. (1979) found that chimpanzees orally administered naphthalene at 200 mg/kg did not excrete naphthalene as mercapturic acids in urine. These data suggest that mercapturic acid conjugation is of little importance in nonhuman primates.

Animal studies indicate that oral doses of naphthalene at 300 to 500 mg/kg/day are lethal to mice (Plasterer

et al., 1985), but rats (Yamauchi et al., 1986) and rabbits (Rossa and Pau, 1988) tolerated doses up to 1,000 mg/kg.

In a study to determine the lethal dose of naphthalene in rats, Fait and Nachreiner (1985) reported that upon 4-hour exposure to 78 ppm (the highest level that could be generated in their inhalation chambers), no deaths occurred, no adverse clinical signs were observed during or 14 days after exposure, and no gross pathologic lesions were observed at necropsy.

In rats, no significant respiratory toxicity was seen following administration of naphthalene at doses up to 750 mg/kg/day for 9 weeks (Germansky and Jamall, 1988). In this study, dosages were increased from 100 to 750 mg/kg/day; a time-weighted averaged exposure of 450 mg/kg/day was calculated by the authors. Shopp et al. (1984) reported increased lung weights in female mice administered naphthalene at 267 mg/kg/day for 14 days; however these effects were not seen in either sex at 133 mg/kg/day for 90 days.

Few hematologic changes have been reported in animals, and standard laboratory animals do not appear to be sensitive to the hemolytic effects of naphthalene. In CD-1 mice, naphthalene at doses up to 267 mg/kg/day for 14 days or up to 133 mg/kg/day for 90 days did not result in hemolytic anemia (Shopp et al., 1984). Observed hematologic effects in this study included decreased prothrombin time and an increase in eosinophils in the 14-day study and increased hemoglobin and eosinophils in the 90-day study. The clinical significance of these observations is not clear. The authors concluded that the CD-1 mouse is not an appropriate model for hemolytic anemia. Hemolytic anemia was reported by Zuelzer and Apt (1949) in a dog receiving a single 1,525 mg/kg/day dose of naphthalene in food and in another dog receiving approximately 263 mg/kg/day dose for 7 days in food. The results of this study suggest that the dog may be a suitable model to test the hemolytic effects of naphthalene. Because an acceptable study using an appropriate animal model has not been located, an MRL for oral exposure to naphthalene has not been calculated.

There is limited evidence of hepatic effects in laboratory animals. A 39% increase in liver weight and modest elevations in tissue activities of aniline hydroxylase and lipid peroxidase were observed in male rats treated with naphthalene at 1,000 mg/kg/day for 10 days (Rao and Pandya, 1981). Male rats demonstrated an elevation in hepatic lipid peroxides at naphthalene doses of 1,000 mg/kg/day for 18 days (Yamauchi et al., 1986). Similarly, Germansky and Jamall (1988) reported that in rats administered increasing doses of naphthalene up to 750 mg/kg/day, hepatic lipid peroxidase activity was doubled at the end of 9 weeks of treatment. No effects on liver weight were observed in mice receiving naphthalene at doses up to 267 mg/kg/day for 14 days or 133 mg/kg/day for 90 days (Shopp et al., 1984).

Renal effects have not been conclusively observed in animals exposed to naphthalene. Following 10 days of oral exposure of rats to naphthalene at 1,000 mg/kg/day, no changes were noted in kidney weight or in the activities of alkaline phosphatase, aniline hydroxylase, or lipid peroxidase (Rao and Pandya, 1981). Shopp et al. (1984) reported that no changes were observed in the kidney weights of mice administered naphthalene at doses up to 267 mg/kg/day for 14 days or 133 mg/kg/day for 90 days.

Oral exposure of pregnant rabbits to naphthalene at dosages up to 400 mg/kg/day, using methylcellulose as the vehicle, resulted in no apparent adverse reproductive effects (PRI, 1986). When administered in corn oil to pregnant mice, however, a dosage of 300 mg/kg/day resulted in a decrease in the number of live pups per litter (Plasterer et al., 1985). It is not clear whether the observed differences in response are attributable to species differences or a possible increase in the absorption of naphthalene when it is administered in corn oil. Shopp et al. (1984) did not observe any effect on testicular weights of mice administered naphthalene at doses up to 267 mg/kg/day for 14 days or 133 mg/kg/day for 90 days.

In a two-year feeding study in rats receiving naphthalene at about 41 mg/kg/day, Schmahl (1955) reported that no tumors developed. (Based on tumor data presented for another chemical in the report, it is assumed that at least hepatic and uterine tissue were examined in naphthalene-treated rats. However, no specific tissues were mentioned for naphthalene-treated rats).

Mice treated with naphthalene at oral doses as high as 267 mg/kg/day for 14 days showed no effects on humoral immune responses, delayed type hypersensitivity responses, bone marrow stem cell number, or bone marrow DNA systhesis. Thymic weights were reduced approximately 40% in males and splenic weights were reduced approximately 20% in females. None of these effects were noted at the next lower dose of 53 mg/kg/day. At doses of 133 mg/kg/day for 13 weeks, naphthalene had no effect on immune function (Shopp et al., 1984). The only change noted was a 25% decrease in splenic weight in females, which is of questionable biological significance.

When either naphthalene or 2-methylnaphthalene was applied dermally in combination with (B[a]P), there was an inhibitory effect on the induction of skin tumors in female mice (Schmeltz et al., 1978). These investigators also reported that a mixture containing naphthalene (0.02%), 2-methylnaphthalene (0.02%) and 10 other methylated and ethylated naphthalenes (each at 0.02%) also appeared to inhibit the development of B[a]P-induced skin tumors. The authors suggested that it is likely that certain naphthalenes compete with B[a]P for the same enzyme sites, resulting in alteration of the B[a]P metabolic pathway and decreased production of the active B[a]P metabolite. Dermal application of the naphthalene mixture did not induce tumors in the absence of B[a]P. The results of these studies were not analyzed statistically.

EPA has placed naphthalene in Group D, not classified as to carcinogencity.

13.3 Human Toxicity

Inhalation has been associated with many effects, including headache, nausea, vomiting, abdominal pain, malaise, confusion, mild anemia, jaundice and renal disease (ATSDR, 1989b). Many of these effects, however, are confounded by the simultaneous exposure to other agents.

Anemias are the most frequently reported cases of naphthalene poisoning in humans (ATSDR, 1989). Acute hemolytic anemia was observed in 21 infants exposed to woolen clothes or blankets treated with mothballs. Symptoms include high serum bilirubin values, Heinz bodies, and fragmentation of the red blood cells (ATSDR, 1989). Inhalation was the assumed route of exposure because there was little or no direct skin contact. Anemia has also been reported by individuals exposed to large numbers of mothballs in their homes. The air concentration in one of the homes was measured at 20 ppb (ATSDR, 1989). Other inhalation effects include respiratory tract irritation, headache, nausea, and profuse perspiration depending on the concentration and duration (Clayton & Clayton, 1981). Optic neuritis, corneal ulceration and cataracts have also been observed in workers exposed in industry.

Human deaths have occurred following ingestion of mothballs. A 30 year old female died after swallowing 40 mothballs and a 17 year old male died after swallowing an unknown amount (ATSDR, 1989). From the autopsy of the female, 25 mothballs were recovered and it is estimated that the exposure level was 574 mg/kg (ATSDR, 1989). Other severe effects from ingestion include gastroenteric distress, tremors, and convulsions (Clayton & Clayton, 1981). Heinz bodies appear and the serum may become a yellowish-brown color. Neurological effects from ingestion include confusion, listlessness and lethargy, and vertigo (ATSDR, 1989). Muscle twitching, convulsions, decreased responses to painful stimuli and coma have also been reported at extreme exposure levels.

PAHs generally have been detected at low concentrations in surveys of human adipose tissue and other biological media, presumably because the compounds are fairly rapidly metabolized (ATSDR, 1989c). The U.S. EPA National Human Adipose Tissue Survey (U.S. EPA, 1989b) found that, in 46 composite samples of adipose tissue examined in 1982, naphthalene was detected in 42% of the samples.

The human lung does not appear to be a target organ for naphthalene. No reports of human pulmonary toxicity after inhalation, oral or dermal exposure to the chemical were found. However, some studies have shown that the intraperitoneal administration of naphthalene (125 to 400 mg/kg) caused pulmonary necrosis of Clara cells in some strains of mice (Tong et al., 1981, 1982; Warren et al., 1982). Clara cells are rich in cytochrome P-450 enzymes and thus may be capable of producing cytotoxic metabolites of naphthalene. Because Clara cell damage has only been reported to occur in certain strains of mice following intraperitoneal administration, the relationship of this effect to potential human health effects is not evident.

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malaise, confusion, mild anemia, jaundice and renal disease (ATSDR, 1989b). Many of these effects, however, are confounded by the simultaneous exposure to other agents.

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Human deaths have occurred following ingestion of mothballs. A 30 year old female died after swallowing 40 mothballs and a 17 year old male died after swallowing an unknown amount (ATSDR, 1989c). From the autopsy of the female, 24 mothballs were recovered and it is estimated that the exposure level was 574 mg/kg (ATSDR, 1989c). Other severe effects from ingestion include gastroenteric distress, tremors, and convulsions (Clayton & Clayton, 1981). Heinz bodies appear and the serum may become a yellowish-brown color. Neurological effects from ingestion include confusion, listlessness and lethargy, and vertigo (ATSDR, 1989c). Muscle twitching, convulsions, decreased responses to painful stimuli and coma have also been reported at extreme exposure levels.

14.0 PHENANTHRENE

Phenanthrene is a solid at room temperature. It is not produced commercially but is present in crude oil, coal tar, gasoline, and cigarette smoke (IARC, 1983). Physical and chemical properties of phenanthrene are listed in Table 13.

14.1 Genotoxicity

The majority of mutagenicity test results for phenanthrene are negative, although positive results have been reported for this chemical in at least one *in vitro* test. Phenanthrene was mutagenic in *Salmonella typhimurium*, and positive results were obtained in several *in vitro* mammalian cell systems (ATSDR, 1989c).

Phenanthrene is among the PAHs generally considered inactive as tumor initiating agents (ATSDR, 1989c).

14.2 Animal Toxicity

The intraperitoneal LD_{50} in mice for phenanthrene is 700 mg/kg body weight (Gerarde, 1960; Salamone et al., 1981).

Rats exposed intragastrically to 100 mg/kg/day of phenanthrene exhibited a 30% increase in carboxylesterase activity of the intestinal mucosa (Nousiainen et al., 1984). Enzyme alteration in the absence of other signs of gastrointestinal toxicity was not considered an adverse health effect, but may precede the onset of more serious effects. Given the selectivity of PAHs for rapidly proliferating tissues such as gastrointestinal mucosa, oral exposure to PAHs at higher doses could lead to adverse gastrointestinal effects (ATSDR, 1989c).

The ability to induce aldehyde dehydrogenase (ADH) in animals has been correlated with carcinogenic potency. Rats intragastrically administered 100 mg/kg/day of phenanthrene for four days exhibited cytosolic ADH induction (Torronen et al., 1981). However, the authors concluded that phenanthrene, which has been characterized as a non-carcinogen, is a poor ADH inducer (Torronen et al., 1981).

The extent of liver regeneration, which is indicative of the ability to induce a proliferative response, also has been examined following acute oral exposure to various PAHs. Partially hepatectomized rats were fed diets containing various PAHs for 10 days. Administration of 514 mg/kg/day phenanthracene had no effect on the extent of liver regeneration. Diets containing 180 mg/kg/day phenanthrene produced no increase in the liver-to-body weight ratio (Gershbein, 1975).

EPA has placed phenanthrene in Group D, not classified as to carcinogenicity.

14.3 Human Toxicity

PAHs are absorbed through the skin of humans. Application of 2% crude coal tar to the skin of humans for eight hour periods on two consecutive days yielded evidence of PAH absorption (Storer et al., 1984). Phenanthrene was detected in the blood, but absorption of PAHs in crude coal tar was variable and dependent on the chemical species.

Quantitative studies were not found regarding the distribution, accumulation or excretion of PAHs in humans. However, it appears that there is little tendency for long-term bioaccumulation of PAHs in human tissue (Lee et al., 1972; Ahokas et al., 1975). PAHs generally have been detected at low concentrations in surveys of human adipose tissue and other biological media, presumably because the compounds are fairly rapidly metabolized (ATSDR, 1989c). The U.S. EPA National Human Adipose Tissue Survey (U.S. EPA, 1989b) found that, in 46 composite samples of adipose tissue examined in 1982, phenanthrene was present in 14% of the samples.

15.0 PYRENE

Pyrene is a colorless solid, and is formed as a result of incomplete combustion of fossil fuels. It is present in high concentrations in coal tar (IARC, 1983). Human exposure to this compound is primarily through smoking of tobacco, inhalation of polluted air, and by ingestion of food and water contaminated by combustion byproducts (IARC, 1983). Physical and chemical properties of pyrene are listed in Table 14.

15.1 Genotoxicity

The majority of mutagenicity test results for pyrene are negative, although positive results have been reported for this chemical in at least one *in vitro* test. Pyrene was mutagenic in *Salmonella typhimurium*, and positive results were obtained in several *in vitro* mammalian cell systems (ATSDR, 1989c).

Pyrene is among the PAHs generally considered inactive as tumor initiating agents (ATSDR, 1989c).

15.2 Animal Toxicity

Oral LD_{50} values for the mouse and rat are 800 and 2,700 mg/kg, respectively. The inhalation LC_{50} for rat is 170 mg/m^3 (RTECS, 1987).

The intraperitoneal LD_{50} in mice for pyrene is 680 mg/kg body weight (Gerarde, 1960; Salamone et al., 1981).

Dilated tubules were observed in the kidneys of mice administered pyrene in the diet for 25 days (Rigdon and Giannukos, 1964); the toxicological significance of this effect is not known. Additional effects in the rat from inhalation were hepatic, pulmonary, and intragastric pathologic changes, plus a decrease in the number of some blood components neutrophils, leukocytes, and erythrocytes) (Clayton & Clayton, 1981). Application of pyrene (5 µmol to 5 mmol in ethanol) to guinea pig skin produced a strongly phototoxic response following 20 hours of exposure (HSDB, 1989c).

Mice chronically administered a 10% pyrene solution throughout their lifetimes did not develop skin tumors (Wynder and Hoffman, 1959). However, prolonged dermal exposure of mice to 0.5% pyrene in decalin:n-dodecane solvent produced a slightly elevated (15%) skin carcinoma incidence; the level of statistical significance was not provided (Horton and Christian, 1974).

15.3 Human Toxicity

Autopsies performed on cancer-free patients found total PAH levels ranging from 11 to 2,700 ppt (parts

per trillion; ng/kg) in fat samples. Several PAHs were detected with pyrene being detected in the highest concentrations (Obana et al., 1981). A similar study done on liver tissue from cancer-free patients reported levels ranging from 6 to 500 ppt of the same PAH. As in the fat samples, pyrene appeared in the highest concentrations in the liver, but the concentrations were less than in fat (Obana et al., 1981).

Pyrene is absorbed through the skin of humans. Pyrene was detected in the blood after application of 2% crude coal tar to the skin of humans for eight hour periods on two consecutive days (Storer et al., 1984).

The U.S. EPA National Human Adipose Tissue Survey (U.S. EPA, 1989b) found that, in 46 composite samples of adipose tissue examined in 1982, pyrene was not detected.

TABLE 16

Carcinogen Classification of Selected PAHs

Compound	IARC* (1987)	EPA** (HEAST, 1990)
Acenaphthylene		D***
Anthracene	3	D
Benzo(a)anthracene	2A	B2
Benzo(b)fluoranthene	2B	B2
Benzo(g,h,i)fluoranthene	3	
Benzo(j)fluoranthene	2B	
Benzo(k)fluoranthene	2B	B2
Benzo(a)fluorene	3	
Benzo(b)flurorene	3	
Benzo(c)flurorene	3	
Benzo(g,h,i)perylene	3	D
Benzo(c)phenanthrene	3	
Benzo(a)pyrene	2A	B2
Benzo(e)pyrene	3	
Chrysene	3	B2
Dibenzo(a,c)anthracene	3	
Dibenzo(a,h)anthracene	2A	B2
Dibenzo(a,e)fluoranthene	3	
Dibenzo(a,e)pyrene	2B	
Dibenzo(a,e)pyrene	2B	
Dibenzo(a,h)pyrene	2B	
Dibenzo(a,i)pyrene	2B	
Dibenzo(a,l)pyrene	2B	
Fluoranthene	3	D
Fluorene		D
Indeno(1,2,3-cd)pyrene	2B	B2
Naphthalene		D
Phenanthrene	3	D
Pyrene	3	D

* IARC Group:
2A-Limited evidence of carcinogenicity to humans.
2B-Sufficient evidence in animals and inadequate data in humans.
3- Cannot be classified as to its carcinogenicity to humans.

** EPA Group B2- Probable human carcinogen; sufficient evidence from animal studies and insufficient evidence from human epidemiologic studies.

*** Group D- Not classified as to human carcinogenicity.

TABLE 17

Regulations, Standards and Guidelines for Polynuclear Aromatic Hydrocarbons*

EPA Ambient Water Criteria*		HSDB, 1989c
Organism and water consumption	2.8 ng/L for $1x10^{-6}$ risk	
Organism consumption only	31.1 ng/L for $1x10^{-6}$ risk	
OSHA-TLV		
Coal tar volatiles	0.02 mg/m^3	OSHA, 1989
Naphthalene	50 mg/m^3	OSHA, 1989
Benzo(a)pyrene	0.2 mg/m^3	OSHA, 1989
State of Florida Surface Water Quality Standards*		FDER, 1990
Class I (potable) waters Organism and water consumption	2.8 mg/L for 10^{-6} risk	
Class II and III waters Organism consumption	31.1 mg/L for 10^{-6} risk	

* Guidelines presented here are for PAHs as a class.

16.0 DOSE RESPONSE ASSESSMENT FOR PAHs

Dose-Response Assessment is the process of characterizing the quantitative relationship between the dose of a chemical or agent and the incidence of an adverse health effect in exposed populations (NRC, 1983). The end result of the dose-response assessment is a probability estimate of the incidence of the adverse effect as a function of human exposure to the chemical. Any given adverse health effect is evaluated separately. This section focuses on both the carcinogenic and noncarcinogenic dose-response relationships for PAHs.

16.1 Noncarcinogenic PAHs

Evaluation of noncarcinogenic effects is based on a comparison of an estimated daily exposure level to an allowable daily exposure level, often represented by the U.S. EPA Reference Dose (RfD). In general, the RfD is an estimate (with uncertainty spanning up to three orders of magnitude) of a daily dose to the human subpopulation (including sensitive subgroups) that is likely to result in negligible risk of deleterious effects during a lifetime of exposure (HEAST, 1989). The RfD is based on the assumption that a threshold exists which must be overcome before adverse effects are observed. The safety factor (or uncertainty factor) used in the derivation of an RfD generally consists of multiples of 10, each factor representing an area of uncertainty in the available data. The safety factors are applied to the No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL). The NOAEL is an experimentally determined dose below which there was no statistically or biologically significant indication of the toxic effect of concern. The LOAEL, however, is the lowest experimentally determined dose at which effects were observed. In cases where the NOAEL has not been demonstrated experimentally, the LOAEL is used. A factor of 10 may be applied to the NOAEL to account for differences in responsiveness between humans and animals in prolonged exposure studies and an additional factor of 10 may be used to account for variability in susceptibility among individuals in the human population. Typically, a factor of 10 also is applied if the LOAEL, rather than the NOAEL is used.

For pyrene, the RfD has been established at 3E-02 mg/kg/day (U.S. EPA, 1990; personal communication). A study by U.S. EPA (1989d) found nephropathy and decreased kidney weight in mice given 75 mg/kg/day by gavage for 13 weeks. This study led to the establishment of the NOAEL at 75 mg/kg/day.

The RfD for anthracene is 3E-01 mg/kg/day (U.S. EPA, 1990; personal communication). This value was derived from a study by U.S. EPA (1986b) in which mice were given 1,000 mg/kg/day by gavage for 90 days. This is the highest dose that has been tested and, since no adverse effects were reported, 1,000 mg/kg/day was established as the NOEL.

An RfD of 4E-02 mg/kg/day has been established for fluoranthene (U.S. EPA, 1990; personal communication) based on a study by U.S. EPA (1988). Nephropathy, increased relative liver weights, hematological and clinical effects were observed in this study when mice were administered 125 mg/kg/day or 250 mg/kg/day by gavage. From this study, the NOAEL of 125 mg/kg/day and the LOAEL of 250 mg/kg/day were established.

The RfD for fluorene is 4E-02 mg/kg/day (U.S. EPA, 1990; personal communication). This value was derived from a study by U.S. EPA (1989c) in which mice were administered 125 mg/kg/day or 250 mg/kg/day by gavage for 13 weeks. Adverse effects of such treatment were decreased red blood cell counts and packed cell volume and hemoglobin. This study led to the establishment of the NOAEL at 125 mg/kg/day and the LOAEL of 250 mg/kg/day.

The RfD for naphthalene has been established at 4E-03 mg/kg/day (U.S. EPA, 1990; personal communication). This value was derived from studies by Schmahl (1955) and U.S. EPA (1988) in which rats were given 10-20 mg/day in the diet for 6 days/week for approximately 700 days (converted to 41 mg/kg/day). The rats were observed to have ocular and internal lesions at this dose, but not at 4 mg/kg/day.

16.2 Carcinogenic PAHs

The assessment of carcinogenic effects is a weight-of-evidence determination of whether or not a chemical is a human or animal carcinogen or both based on human or animal data. Carcinogenesis is currently considered by the U.S. EPA to be a nonthreshold phenomenon (i.e., it is assumed that no dose of a carcinogenic agent is without some risk of carcinogenic response). For chemicals classified as known human (Group A) or probable human (Group B1 and B2) carcinogens, a toxicity value (in this case a cancer potency factor) is derived from the plot of the incidence of cancer versus the dose of the substance, and is expressed in units of $(mg/kg\text{-}day)^{-1}$. Low-dose incidence of cancer is estimated through the use of a mathematical model which extrapolates low-dose cancer incidence from high-dose experimentally

determined data. The U.S. EPA uses a linearized multi-stage (LMS) model to calculate the CPF. The selection and applicability of the modeling output from the LMS for health-based risk assessment is based on three assumptions: 1) human and animal physiological response is equal, 2) the dose-response curve is linear in the low dose region and passes through the origin (i.e., non-threshold), and 3) the value which represents the upper 95 % confidence limit on the data is a de minimis risk (i.e., there is a probability of 5% that a carcinogenic response will be higher than the estimate predicted by the model (U.S. EPA, 1989).

16.3 Route-Specific Cancer Potencies for B(a)P

Currently, cancer potency factors have been developed for only B(a)P. The following sections summarize the derivation of the cancer potency estimate for B(a)P. These values are considered interim, and are under review by EPA.

16.3.1 Oral Cancer Potency

U.S. EPA based its present oral B[a]P potency factor on a study published by Neal and Rigdon in 1967 (U.S. EPA, 1984). Because of an unconventional study design in which there were differing lengths of exposure time and study duration among the study groups, only the data from low dose exposures were used by U.S. EPA in the potency estimate. The Carcinogen Assessment Group of U.S. EPA is now reevaluating this study using statistical techniques that will allow the incorporation of all the Neal and Rigdon data in a final potency estimate. The current U.S. EPA oral cancer potency factor for B(a)P is 11.5 $(mg/kg\text{-}day)^{-1}$; this cancer potency factor was also utilized to assess dermal exposure.

Neal and Rigdon (1967) fed doses of 0 to 250 ppm B[a]P in food to male and female CFW-Swiss mice. Treatment groups varied from 9 to 73 animals. U.S. EPA assumed the average weight of a Swiss mouse was .035 kg and the average daily intake of food was approximately 4.55 g/day. These values result in a dose rate of 0 to 32.5 mg/kg-day. Exposure durations ranged from 1 to 197 days and total duration of the study for individual treatment groups ranged from 88 to 300 days. Papillomas and

carcinomas of the forestomach were observed in mice consuming 20 or more ppm B(a)P; results were combined for the determination of total tumor incidence. No distinction was made between benign and malignant neoplasms in the calculation of the carcinogenic potency factor.

16.3.2 Inhalation Cancer Potency

The U.S. EPA based its inhalation B(a)P potency factor of 6.11 $(mg/kg\text{-}day)^{-1}$ on the study by Thyssen *et al.* (1981) in which male hamsters developed respiratory tumors following administration of B[a]P in an aerosol of NaCl solution (U.S. EPA, 1984; Thyssen et al., 1981).

Syrian golden hamsters were exposed to levels of 0, 2.2, 9.5 or 45 mg/m^3 B(a)P for 4.5 hours/day for 10 weeks followed by 3 hours/day (7 days/week) for up to 675 days. No animals in the low dose group developed respiratory tumors. The mid-dose (9.5 mg/m^3) and high-dose groups (45 mg/m^3) exhibited respiratory tract tumors in the nasal cavity, larynx, and trachea. Other tumors thought to be dose-related because of mucociliary particle clearance include those found in the pharynx, esophagus and forestomach.

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B

GROUNDWATER
TECHNOLOGY, INC.

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO
PROJECT NUMBER 0110-5470-0220
DATE OF SAMPLING 8/23/90
CONTAMINANTS - BTEX
- PNA's

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID MICROTIP	11.7eV PID	% LEL	% O_2	mR/hr			
1:00		0					PARKING LOT NEAR BIOCELL	H_2S	MLG
1:50		0					" "	"	"
2:30		0					" "	"	"
3:10		0					" "	"	"
4:30		0					" " IN VICINITY OF VP-1	"	"
4:49		0					" " IN VICINITY O VP-1	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO
PROJECT NUMBER 01110 5470 0220
DATE OF SAMPLING 8/24/90
CONTAMINANTS • BTEX
• PNA

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID	11.7eV PID	% LEL	% O_2	mR/hr			
8:45		0					VICINITY OF VP-2	H_2S	"
10:30		0					VICINITY OF VP-2	"	"
11:55		0					VICINITY OF VP-3	"	"
12:30		0					VICINITY OF VP-3	"	"
1:15		0					VICINITY OF VP-3	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO
PROJECT NUMBER 01110 5470
DATE OF SAMPLING 10/1/90
CONTAMINANTS BTEX & PNA

CALIBRATED MICROTIP @ 9:15

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID	11.7eV PID	% LEL	% O_2	mR/hr			
9:45		0.0					MW-8	H2S	MLG
10:00		0.0					"	"	"
~~9:45~~ 16:45		0.0					"	"	"
10:55		0.0					"	"	"
11:30									
2:15		0.0					"	"	"
2:49		0.0					MW-8	"	"
9:22		0.0					CW-1	"	"
9:27		1.0					"	"	"
10:00		0.0					CW-1	"	"
10:35		0.0					"	"	"
10:52		0.0					"	"	"
12:30		0.0					"	"	"
1:55		0.0					"	"	"
2:40		0.0					"	"	"
3:20		0.0					"	"	"
5:10		0.0					"	"	"
6:40		0.0					"	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME: OSMOSE / BUFFALO
PROJECT NUMBER: 01110-5470-0203
DATE OF SAMPLING: 10/3/90
CONTAMINANTS: CREOSOTE, #2 FUEL OIL, CRESOL

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID MICROTIP	11.7eV PID	% LEL	% O_2	mR/hr			
8:30		0.0					VICINITY OF CW-1	H&S	MLG
8:45		0.0					"	"	"
9:00		0.0					"	"	"
9:17		0.0					"	"	"
9:41		0.0					"	"	"
3:51		0.0					"	"	"
4:01		0.0					"	"	"
5:42		0.0					"	"	"
10/4									
8:00		0.0					VICINITY OF CW-1	"	"
9:30		0.0					"	"	"
10:00		0.2					"	"	"
10:15		0.0					"	"	"
11:30		0.0					"	"	"
12:45		1.0					"	"	"
1:40		0.0					"	"	"
2:10		2.3					"	"	"
3:40		1.2					"	"	"
5:10		0.0					"	"	"
7:00		0.0					"	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO
PROJECT NUMBER 01110 5470 0203
DATE OF SAMPLING 10/5/90
CONTAMINANTS CREOSOTE, #2 FUEL OIL, CREOSOTE

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID	11.7eV PID	% LEL	% O_2	mR/hr			
7:30		0.0					VICINITY OF CW-2	11:5	MLG
8:00		0.0					" "	"	"
8:15		0.0					" "	"	"
9:15		0.0					" "	"	"
10:00		0.0					" "	"	"
10:20		0.0					" "	"	"
11:30		0.0					" "	"	"
12:15		0.0					" "	"	"
1:00		0.0					" "	"	"
1:20		0.0					" "	"	"
2:30		0.0					" "	"	"
2:50		0.0					" "	"	"
3:10		0.0					" "	"	"
4:15		0.0					" "	"	"
6:10		0.0					" "	"	"
8:00		0.0					" "	"	"
8:30		0.0					" "	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME: OSMOSE / BUFFALO
PROJECT NUMBER: 0111 0 5470 0203
DATE OF SAMPLING: 10/6/90
CONTAMINANTS: CREOSOL, CREOSOTE, #2 F.O.

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID	11.7eV PID	% LEL	% O_2	mR/hr			
7:00		0.0					MW-10	H & S	MLG
7:15		0.0					"	"	"
8:20		0.0					"	"	"
9:00		0.0					"	"	"
10:15		0.0					"	"	"
10:35		0.0					"	"	"
10:40		0.0					"	"	"
11:00		0.0					"	"	"
12:15		0.0					"	"	"
12:45		0.0					"	"	"
1:30		0.0					"	"	"
2:10		0.0					"	"	"
3:10		0.0					"	"	"
4:30		0.0					SB-1	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO
PROJECT NUMBER 01110 - 5470 - 0203
DATE OF SAMPLING 10/16/90
CONTAMINANTS CREOSOL, CREOSOTE, #2 F.O.

RESULTS OF VAPOR MONITORING

TIME	IONIZATION DETECTOR READING			EXPLOSIMETER READING		RADIATION MONITOR READING	LOCATION	PURPOSE	INITIALS
	FID	10.2eV PID	11.7eV PID	% LEL	% O_2	mR/hr			
8:45		0					SB-1	11 3 5	MLG
9:30		0					"	"	"
10:00		0					"	"	"
10:15		0					"	"	"
10:30		0					"	"	"
11:30		0					"	"	"
12:10		0					MW-11	"	"
12:40							"	"	"
1:50		0					"	"	"
2:30		0					"	"	"
3:00		0					"	"	"
3:15		0					"	"	"
3:30		0					"	"	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE/BUFFALO

PROJECT NUMBER 01110 5470 0203

SAMPLING DATE 10/1/90

MINI-RAM SAMPLING LOG

TIME	READING	SA	~~TWA~~ TWA	SAMPLE LOCATION
9:49	0.0	0	0	MW-8
10:03	0.0	0	0	MW-8
10:21	0.0	0.22	2.75	"
10:42	0.0	0.36	3.09	"
10:55	0.0	0.29	2.79	"
11:30	4.3	0.29	2.28	"
2:15	0.29	0.00	0.11	"
2:50	0.00	0.01	0.08	"
10/2 9:24	0.49	0.31	2.24	CW-1
10:13	0.21	0.40	1.66	"
10:32	0.15	0.44	1.57	"
10:51	1.11	0.50	1.58	"
12:30	0.32	0.03	0.10	"
10/3 8:47	0.00	0.03	2.22	"
9:00	0.0	0.05	1.34	"
9:15	7.12	0.17	2.30	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO

PROJECT NUMBER 0110 5470 0203

SAMPLING DATE 10/3/90

MINI-RAM SAMPLING LOG

TIME	READING	SA	TWA ~~TLV~~	SAMPLE LOCATION
9:41	4.0	0.34	2.65	VICINITY OF GW-1
3:47	0	0	0.55	VICINI "
4:02	0.0	0.02	0.57	"
5:27	0	0.03	0.14	"
5:43	0	0.03	0.13	"
6:41	0	0.03	0.09	
10/5/90 7:35	0	0.00	0.00	VICINITY OF GW-2
8:10	0	0.01	0.08	"
8:30	0	0.01	0.07	"
9:10	0.09	0.03	0.11	"
10:15	0	0.02	0.13	"
11:00	1.32	0.21	1.18	"
12:10	0.09	0.11	0.95	"
12:30	0	0.22	1.23	"
1:00	0.25	0.33	1.28	"
2:10	0.11	0.25	1.38	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO

PROJECT NUMBER 0110 5470 0203

SAMPLING DATE 10/5/90

MINI-RAM SAMPLING LOG

TIME	READING	SA	TWA ~~TLV~~	SAMPLE LOCATION
2:45	1.2	0.09	3.21	MW-9
3:30	0	0.22	2.31	"
4:10	0	0.18	1.95	"
5:20	1.1	0.09	2.80	"
6:10	0.0	0.03	1.18	"
8:00	0.0	0.02	1.11	"
10/6/90				
7:00	1.8	0.03	1.01	MW-10
8:10	1.1	0.09	1.28	"
9:30	0.0	0.01	0.09	"
10:00	1.0	0.51	1.63	"
10:40	4.3	0.29	2.73	"
11:00	0.49	0.38	2.21	"
11:41	1.11	0.59	1.63	"
12:15	0.0	0	0	"
12:35	0	0.22	2.75	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO

PROJECT NUMBER 01110 5470 0203

SAMPLING DATE 10/6/20

MINI-RAM SAMPLING LOG

TIME	READING	SA	TWA ~~TLV~~	SAMPLE LOCATION
1:15	0.38	0.02	0.10	MW-10
2:30	0.45	0.01	0.23	"
3:10	0.56	0.32	2.83	SB-1
4:10	1.63	0.19	1.62	"
4:30	1.23	0.52	1.83	"

GROUNDWATER TECHNOLOGY, INC.

PROJECT NAME OSMOSE / BUFFALO

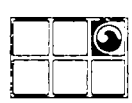
PROJECT NUMBER 01110 5470 0203

SAMPLING DATE 10/16/90

MINI-RAM SAMPLING LOG

TIME	READING	SA	TLV	SAMPLE LOCATION
9:20	0	0	0.55	VICINITY OF SB-1
9:45	0	0	0.12	"
10:00	0	0	0.13	"
10:15	0	0	0.09	"
10:30	1.2	0.8	1.08	"
11:00	0.9	0.35	1.00	"
1:00	0.0	0.01	0.08	MW-11
1:15	0.15	0.43	1.60	"
2:00	0.0	0.03	2.25	"
2:15	1.11	0.50	1.53	"
2:30	0	0.05	1.33	"
3:00	0.21	0.10	1.62	"

C



GROUNDWATER
TECHNOLOGY, INC.

8/23/90
SOIL VENT SURVEY

5470-0220

SOIL TAKEN?	SAMPLING LOCATION	PID READING	PNA START	PNA FINISH	PNA FLOW RATE	BTEX START	BTEX FINISH	BTEX FLOW RATE
No 8/23	EQUIP. BLANK	N/A	3:00	3:20	20L @ 1L/MIN	3:27	3:32	5L @ 1L/MIN
No	TRIP BLANK	0 (AMBIENT)	2:45	N/A	N/A	2:42	N/A	N/A
No	VP-1	2.1	4:45	5:05	20L @ 1L/MIN	4:35	4:40	5L @ 1L/MIN
No 8/24	VP-2	10.9 *	10:15	10:35	20L @ 1L/MIN	10:43	10:48	5L @ 1L/MIN
No	3	28.4 *	11:14	11:34	"	11:05	11:10	"
No	4	54.5 *	12:07	12:27	"	12:35	12:40	"
YES	5	23.4 *	1:10	1:30	"	1:00	1:05	"
YES	6	0	2:00	2:20	"	2:45	2:50	"
YES	7	2.3	3:48	4:08	20 MIN @ 32.5	3:30	3:35	5MIN/32.5
YES 8/25	8	1.0	11:21	11:41	20MIN @ 30.0	11:44	11:50	6MIN/30.0
YES	9	1.6	12:17	12:37	20 MIN @ 30.0	12:40	12:45	5MIN/30.0
YES	10	2.6	1:00	1:20	20 MIN @ 30.0	1:22	1:27	5MIN/29.0
YES	11	1.3	2:11	2:39	28 MIN @ 30.0	2:40	2:47	7 MIN/30.0
YES	12	1.1	3:04	3:32	28 MIN @ 30.0	3:39	3:46	7MIN/30.0
YES	13	3.1	4:23	4:51	28 MIN @ 30.0	4:52	4:59	7MIN/30.0
YES	14	1.8	5:13	5:41	28 MIN @ 30.0	5:45	5:52	7MIN/30.
YES	15	4.6 *	7:00	7:28	28 MIN @ 30.0	7:30	7:37	7 MIN/30.0
YES	16	1.2	7:59	8:27	28 MIN @ 30.0	7:50	7:57	7/MIN/30.0
Yes	17	1.0	8:35	9:03	28 MIN @ 30.0	9:05	9:12	7MIN/30.0
* VP-6 BLIND DUP	18	0	2:22	2:42	20L @ 1L/MIN	3:00	3:05	5MIN/28.0

SOILS:	TIME SAMPLED	NO. OF CONTAINERS	BLIND DUP?
8/24 VP-5	1:40	2	
VP-6	3:35	2	YES: VP-18
VP-18	3:30	2	
8/25 VP-7	11:36	2	
VP-8	12:24	2	
VP-9	1:15	2	
FIELD BLANK	1:30	2 (2.5 GOAL PLASTIC)	
VP-10	2:12	2	
VP-11	3:15	2	
VP-12	4:27	2	
VP-13	5:20	2	YES: VP-19
VP-19	5:26	2	
VP-14	6:10	2	
VP-15	8:08	2	
VP-16	9:05	2	
VP-17	9:21	2	

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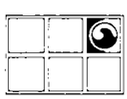


GROUNDWATER
TECHNOLOGY, INC.

GROUNDWATER TECHNOLOGY
Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-3

Project OSMOSE WOOD PRES. Owner _____

Location BUFFALO NY Project Number 110-001-1344

Date Drilled 6/21/89 Total Depth of Hole 18.0' Diameter _____

Surface Elevation _____ Water Level, Initial 8.0' 24-hrs. _____

Screen: Dia. 2.0" Length 15.0' Slot Size .020"

Casing: Dia. 2.0" Length 3.0' Type PVC

Drilling Company GTI Drilling Method HOLLOW STEM AUGER

Driller MUTHERN/BERNHARD Log by J. MEIER

Sketch Map

Notes

Depth (Feet)	Well Construction	Notes	Sample Number	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
0		ROADBOX	PID READING IN PPM		0.0-3.0' DARK BLACK FINE-MED SAND AND CLAY, SOME FINE-COARSE GRAVEL, LITTLE SILT.
1		CEMENT			
2		BENTONITE			
3		WELL CASING			3.0-5.0' MEDIUM BROWN DRY SILT AND CLAY, TRACE OF FINE SAND.
4		WELL SCREEN			
5			1.0		SPOON 5.0-7.0 DARK BROWN & GRAY MOTTLED DRY CLAY.
6					
7					
8					
9		SAND PACK			
10			95.0		SPOON: 10.0-12.0' LIGHT BROWN WET SILT AND FINE SAND,
11					
12					
13					
14					
15					SPOON: 15.0-17.0' DARK BROWN WET FINE SAND AND SILT
16			100.0		
17		BOTTOM OF BORING @ 18.0'			
18					

02100144

GROUNDWATER TECHNOLOGY
Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-5

Project OSMOSE WOOD PRES. Owner _____

Location BUFFALO NY Project Number 110-001-1344

Date Drilled 6/22/89 Total Depth of Hole 18.0' Diameter _____

Surface Elevation _____ Water Level, Initial 8.0' 24-hrs. _____

Screen: Dia. 2.0" Length 15.0' Slot Size .020"

Casing: Dia. 2.0" Length 3.0' Type PVC

Drilling Company GTL Drilling Method Hollow Stem Auger

Driller MULHERN/BERNHARD Log by MEIER

Sketch Map

Notes

Depth (Feet)	Well Construction	Notes	Sample Number	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
0		ROADBOX	PID READING IN PPM		0.0-3.0' DARK BROWN-BLACK DRY FINE - MED GRAVEL AND FINE-COARSE SAND.
1		CEMENT			
2		BENTONITE			
3		WELL CASING			3.0-5.0' LIGHT BROWN-CLAY AND SILT
4		WELL SCREEN			
5			0.0		SPOON: 5.0-7.0' DARK BROWN DRY DENSE SILT AND FINE SAND.
6		SAND PACK			
7					
8					
9					
10					SPOON: 10.0-15.0' LIGHT BROWN WET FINE SAND AND SILT
11			5.0		
12					
13					
14					
15					SPOON: 15.0-17.0' LIGHT BROWN WET FINE
16			5.0		- MED SAND, SOME SILT
17		BOTTOM OF BORING @ 18.0'			
18					
19					
20					

02100144

GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-7

Project OSMOSE WOOD PRES. Owner _____

Location BUFFALO NY Project Number 110-001-1344

Date Drilled 6/23/89 Total Depth of Hole 18.0' Diameter _____

Surface Elevation _____ Water Level, Initial 7.0' 24-hrs. _____

Screen: Dia. 2.0" Length 15.0' Slot Size .020"

Casing: Dia. 2.0" Length 3.0' Type PVC

Drilling Company G.T.I. Drilling Method HOLLOW STEM AUGER

Driller MULLHERN/BERNHARD Log by MEIER

Sketch Map

Notes

Depth (Feet)	Well Construction	Notes	Sample Number	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
0		ROADBOX	PID READINGS IN PPM		0.0 - 5.0 LIGHT BROWN DRY SILT AND FINE - COARSE GRAVEL
1		CEMENT			
2		BENTONITE			
3		WELL CASING			
4					
5			0.0		SPOON: 5.0 - 7.0 DARK BROWN AND GRAY MOTTLED CLAY, TRACE OF SILT AND FINE SAND.
6		WELL SCREEN			
7					
8					
9		SAND PACK			
10			10.0		SPOON: 10.0 - 12.0' DARK BROWN WET FINE SAND AND SILT
11					
12					
13					
14					
15			2.0		SPOON: 15.0 - 17.0' DARK BROWN WET FINE SAND
16		BOTTOM			
17		OF BORING			
18		@ 18.0'			
19					
20					

02100144

GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-8

Project OSMOSE Owner

Location BUFFALO Project Number

Date Drilled 10/1/90 Total Depth of Hole 22' Diameter 10.5" OD

Surface Elevation Water Level, Initial 15.5' 24-hrs.

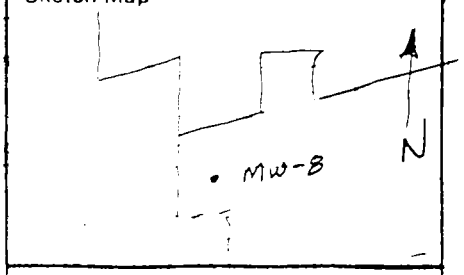
Screen: Dia. 2" Length 5' Slot Size 0.010

Casing: Dia. 2" Length 16' Type FRP

Drilling Company GTI Drilling Method HSA

Driller M. MULHERN Log by MLG

Sketch Map



Notes

Depth (Feet)	Well Construction	Notes	Sample Number / Blows / FT	Graphic Log / PID	Description/Soil Classification (Color, Texture, Structures)
0		LOCKING COPPER		8.3	SPOON 0-2': SLIGHTLY DAMP, GREY/BRN COARSE SAND SOME F-C GRAVEL. SOME CONCRETE. NO ODOR. FIRM. RECOV = 0.3'
2		BENTONITE GROUT			
		FILL		61	SPOON 2-4': SLIGHTLY DAMP, GREY-BRN MED-COARSE SAND. SOME F-C GRAVEL. SLIGHT ODOR
4		2" FRP RISER			
6				11.3	SPOON 4'-6': SLIGHTLY DAMP, DK. BRN, MED-COARSE SAND, TRACE F. SAND. SOME COARSE GRAVEL
8				0.7	SPOON 6-8': DAMP, TAN-GREY SILT, SOME CLAY W/ SILT PARTINGS. NO ODOR
10		CLAY		0.0	SPOON 8-10': DAMP, RED/BRN/GREY MOTTLED CLAY, NO ODOR. MED. STIFF
12		BENTONITE SEA		0.0	SPOON 10'-12': DAMP, RED-BRN, SLIGHTLY MOTTLED CLAY, NO ODOR. STIFF.
14		#1 WHITEHEAD SAND	25	0.0	SPOON 12'-14': DAMP, REDDISH-BRN/GREY/BRN SILT, FINE SAND AND CLAY PARTINGS. MED STIFF. NO ODOR
16		0.010 FRP SCREEN	9	0.0	
				15.5' ▽	
18		SAND	7	0.0	SPOON 14'-16': DAMP → SAT, RED-BRN/GREY BRN SILT, F. SAND & CLAY PARTINGS. LITTLE DK. BRN MED-COARSE SAND. LOOSE NO OD
			7	0.0	SPOON 16'-18': SAT. RED-BRN FINE SAND, SOME SILT. LOOSE. NO ODOR
20			5	0.0	SPOON 18'-20': SAT. BLANCHED-BROWN-ROSE, F-MED. SAND. CLAY SEAM @ 19'. LOOSE. NO ODOR
22		BENTONITE CLAY	4	0.0	SPOON 20'-22': SAT. RED-BRN F-M SAND INTO RED CLAY @ 21'.
			5	0	SPOON 22'-24': SAT. RED-BRN. CLAY INTO VARVED CLAY/F-M SAND @ 23.5'

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GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number CW-1

Project OSMOSE Owner

Location BUFFALO Project Number 5470.0203

Date Drilled 10/2/90 Total Depth of Hole 63' Diameter 10.5" OD

Surface Elevation Water Level, Initial 8.8' 24-hrs.

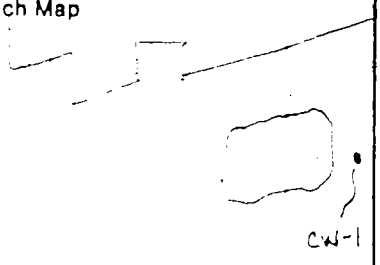
Screen: Dia. 2" Length 5' Slot Size 0.010

Casing: Dia. 2" Length 57' Type FRP

Drilling Company GTI Drilling Method HSA

Driller M. MULHERN Log by MLG

Sketch Map



Notes

Depth (Feet)	Well Construction	Notes	Sample Number / Blows/ft	Graphic Log / PID	Description/Soil Classification (Color, Texture, Structures)
0			15	0.9	SPOON 0'-2': DAMP, DK. GREY-BRN. F-M SAND, LITTLE COARSE SAND → F. GRAVEL. NO ODOR. FIRM
			20		SPOON 2'-4': A/A
2		F-M SAND	22	0.0	SPOON 4'-6': A/A INTO TAN/BRN/GREY SLIGHTLY MOTTLED CLAY @ 5' W/ SILT PARTINGS.
			14	39	SPOON 6'-8': DAMP, REDDISH-BRN CLAY W/ OCCASIONAL SILT PARTINGS. MED. STIFF
4			8	316	SPOON 8'-10': A/A INTO SAT. RED-BRN F-M SAND, TRACE SILT @ 8.8'. STRONG ODOR. LOOSE
6		CLAY	9	49	SPOON 10'-12': SAT. REDDISH-GREY-BRN, F-M SAND, TRACE SILT. STRONG ODOR. SOME SHEEN NOTED. LOOSE
8			6	11.3	SPOON 12'-14': SAT. GREY-BRN, F-M SAND, TRACE SILT. MODERATE ODOR. LOOSE
			6	35	SPOON 14'-16': SAT, GREY-BRN-RED, F-M SAND, TRACE SILT. SLIGHT ODOR. SOME SHEEN. LOOSE
10			7	44	SPOON 16'-18': A/A, W/ TRACE EMBEDDED GRAVEL (CHERT
12		F-M SAND & SILT & TRACE CLAY	7	44	SPOON 18'-20': SAT. GREY-BRN-RED, F-M SAND, SOME CLAY, 0.3' CLAY SEAM @ 18.5'. LOOSE, NO
			9	6.3	SPOON 20'-22': SAT. GREY-BRN-RED, F-M SAND, TRACE SILT. TRACE CLAY. EMBEDDED W/ TRACE FINE GRAVEL. LOOSE
14			8	3.7	SPOON 22'-24': SAT. GREY-BRN-RED, F-M SAND, SOME SILT AND CLAY. EMBEDDED W/ TRACE FINE GRAVEL. MED. STIFF
16		VARVED SOIL	8	9.8	SPOON 24'-26': A/A
18			11	0.2	SPOON 26'-28': GREY-BRN-RED, F-M SAND, TRACE SILT. TRACE CLAY. FIRM. NO ODOR.
		CLAY	9	5.3	SPOON 28'-30': SAT. GREY-BRN-RED, FINE SAND AND SILT, SOME CLAY. MED STIFF. SLIGHT ODOR
20		A/A	13	5.4	SPOON 30'-32': SAT. GREY-BRN-RED, SILT AND CLAY W/ 0.3' SEAM OF BLACK/WHITE COARSE SAND @ 30.1'. MED. STIFF
22			11	8.6	SPOON 32'-34': NO RECOVERY
24			11	7.1	SPOON 34'-36': SAT. BRN-BLACK, FINE SAND. SEAM OF F-C GRAVEL @ 35.6'.

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GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Well Number CW-1 (CONTINUED) — Drilling Log

Project OSMOSE — Owner

Location BUFFALO — Project Number 01110 5470 0203

Date Drilled 10/3/90 — Total Depth of Hole 63' — Diameter 10.5" OD

Surface Elevation — Water Level, Initial 8.8' — 24-hrs.

Screen: Dia. 2" — Length 5' — Slot Size 0.010

Casing: Dia. 2" — Length 57' — Type FRP

Drilling Company GTI — Drilling Method HSA

Driller M. MULHERN — Log by MLG

Sketch Map

Notes

Depth (Feet)	Well Construction	Notes	Sample Number / Blows	PID	Description/Soil Classification (Color, Texture, Structures)
26	TOP OF SURFACE SEAL	FINE SAND & SILT & SOME CLAY	6	0.9	SPOON: 36'-38': SAT., BLACK/BRN/RED, FINE SAND, LITTLE SILT. TRACE COARSE SAND-F. GRAVEL. V. LOOSE. POOR RECOV.
28			5	3.5	SPOON: 38'-40': SAT., BLACK/BRN, F. SAND INTO MED. SAND @ 40' THEN INTO COARSE SAND-FINE GRAVEL @ 40.5'. NO ODOR.
30		COARSE SAND	10	5.5	SPOON: 40'-42': SAT, TAN-BRN, MED SAND TO 40.2', 0.2' PARTING OF CLAY, THEN COARSE SAND-FINE GRAVEL TO 42'. LOOSE
32		SILT & CLAY	15	N/A	SPOON: 42'-44': SAT TAN-BRN, M-C SAND, LITTLE F. GRAVEL. NO ODOR. FIRM
34		NO RECOVERY	17	6.8	SPOON: 44'-46': SAT TAN-BRN, M-C SAND, TRACE F. SAND. NO ODOR. FIRM
36		F-C GRAVEL	13	4.6	SPOON 46'-48': SAT TAN-BRN, M-C SAND TRACE FINE SAND. NO ODOR. LITTLE ROCK FRAGMENTS (DOLOMITE). FIRM
		VARVED F-C SAND & SOME F GRAVEL	23	6.3	SPOON 48'-50': SAT, TAN-BRN VARVED MED. SAND-COARSE GRAVEL. FIRM. NO ODOR.
			18	12.9	SPOON 50'-52': SAT, TAN-BRN, M-C SAND, TRACE FINE GRAVEL. FIRM. NO ODOR
			15	3.1	SPOON 52'-54': SAT., TAN-BRN, F-M SAND, TRACE COARSE SAND.
54			10	11.9	SPOON 54'-56': SAT., TAN-BRN, MED. SAND.
			22	0.0	SPOON 56'-58': SAT., TAN-GREY-BRN, MED. SAND. FIRM & UNIFORM.
56			26	3.2	SPOON 58'-60': SAT, TAN-GREY-BRN A/A
58			10/1		
			20	9.8	SPOON 60'-62': SAT, TAN-GREY-BRN, F-M SAND FIRM & UNIFORM
60		#1 WHITEHEAD SAND PACK	100/0.3'	32	SPOON 62'-64': A/A TO 62.5'. THEN INTO COARSE SAND-GRL SUSPECT ON BEDROCK; FEED RATE = 2"/11 MIN 22 SEC DOWN PRESSER = 700 PSI RPM = 1600
62					CONTINUED GRINDING FOR 30 MIN. ADVANCED APPROX. 4"
63'	SLUMP	C SAND & F GRAVEL BEDROCK			

GROUNDWATER TECHNOLOGY, INC.

Well Number CW-2

Project Number

Project: OSMOSE / BUFFALO

Date Drilled 10/5/90 T.D. of Hole 6' Diameter 10.5"

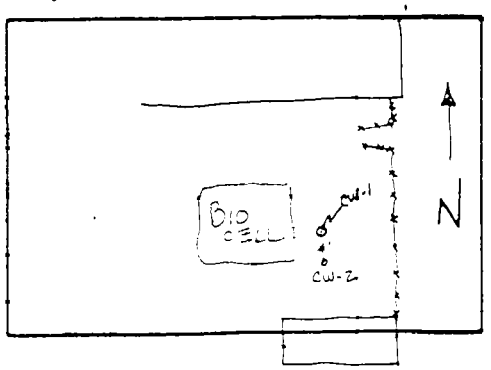
T.O.C. Water Depth, Initial NONE

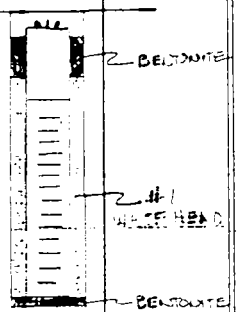
Screen: Diam. 2" Length 4' Slot Size 0.010

Casing: Diam. 2" Length 1.5 Type FRP

Drilling Co. GTI Drilling Method HSA

Driller M. MULHERN Log by MLG Sampling Method SPLIT-SPOON



Depth (Feet)	Well Construction	Notes	Sample # (Blows)	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
0		BENTONITE; #1 WATER HEAD; BENTONITE TO SEAL SPLIT SPOON	14/6"	T.I	SPOON 6'-8': DAMP, REDDISH-BRN CLAY W/ SOME GREY SILT PARTINGS MED. STIFF.
1					
2					
3					
4					BOTTOM OF BORING @ 6'
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					
21					
22					
23					

GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-9

Project OSMOSE Owner

Location BUFFALO Project Number 0110 5470 0203

Date Drilled 10/5/90 Total Depth of Hole Diameter 10.5" OD

Surface Elevation Water Level, Initial 9.5' 24-hrs.

Screen: Dia. 2" Length 20' Slot Size 0.010

Casing: Dia. 2" Length 8' Type FRP

Drilling Company GTI Drilling Method HSA

Driller M. MOLHERN Log by MLG

Sketch Map

SEE MW-9 (CONT)

Notes

Depth (Feet)	Well Construction	Notes	Sample Number / BLOWS/FT	Graphic Log / PID	Description/Soil Classification (Color, Texture, Structures)
0			6	0	SPOON: 0'-2': DAMP → MOIST DK. BRN. SILTY FINE SAND. → RED-BRN CLAY @ 1.4' EMBEDDED W/TRACE BRICK FRAGMENT.
2			7	0	SPOON: 2'-4': DAMP → MOIST DK. BRN SILTY CLAY EMBEDDED W/TRACE COARSE SAND → FINE GRAVEL.
4			15	0	SPOON 4'-6': DAMP, REDDISH-BRN CLAY, W/ GREY & BROWN SILT PARTINGS, SLIGHTLY MOTTLED, MED. STIFF. NO ODOR
6			17	0	SPOON 6'-8': SLIGHTLY DAMP, REDDISH-BRN CLAY INTO CLAYEY SILT @ 7.6'. SLIGHLY MOTTLED, OCCASSIONAL SILT PARTINGS NO ODOR. STIFF.
8					
10		▼	16	0	SPOON 8'-10': 8 → 8.5: A/A 8.5 → 9.5: MOIST → SAT. SILTY FINE SAND AND CLAY. 9.5 → 10.0': SAT. PINKISH BRN. F. SND AND SILT, LITTLE CLAY.
12			12	12.9	SPOON 10'-12': SAT. PINKISH BRN F. SND SOME SILT TRACE CLAY. MED. STIFF.
14			6	12.3	SPOON 12'-14': A/A
16			8	2.0	SPOON 14'-16': SAT. PINKISH BRN, SILTY CLAY, TRACE FINE SAND TO 15.5', THEN FINE SAND.
18			6	1.9	SPOON 16'-18': SAT. GREY BRN FINE SAND INTO CLAYEY FINE SAND @ 17.2'
			7	0	SPOON 18'-20': SAT, PINKISH-GREY-BRN, CLAYEY FINE SAND LITTLE SILT.
24			7	0.1	SPOON 20'-22': SAT, PINKISH-BRN, VARVED FINE SAND, SILT AND SILTY CLAY.
			4	0.4	SPOON 22'-24': A/A
26			5	0.6	SPOON 24'-26': SAT, PINKISH-BRN, VARVED F → M SAND SILT AND SILTY CLAY.
28			6	0.5	SPOON 26'-28': SAT, PINKISH-BRN, CLAYEY FINE SAND AND SILT, OCCASIONAL MED SAND PARTING.

GROUNDWATER TECHNOLOGY
Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-9 (CONT.)

Project OSMOSE Owner

Location BUFFALO Project Number 01110 5470 0203

Date Drilled 10/5/90 Total Depth of Hole Diameter 10.5" OD

Surface Elevation Water Level, Initial 9.5' 24-hrs.

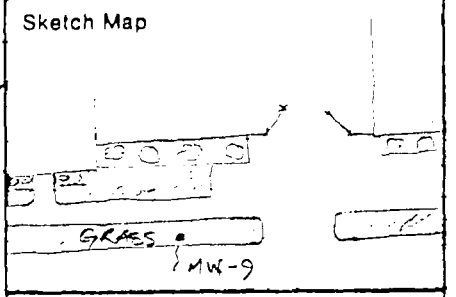
Screen: Dia. 2" Length 20' Slot Size 0.010

Casing: Dia. 2" Length 8' Type FRP

Drilling Company GTI Drilling Method HSA

Driller M. MULHERN Log by MLG

Sketch Map



Notes

Depth (Feet)	Well Construction	Notes	Sample Number	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
			6	1.3	SPOON 28'-30': PINKISH-BRN CLAYEY-FINE SAND AND SILT TO 28.5 INTO GREY-BRN F-M SAND TO 29.2 INTO GREY-BRN M-C SAND, LITTLE F GRAVEL.
			5	0.3	SPOON 30'-32': SIM., GREY BRN. UNIFORM SAND. SCREENED SOME CLEAN ZIPLOC BAGS AND HIT MAX. OF 12.3. DECIDED THAT THE LOW LEVELS MAY BE DUE TO BAGGIES. (ALL SAMPLES @ THIS LOCATION WERE PLACED DIRECTLY INTO SAMPLE JARS FOR VOA'S). DECIDED TO SEAL OFF BOTTOM OF BORING W/ BENTONITE UP TO ZONE OF CLAYEY SOILS TO PREVENT POSSIBLE CONTAM. MIGRATION INTO SAND.

GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-10

Project OSMOSE Owner

Location BUFFALO Project Number 0110 5470 0203

Date Drilled 10/16/90 Total Depth of Hole 25' Diameter 10.5"

Surface Elevation Water Level, Initial ~11' 24-hrs.

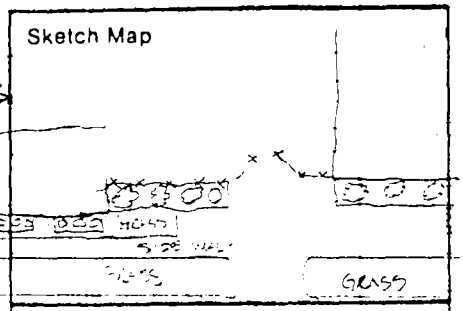
Screen: Dia. 2" Length 14' Slot Size 0.010

Casing: Dia. 2" Length 11' Type FRP MW-10

Drilling Company GTI Drilling Method HSA

Driller M. MULHERN Log by MLG

Sketch Map



Notes

Depth (Feet)	Well Construction	Notes	Sample Number / Blows/ft	Graphic Log	Description/Soil Classification (Color, Texture, Structures)
0			6	0	SPOON 0-2': MOIST, DK. BRN SILTY FINE SAND EMBEDDED W/ LITTLE FINE GRAVEL LITTLE CLAY. NO ODOR
2			10	0.4	SPOON 2-4': DAMP, MOTTLED, PINK-BRN/GREY/ORANGE CLAYEY SILT, EMBEDDED W/ TRACE COARSE SAND. NO ODOR
4			11	0.0	SPOON 4'-6': A/A BUT SOMEWHAT MORE LAMINATED SEAM OF DK. BRN CLAYEY SILT @ 5.5'. NO ODOR.
6			12	0.2	SPOON 6'-8': DAMP, DK. BROWN SEAM A/A CONTINUED TO 7.1' INTO RED-BRN CLAY, TRACE SILT W/ GREY SILT PARTINGS. NO ODOR
8			12	0.2	SPOON 8'-10': DAMP. PINK-BRN LAMINATED CLAY W/ SILT PARTINGS TO 9.3'. 9.3-10': MOIST CLAYEY FINE SAND, LITTLE SILT. NO ODOR.
10			12	0.0	SPOON 10'-12': SAT. PINK-BRN F. SAND, SOME SILT. UNIFORM NO. ODOR
12			16	0.2	SPOON 12'-14': SAT. PINK-BRN, FINE SAND, LITTLE SILT TRACE CLAY 13.8-14.0: GREY BRN FINE SAND.
14			18	0.1	SPOON 14'-16': SAT. GREY-BRN VARVED MED SAND, FINE SAND AND CLAYEY F. SAND STIFF AND NO ODOR.
16			12	0.1	SPOON 16'-18': SAT. PINK-BRN. CLAYEY FINE SAND AND SILT. OCCASIONAL F. SAND PARTINGS
18			13	0.0	SPOON 18'-20': SAT. PINK-BRN CLAYEY FINE SAND AND SILT.
20			12		SPOON 20'-22': NO RECOVERY
22			12	0.1	SPOON 22'-24': SAT. PINK-BRN CLAYEY F-M SAND & SILT. 0.1' SEAM OF COARSE SAND @ 22.6'
24			15	0.1	SPOON 24-26 SAT. PINK-BRN FINE SAND TO 26.2' SEAM OF COARSE SAND → F. GRAVEL INTO CLAYEY FINE SAND & SILT.

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GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number SB-1

Project OSMOSE/BUFFALO Owner

Location BUFFALO Project Number

Date Drilled 10/16/90 Total Depth of Hole Diameter 6 5/8" OD

Surface Elevation Water Level, Initial 8.3' 24-hrs.

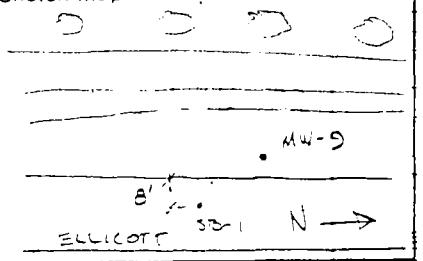
Screen: Dia. N/A Length N/A Slot Size N/A

Casing: Dia. N/A Length Type

Drilling Company EARTH DIMENSION Drilling Method HSA

Driller DALE GRAMZA Log by MLG

Sketch Map



Notes

Depth (Feet)	Well Construction	Notes	Sample Number & Type	Graphic Log PID	Description/Soil Classification (Color, Texture, Structures)
0	N/A		1.2.5.8	5.9	SPOON 0-2': SLIGHTLY DAMP, DK BRN-BLACK SILTY FINE SAND, TRACE CLAY (SMELLED OF ASPHALT) INTO RED/BRN/GREY MOTTLED CLAY. MED. STIFF @ 1.5'
2			5.8.13.15	4.0	SPOON 2-4': SLIGHTLY DAMP, RED/BRN/GREY MOTTLE CLAY. HARD, NO ODOR.
4			6.7.11.14	4.7	SPOON 4-6': DAMP A/A INTO RED CLAY W/ BLACK VEINS, OCCASSIONAL SILT → FINE SAND PARTINGS.
6			4.8.8.11	2.5	SPOON 6'-8': DAMP RED-BRN CLAY, UNIFORM AND STIFF.
8		▽ 8.3			
10			2.4.5.4	1.9	SPOON 8'-10': SAT. RED-BRN. FINE SAND. UNIFORM NO ODOR.
12			2.4.7.7	1.1	SPOON 10'-12': A/A
14			5.7.8.7	0.0	SPOON 12'-14': A/A, LITTLE SILT.
16			6.6.3.3	0.0	SPOON 14'-16': 14.0'-14.5': SAT. RED-BRN, CLAYEY FINE SAND, LITTLE SILT. UNIFORM 14.5'-15.0': MOIST, RED-BRN, FINE SAND, TRACE COARSE GRAVEL (ROUNDED). 15.0'-16.0': SAT, RED-BRN, CLAYEY FINE SAND.
18					
20					

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GROUNDWATER TECHNOLOGY

Division of Oil Recovery Systems, Inc.

Drilling Log

Well Number MW-11

Project OSMOSE / BUFFALO Owner

Location BUFFALO NY Project Number 0110 5470 -0203

Date Drilled 10/16/90 Total Depth of Hole Diameter 10.5 OD

Surface Elevation Water Level, Initial 24-hrs.

Screen: Dia. 2" Length Slot Size 0.010

Casing: Dia. 2" Length Type FRP

Drilling Company EARTH DIMENSIONS Drilling Method HSA

Driller DALE GRAMZA Log by MLG

Sketch Map

Notes

Depth (Feet)	Well Construction	Notes	Sample Number / Blows	Graphic Log / PID	Description/Soil Classification (Color, Texture, Structures)
0			3-4-4-4	0	SPOON 0'-2': DAMP, DK. BRN FINE SAND AND SILT WITH 0.3' SEAM OF TAN F. SAND TRACE SILT @ 1.5'.
2			3-4-6-7	0	SPOON 2'-4': 2.0-2.7 DAMP DK BRN SILTY FINE SAND, LITTLE CLAY EMBEDDED W/TRACE COARSE SAND. 2.7-4.0 DAMP, RED-BRN/ORANGE/GREY MOTTLED CLAY, LITTLE FINE SAND, MED. STIFF, NO ODOR.
4		2 GROUT			
6		CLAY	6-7-9-11	0	SPOON 4'-6': DAMP, RED-BRN/ORANGE/GREY SLIGHTLY MOTTLED CLAYEY SILT. TRACE F. SAND.
8					
10		9.5'	5-6-8-14	0	SPOON 6'-8': DAMP, RED-BRN CLAY, TRACE SILT. OCCASIONAL SILT PARTINGS NO ODOR. STIFF.
12			6-7-10-12	0	SPOON 8'-10': 8-8.3': DAMP RED-BRN CLAYEY SILT LAMINATED W/GREY SILTY CLAY PARTINGS. 8.3-9.5': DAMP RED-BRN/GREY/ORANGE SLIGHTLY MOTTLED CLAY. 9.5-10.0: SAT. RED-BRN FINE SAND. UNIFORM.
14		FINE SAND			
16					
18		BENT. SEAL	3-5-7-7	0	SPOON 10'-12': SAT. RED-BRN UNIFORM FINE SAND, SOME SILT, TRACE CLAY. FIRM. NO ODOR
20		#1 SAND	3-6-7-9	0	SPOON 12'-14': A/A.
22			2-2-2-4		SPOON 14'-16': SAT. RED-BRN CLAYEY F. SAND
			W-1-2-3	0	SPOON 16'-18': 16'-17.0': SAT. RED-BRN, CLAYEY FINE SAND, TRACE F. GRAVEL FRAGMEN 17'-18': SAT. RED-BRN CLAY.
			1-1-2	0	SPOON 18'-20': SAT. RED BRN. CLAYEY F. SAND.

02100144

E

APPENDIX E

WELL DEVELOPMENT DETAILS

	MW-8	MW-9	MW-10	MW-11	CW-1
WATER REMOVED (gallons)	30	87	35	22	55
TURBIDITY PRIOR TO DEVELOPMENT (NTUs)	29	5.6	4.0	2.7	18
TURBIDITY AFTER DEVELOPMENT (NTUs)	>200	>200	>200	>200	>200

F

GROUNDWATER
TECHNOLOGY, INC.

CREOSOTE COMPOUNDS

	Formula	Boiling Point	Concentration Range
Coumarone	C_8H_6O	174	A
p-Cymene	$C_{10}H_{14}$	177	A
Indene	C_9H_8	182	A
Phenol	C_6H_6O	181	A
O-Cresol	C_7H_8O	190	A
Benzonitrile	C_7H_5N	191	A
m-Cresol	C_7H_8O	202	A
Naphthalene	$C_{10}H_8$	218	D
Thionaphthene	C_8H_6S	222	A
Quinoline	C_9H_7N	243	A
2-Methylnaphthalene	$C_{11}H_{10}$	241	B
Isoquinoline	C_9H_7N	238	A
1-Methylnaphthalene	$C_{11}H_{10}$	245	A
4-Indanol	$C_9H_{10}O$	245	B
2-Methylquinoline	$C_{10}H_9N$	247	A
Indole	C_8H_7N	252	A
Diphenyl	$C_{12}H_{10}$	255	A
1, 6-Dimethylnaphthalene	$C_{12}H_{12}$	262	A
2, 3-Dimethylnaphthalene	$C_{12}H_{12}$	266	A
Acenaphthene	$C_{12}H_{10}$	281	D
Dibenzofuran	$C_{12}H_{10}O$	287	D
Fluorene	$C_{13}H_{10}$	299	D

	Formula	Boiling Point	Concentration Range
1-Naphthonitrile	$C_{11}H_7N$	297	A
3-Methyldiphenylene	$C_{13}H_{10}O$	298	B
2-Naphthonitrile	$C_{11}H_7N$	304	A
9, 10-Dihydroanthracene	$C_{14}H_{10}$	305	B
2-Methylfluorene	$C_{14}H_{12}$	318	B
Diphenylene Sulfide	$C_{12}H_8S$	332	B
Phenanthrene	$C_{14}H_{10}$	340	D
Anthracene	$C_{14}H_{10}$	342	C
Acridene	$C_{13}H_9N$	346	A
3-Methylphenanthrene	$C_{13}H_{12}$	350	B
Carbazole	$C_{12}H_9N$	352	B
4, 5-Methylenephenanthrene	$C_{15}H_{10}$	353	B
2-Methylanthracene	$C_{15}H_{12}$	360	A
9-Methylanthracene	$C_{15}H_{12}$	361	B
2-Methylcarbazole	$C_{13}H_{11}N$	363	B
Fluoranthene	$C_{16}H_{10}$	382	D
1, 2-Benzodiphenylene	$C_{16}H_{10}O$	395	B
Pyrene	$C_{16}H_{10}$	393	B
Benzofluorene	$C_{17}H_{12}$	413	B
Chrysene	$C_{18}H_{12}$	448	B
Unidentified Compounds in Distillate			D

A = Compounds having a concentration less than 0.5%
B = Compounds having a concentration greater than 0.5% and less than 3.0%
C = Compounds having a concentration greater than 3.0% and less than 5.0%
D = Compounds having a concentration greater than 5.0%

G

GROUNDWATER
TECHNOLOGY, INC.

OSMOSE WOOD PRESERVING, INC.
980 ELLICOTT STREET • BUFFALO, NY 14209-2398
(716) 882-5905 FAX 716 882-5139

January 31, 1991

Mr. Bruce Ahrens
Groundwater Technology, Inc.
12 Walker Way
Albany, NY 12205

Dear Bruce:

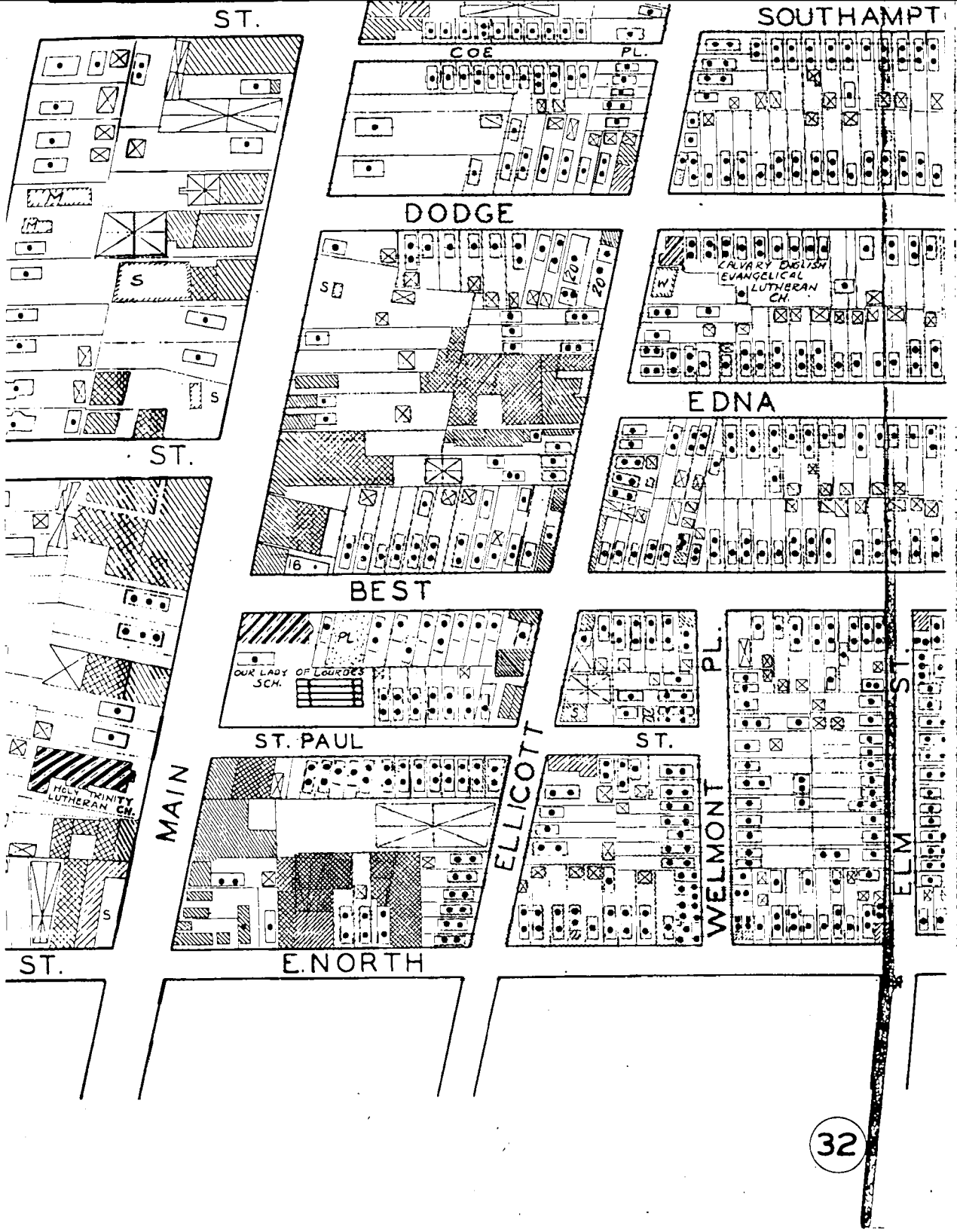
We have discussed the prior use of the area of our property at 980 Ellicott Street in Buffalo, New York. Please note that the area which now has been penetrated by MW8 was formerly a coal bin. It is situated on the south wall of our boiler room. When it was in operation (prior to about 1960) this coal storage and feed area was about 2-4 feet below the present grade and was covered by a shed type roof which attached to the building's wall. The structure was about 15-20 feet square and had a dirt floor. There was a screw conveyor in the pit to transport coal into the boiler room for stoking.

We have checked our corporate files but cannot find any pictorial record of the coal bin and feeding equipment. Also the area adjacent to MW11 is the area where we photographed the asphalt piles left by National Fuel Gas during their street excavations during the summer of 1990. To our knowledge these piles still exist to the present.

Regards,

Michael E. Rider
Plant Manager

MER:c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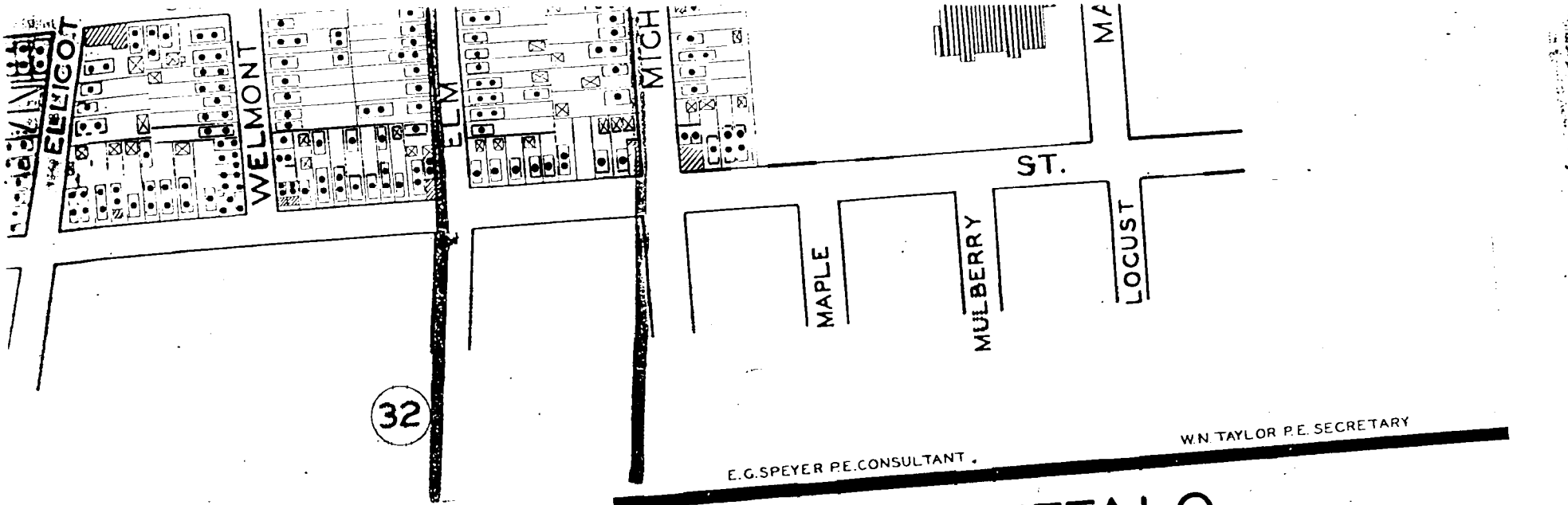


E.G. SPEYER P.E. CONSULTANT.

W.N. TAYLOR P.E. SECRETARY

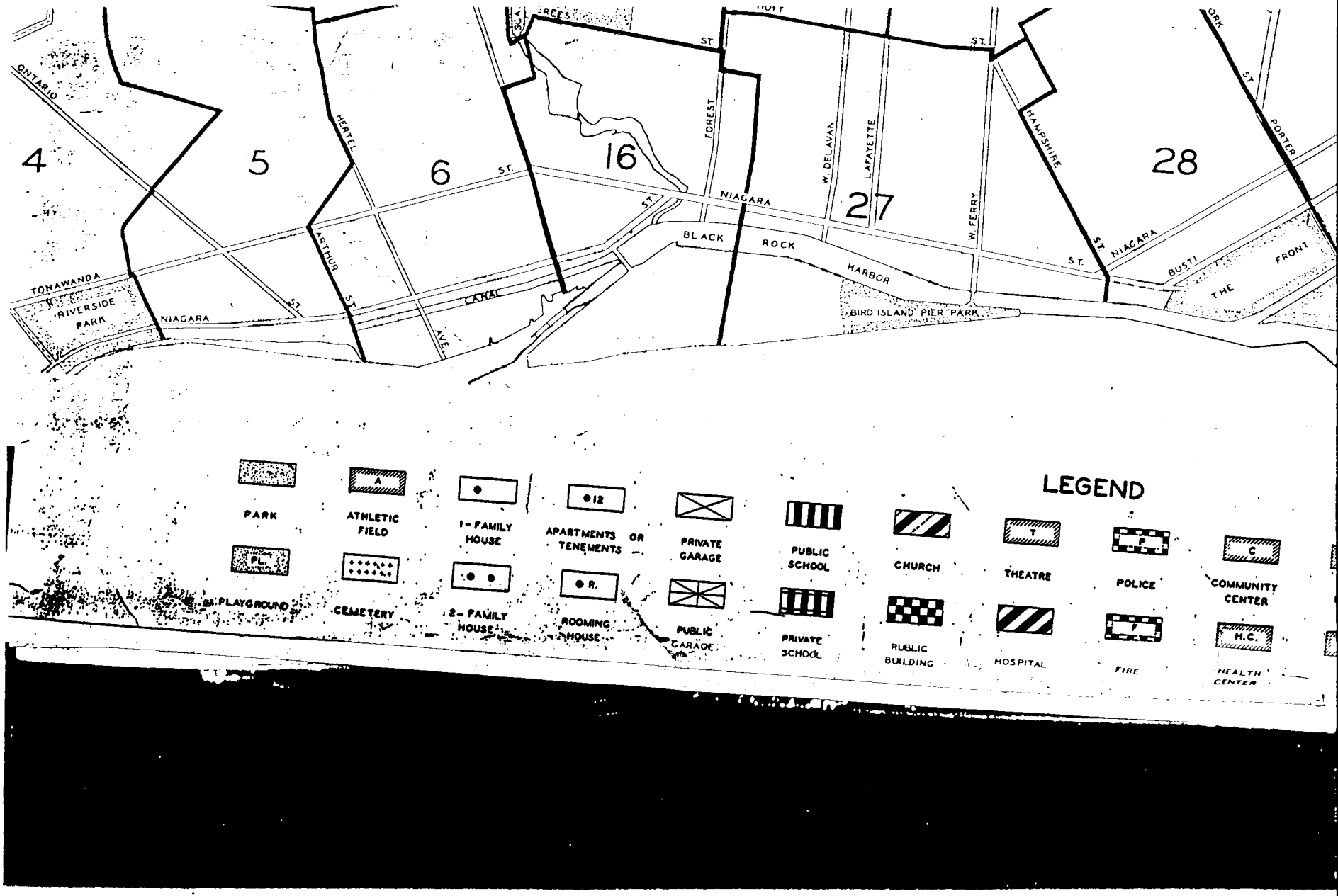
BUFFALO
BASE ZONE MAP No. 26
PREPARED BY
BUFFALO PLANNING BOARD

SCALE 1 IN = 200 FT.

SEPTEMBER 1933

SCALE 1 IN. = 200 FT.

MARCH 1934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NIAGARA
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BLACK ROCK HARBOR
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TONAWANDA
RIVERSIDE PARK
ARTHUR
CANAL
AVE.
BIRD ISLAND PIER PARK
LEGEND
PARK
ATHLETIC FIELD
1-FAMILY HOUSE
APARTMENTS OR TENEMENTS
PRIVATE GARAGE
PUBLIC SCHOOL
CHURCH
THEATRE
POLICE
COMMUNITY CENTER
PLAYGROUND
CEMETERY
2-FAMILY HOUSE
ROOMING HOUSE
PUBLIC GARAGE
PRIVATE SCHOOL
PUBLIC BUILDING
HOSPITAL
FIRE
HEALTH CENTER

H

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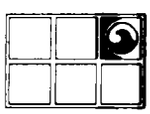

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