UOP, Incorporated

Des Plaines, IL

Risk Assessment Report
Areas 1, 1A, 2, 4 & 5
UOP Site,
East Rutherford, NJ

ENSR Consulting and Engineering

(Formerly ERT)

October 1988

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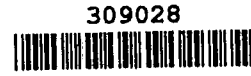

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October 13, 1988

Mr. James Schnitzer
Case Manager
NJDEP Bureau of Federal
Case Management
401 East State Street
5th Floor, West Wing
Trenton, New Jersey 08625

RE: Report Entitled: "Risk Assessment Report, Areas 1, 1A, 2, 4 and 5, UOP Site, East Rutherford, NJ, Revision 2"

Dear Mr. Schnitzer:

Enclosed are seven copies of Revision 2 of the Human Health Risk Assessment Report for the UOP Site, East Rutherford, New Jersey. This document includes significant changes to Revision 1 generated by:

- correspondence from NJDEP to UOP, Inc. dated March 10, 1988 and August 22, 1988,
- correspondence from ERT to NJDEP dated June 6, 1988, and
- a meeting attended by representatives of NJDEP, UOP, Inc. and ERT on April 6, 1988.

The correspondence cited above contain a set of numbered comments. In order to facilitate NJDEP's quick review, Table 1 refers to the location in the report that addresses each comment. As an additional review aid, the report text is marked with author annotations (vertical line in the right hand margin). Because they are entirely revised, the tables and appendices are not marked with author annotations.

Please note that the data reduction method has been revised. Previously, soil concentrations less than 1 mg/kg and groundwater concentrations less than water quality criteria were deleted from the average concentration computation. A review of how the exposure scenarios are formulated and the

recognition that the previous averaging method produces an average concentration that is not realistic and conservatively high, led us to recalculate the averages using a more realistic and currently-acceptable method. The technique used, averages all the data for a given medium and incorporates a value of zero for non-detected compounds.

Please also note that rather than using the overly conservative assumption that all chromium was hexavalent, a more realistic hexavalent-trivalent ratio was used in the assessment (refer to Section 2.7 and Appendix D of the report).

If you have any questions regarding this submittal please contact Lawrence Geyer, UOP at 312-391-2675.

Sincerely,

ENSR

Michael C. Worthy, P.E.
Project Manager

William A. Duvel, Jr. Ph.D., P.E.
Vice President

MCW/WAD/lw

attachment

enclosure

TABLE 1

Location in Report of Response to NJDEP Comment

General Comment/Where Addressed

1. Section 1.4

2. Appendix B

3. To be addressed in the forthcoming ecological risk assessment

Specific Comment/Where Addressed

1. Table 1-1

2. Tables 1-2 and 1-3

3. Table 1-2

4. Tables 1-2, 1-3, and 1-4

5. Tables 1-2 and 1-3

6a. Section 1.4

6b. Table 1-4

6c. Section 8 Tables

6d. Table 2-1

7. Section 2

8a. To be addressed in the forthcoming ecological risk assessment

8b. Table 3-2

9a. Section 4.1.2

9b. Section 4.1.2

10a. Sections 5.1.2 and 7.1.1

10b. Section 5.1.1

10c. Table 5-1 and Appendix C

10d. Revision 1 of the Risk Assessment analyzed the potential risk due to discharge of contaminants from ground water into surface water. The risks were computed this way because only Areas 1, 1A, 2 and 5

TABLE 1 (cont.)

of the site were addressed. In the current revision (2), the stream channels (surface water and sediment) are included in the assessment. Therefore, the risks due to contamination in surface water are evaluated as a whole. The human health risks calculated for surface water are very low (see Appendix B). If the risks had been significant, then the sources of the contamination (e.g., sediments, ground water and waste water lagoons) would have been delineated and the contribution of each estimated.

11. Section 6

12a. Section 7.1.1

12b. Section 7, Appendix C

12c. Section 4.1.2

12d. Section 7.1.3 and 7.2.3

12e. Section 7.1.2

12f. Section 7.2.1

13a. Section 8 Tables

13b. Tables 11-1 and 11-2

13c. Tables 8-4, 8-5, and 8-6

14. Section 9

15a. Section 11, p. 11-1

15b. Section 11, pp. 11-2 and 11-3

15c. Tables 11-1, 11-2 and 11-3

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PREFACE

The following report presents a baseline health risk evaluation of the upland portions (Areas 1, 1A, 2, and 5) of the UOP Site in East Rutherford, New Jersey. A partial baseline health risk assessment of the tidal stream channels (Area 4) is included in Appendix B. Food chain exposures from the stream channels will be evaluated in a forthcoming environmental risk assessment. This report has been prepared by ENSR Consulting and Engineering (formerly ERT) to support the forthcoming UOP Site Feasibility Study (FS).

The risk evaluation is based on field observations and analytical data as presented in the Phase II Investigation (May 1985), the Remedial Investigation (RI) Report (Phase III, May 1988) by Geraghty & Miller, Inc. and the report entitled: "Conceptual Plan for the Remediation of Ackerman's Creek Sediments, February 1988" by ERT, Inc., (also a Phase III Investigation). The methods for this risk evaluation follow the guidance provided in the Superfund Public Health Evaluation Manual, (SPHEM; EPA 1986) and it is formatted to comply with draft guidance (November 1986) from the New Jersey Department of Environmental Protection on health assessments of hazardous waste sites.

The baseline evaluation is a health risk assessment of the current condition of the UOP Site and, as such, represents a health risk evaluation of the "no-action alternative." The baseline evaluation will indicate if a remedial action is needed at the UOP Site to provide an adequate level of public health protection for present and probable future use of the site.

The SPHEM suggests that health-based criteria can be useful in deriving acceptable residual levels of constituents in soil (design goals). The baseline assessment will provide the framework for developing design goals for the UOP Site, if they are required. The design goals may then be used for developing and screening remedial alternatives during the FS process.

In this report, design goals will be developed which, if achieved by site remediation, would provide public health protection at the potential exposure points at the site. Specifically, design goals will be set which ensure exposure below toxic levels to non-carcinogenic constituents and provide for low risk from carcinogenic substances. These values will provide objective, health-based criteria for developing and screening remedial alternatives. In compliance with the Guidance on Feasibility Studies under CERCLA (EPA, 1985), a range of design goals for carcinogens associated with cancer risk of 1 chance in 10,000 (10^{-4}) to 1 chance in 10,000,000 (10^{-7}) will be provided.

The Risk Assessment is organized as follows. Section 1 describes the process for selecting a set of "Indicator Compounds" that are representative of all the compounds found at the site. The Indicator Compounds are used solely for the risk evaluations. Section 2 describes the carcinogenic and non-carcinogenic toxic characteristics for each of the Indicator Compounds. Section 3 describes the potential pathways of contaminants through the air, ground water, surface water, soils and the sediments to human populations. Section 4 describes what type of people (i.e., children, adults, construction workers) are expected to be exposed based on current and projected land uses. Section 5 develops the concentration of contaminants available for human contact through the air, water and soil. Section 6 describes these concentrations relative to relevant and applicable standards. Section 7 develops the dose of contaminants received by the exposed populations. Section 8 evaluates the carcinogenic and non-carcinogenic risks associated with the doses received. Section 9 summarizes the risk factors developed in Section 8. Section 10 reviews the assumptions used in developing the risk scenarios and the inherent uncertainty in the various steps of the risk analysis. Section 11 summarizes the risk assessment and presents the major findings.

1. SELECTION OF INDICATOR COMPOUNDS

1.1 General

A set of indicator compounds which characterize the potential public health threat at the UOP site were identified. The indicator chemicals were selected from the analytical data compiled during the Phase II and III Remedial Investigations at the site. The chief criteria for selection of the compounds were the relative concentrations of the substances in the various media at the UOP site and their relative toxicity. Following the initial screening, the exposure risk of the compounds was evaluated on the basis of the relative frequency of detection of the candidate compounds. The selection of indicator compounds generally followed the steps outlined in the EPA Superfund Public Health Evaluation Manual (SPHEM) (EPA, 1986).

1.2 Identification of Contaminants

All chemical contaminants detected in ground water and soil samples were considered in the selection of indicator chemicals. Indicator chemicals for Area 4 were determined separately based on sediment and surface water concentrations (Appendix B). The highest concentration and a representative mean were used in the calculations described subsequently. Although the RI apportioned the analytical data sets according to the four sub areas (1, 1A, 2, & 5) of the site in which they were detected, during the selection of indicator chemicals, the four areas were considered as a single site. Thus, the site was characterized as a whole, rather than as four distinct areas. Analytical data points were grouped according to the environmental media in which they occurred: ground water, surface soil and sub-surface soil. This grouping reflects the distinctions in the probable routes of exposure which could be expected to result from a "no-action" site remediation

scenario. It also facilitates the indicator scoring, as the toxicity constants presented in the SPHEM are medium-specific.

Within each medium, an arithmetic mean of analytical data points was calculated. The arithmetic mean was calculated using all samples. The concentration of chemicals in non-detect samples was assumed to be zero. The frequency of detection for each contaminant was recorded separately as a ratio of: the number of samples in which the compound was detected to the total number of samples analyzed. For each compound in each medium, the maximum concentration detected was also recorded.

1.3 Toxicity Ranking of Indicator Chemicals

Following the procedure outlined in the SPHEM, an indicator score for each chemical was calculated from the following algorithm:

$$IS_{ij} = (C_{ij} \circ T_{ij})$$

where IS_{ij} = indicator score for chemical i in medium j. (unitless)

C_{ij} = concentration of chemical i in medium j. The units are:

	Medium	Units
1.	Groundwater	mg/L
2.	Surface Soils	mg/kg
3.	Sub-surface Soils	mg/kg

T_{ij} = a toxicity constant for chemical i in medium j (units are the inverse of above concentration units).

The toxicity constants, as listed in the SPHEM, are medium specific, calculating the relative toxicity of a given compound in water and soil. For each medium there are two distinct constants: one for carcinogenic toxicity and one for non-carcinogenic toxicity. The two sets are not interchangeable, and thus the indicator scores for carcinogens and non-carcinogens cannot be validly compared.

Toxicity constants for non-carcinogens (Tn) are derived from the minimum effective dose (MED) for chronic effects, a severity-of-effect factor, and standard factors for body weight and oral or inhalation intake (e.g., 70 kg body weight, 2 L/day of drinking water, 20 m^3/day of air). Toxicity constants for potential carcinogens (Tc) are based on the dose at which a 10 percent incremental carcinogenic response is observed (ED_{10}) and the same standard intake and body weight factors. The intake factor for soil toxicity constants is based on an assumption of 100 mg of soil consumed per day for 2- to 6-year-olds (EPA, 1984a). Toxicity constants for constituents at the UOP site are given in Table 1-1. Worksheets indicating maximum and representative concentrations of compounds and the resulting maximum and representative IS scores are given in Tables 1-2, 1-3, and 1-4.

Although the SPHEM suggests calculating an overall indicator score ($IS = \sum_{j=1}^{n} C_{ij} \cdot T_{ij}$), ENSR chose to evaluate individual media indicator scores to select indicator chemicals. This was done because the UOP Site is different from many sites in having relatively different constituents in the different media, and disparate relative importance of each media for various exposure scenarios. Thus, separate scores are more reflective of the actual health impact potential of the site than is a combined score.

TABLE 1-1

TOXICITY CONSTANTS FOR SELECTED COMPOUNDS PRESENT AT THE UOP SITE

Compound	Carcinogenic Classification	Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/mg)	Non-Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/kg)
Acenapthene		a	a	a	a	a	a
Acenaphthylene		a	a	a	a	a	a
Acetone		a	a	a	a	a	a
Alkane		b	b	b	b	b	b
Anthracene		a	a	a	a	a	a
Antimony		a	a	a	4.35E+00	2.17E-04	2.29E+02
Arsenic	A	4.07E+00	2.03E-04	4.07E+01	1.80E+01	9.00E-04	1.80E+02
Benzene	A	7.71E-03	3.86E-07	7.71E-02	1.17E-01	5.85E-06	1.18E+02
Benzene, acetic acid		b	b	b	b	b	b
Benzene, acetonitrile		b	b	b	b	b	b
Benzene, -1-chlor-2-methyl		b	b	b	b	b	b
Benzene,1-(1,1 dimethylethyl)		b	b	b	b	b	b
Benzene, 1-1' methylene bis		b	b	b	b	b	b
Benzene, (methyl sulfonyl)		b	b	b	b	b	b
Benzene 1,1-(oxy-bis(methylene))		b	b	b	b	b	b
Benzene, 1,-sulfonyl bis		b	b	b	b	b	b
Benzo(a) anthracene	B2	5.81E-01	2.91E-05	5.81E+00	a	a	a
Benzo(b) fluoranthene	B2	NA	NA	NA	a	a	a
Benzo(k) fluoranthene	D	NA	NA	NA	a	a	a
Benzo (g,h,i) perylene		a	a	a	a	a	a
Benzo(a) pyrene	B2	4.55E+00	2.28E-04	4.55E+01	2.67E+01	1.33E-03	1.91E+01
Benzoic acid		b	b	b	b	b	b
Benzo acid 4-chloro		b	b	b	b	b	b
Benzoic acid, 4(-1,1-dimethylethyl)		b	b	b	b	b	b
Benzoic acid, 3-methyl		b	b	b	b	b	b
Benzyl alcohol		b	b	b	b	b	b
Beryllium	B1	NA	NA	2.28E+01	a	a	1.45E+04
Bicyclo-heptanone-trimethyl		b	b	b	b	b	b
Bis(2-chloroethyl)ether	B2	1.74E-01	8.71E-06	1.74E+00	a	a	a
Bis(2-ethylhexyl)phthalate	B2	5.71E-04	2.86E-08	5.71E-03	a	a	a
Bromodichloromethane		a	a	a	a	a	a
4 Bromophenyl phenyl ether		b	b	b	b	b	b
2-Butanone		a	a	a	7.75E-03	3.85E-07	7.75E-02
Butyl benzyl phthalate		b	b	b	b	b	b

TABLE 1-1 (CONTINUED)

Compound	Carcinogenic Classification	Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/mg)	Non-Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/kg)
Cadmium	B1	NA	NA	1.65E+01	4.45E+00	2.23E-04	3.59E+02
Carbon disulfide		a	a	a	4.24E-01	2.12E-05	4.24E+00
4-Chloroaniline		b	b	b	b	b	b
Chlorobenzene		a	a	a	1.43E-01	7.14E-06	2.79E-01
Chloroform	B2	5.63E-02	2.81E-06	5.63E-01	a	a	a
2-Chlorophenol		a	a	a	a	a	a
4-Chlorophenyl phenyl ether		b	b	b	b	b	b
Chromium	A	NA	NA	1.11E+02	NA	NA	2.50E+01
Chrysene	B2	NA	NA	NA	a	a	a
Copper		a	a	a	7.14E-01	3.57E-05	7.14E+00
Cyanide		a	a	a	a	a	a
Cyclohexane 3,3,5-trimethyl		b	b	b	b	b	b
Dibenzo(a,h)anthracene	B2	7.14E+00	3.57E-04	7.14E+01	a	a	a
Dibenzofuran		b	b	b	b	b	b
Dibromochloromethane		a	a	a	1.82E+00	9.09E-05	1.82E+01
1,2 Dichlorobenzene		a	a	a	5.19E-02	2.60E-06	3.61E-01
1,3 Dichlorobenzene		a	a	a	5.19E-02	2.60E-06	3.61E-01
1,4 Dichlorobenzene		a	a	a	5.19E-02	2.60E-06	3.61E-01
1,1, Dichloroethane		a	a	a	2.58E-02	1.29E-06	2.58E-01
1,2 Dichloroethane	B2	3.71E-03	1.86E-07	3.71E-02	1.76E-02	8.80E-07	1.10E+00
1,1 Dichloroethylene	C	2.48E-01	1.24E-05	2.48E+00	3.71E-01	1.86E-05	5.65E+00
1,2 trans Dichloroethylene		a	a	a	5.29E-02	2.65E-06	5.29E-01
1,2 Dichloropropane		a	a	a	1.00E-01	5.00E-06	1.00E+00
Di-n-butyl phthalate		a	a	a	3.81E-02	1.90E-06	3.81E-01
Di-n-octyl phthalate		b	b	b	b	b	b
Diethylphthalate		a	a	a	2.67E-04	1.34E-08	2.67E-03
1,2 Diphenylhydrazine	B2	1.31E-01	6.53E-06	1.31E+00	3.34E-01	1.67E-05	3.34E+00
Ethane 1,2-bis(2-chloroethoxy)		b	b	b	b	b	b
Ethylbenzene		a	a	a	1.10E-02	5.52E-07	1.10E-01
Fluoranthene		a	a	a	a	a	a
Fluorene		a	a	a	a	a	a
Furan,tetrahydrotetramethyl		b	b	b	b	b	b
Hexachlorobenzene	B2	3.36E-01	1.68E-05	3.36E+00	4.00E-01	2.00E-05	4.00E+00
Hexachlorobutadiene	C	1.69E-02	8.43E-07	1.69E-01	a	a	a
Indeno(1,2,3-c,d)pyrene	C	NA	NA	NA	a	a	a
Iron		a	a	a	a	a	a
Isophorone		a	a	a	a	a	a
Lead		a	a	a	8.93E-01	4.46E-05	8.93E+00

TABLE 1-1 (CONTINUED)

Compound	Carcinogenic Classification	Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/mg)	Non-Carcinogenic Toxicity Constant Water(l/mg)	Soil(kg/mg)	Air(m3/kg)
Manganese		a	a	a	a	a	a
Mercury (inorganic)		a	a	a	1.84E+01	9.21E-04	1.86E+02
Methanone, diphenyl		b	b	b	b	b	b
4-Methyl 2-pentanone		a	a	a	a	a	a
2-Methyl Phenol		b	b	b	b	b	b
4-Methyl Phenol		b	b	b	b	b	b
2-Methylnaphthalene		b	b	b	b	b	b
Methylene chloride	B2	NA	NA	NA	9.20E-04	4.60E-08	9.20E-03
Naphthalene		a	a	a	a	a	a
Nickel	A	NA	NA	2.29E+00	4.26E+00	2.13E-04	1.57E+02
N-nitrosodiphenylamine	B2	a	a	a	a	a	a
Total Carcinogenic PAHs		a	a	a	a	a	a
Pentachlorophenol		b	b	b	b	b	b
Total PCBs	B2	1.44E+00	7.21E-05	1.44E+01	a	a	a
PCB-Aroclor 1248	B2	b	b	b	b	b	b
PCB-Aroclor 1254	B2	b	b	b	b	b	b
Phenanthrene	D	a	a	a	a	a	a
Phenol		a	a	a	1.00E-01	5.02E-06	2.49E+00
Phenol 4(1,1-dimethylethyl)		b	b	b	b	b	b
Phenol 2,6,bis(1,1-di-methylethyl)		b	b	b	b	b	b
Phenol 2,4,bis(1-methylethyl)		b	b	b	b	b	b
Pyrene		a	a	a	a	a	a
Silver		a	a	a	2.00E+01	1.00E-03	2.00E+02
Sulfur		b	b	b	b	b	b
1,1,2,2 Tetrachloroethane	C	4.74E-02	2.37E-06	4.74E-01	4.55E-01	2.27E-05	4.55E+00
Tetrachloroethylene	B2	8.29E-03	4.14E-07	8.29E-02	9.62E-03	4.81E-07	2.75E-02
Thallium		a	a	a	a	a	a
1,2,4 Trichlorobenzene		a	a	a	2.14E-01	1.07E-05	1.52E+00
1,1,2-Trichloroethane	C	1.03E-02	5.14E-07	1.03E-01	a	a	a
Trichloroethylene	B2	2.00E-03	1.00E-07	2.00E-02	1.05E+00	5.26E-05	2.96E+01
Trichlorofluoromethane		b	b	b	b	b	b
Toluene		a	a	a	5.20E-03	2.60E-07	5.20E-02
Total Xylenes		a	a	a	a	a	a
Vinyl Chloride	A	4.29E-03	2.14E-07	4.29E-02	8.77E-02	4.39E-06	8.77E-01
Zinc		a	a	a	1.07E-01	5.33E-06	1.07E+00

a. Compound is included in the PHRED database (as of February 1988) but no values are reported for the parameter.
b. Compound has not been added to the PHRED database (as of February 1988).

TABLE 1-2

HAZARD IDENTIFICATION OF CONSTITUENTS BASED ON GROUNDWATER DATA

Compound	Carcinogenic Classification	Groundwater Concentrations* Maximum mg/l	Representative mg/l	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Acenapthene		0.01	0.00	1/42								
Acenaphthylene				0/42								
Acetone		0.17	0.01	6/42								
Alkane		0.19	0.02	1/9								
Anthracene		0.00	0.00	2/42								
Antimony		0.08	0.02	14/15					3.48E-01	9	9.92E-02	5
Arsenic	A	0.11	0.01	25/42	4.48E-01	2	4.19E-02	2	1.98E+00	4	1.85E-01	3
Benzene	A	44.00	3.53	25/42	3.39E-01	3	2.72E-02	3	5.15E+00	2	4.13E-01	2
Benzene, acetic acid		0.49	0.05	1/9								
Benzene, acetonitrile		3.20	0.36	1/9								
Benzene, -1-chlor-2-methyl		0.07	0.01	1/9								
Benzene,1-(1,1 dimethylethyl)		15.00	1.84	2/9								
Benzene, 1-1' methylene bis		0.39	0.04	1/9								
Benzene, (methyl sulfonyl)		0.05	0.01	1/9								
Benzene 1,1-(oxy-bis(methylene))		1.90	0.21	1/9								
Benzene, 1,-sulfonyl bis		0.35	0.08	3/9								
Benzo(a) anthracene	B2			0/42								
Benzo(b) fluoranthene	B2			0/5								
Benzo(k) fluoranthene	D			0/42								
Benzo (g,h,i) perylene				0/42								
Benzo(a) pyrene	B2			0/42								
Benzoic acid		8.70	0.51	2/17								
Benzo acid 4-chloro		0.23	0.03	1/9								
Benzoic acid, 4(-1,1-dimethylethyl)		0.17	0.03	3/9								
Benzoic acid, 3-methyl		0.43	0.05	1/9								
Benzyl alcohol		0.12	0.01	3/17								
Beryllium	B1	0.00	0.00	7/15								
Bicyclo-heptanone-trimethyl		0.07	0.01	1/9								
Bis(2-chloroethyl)ether	B2	0.13	0.00	5/42	2.26E-02	7	7.66E-04	7				
Bis(2-ethylhexyl)phthalate	B2	0.20	0.01	11/42	1.14E-04	14	7.14E-06	13				
Bromodichloromethane				0/5								
4 Bromophenyl phenyl ether				0/42								
2-Butanone				0/42								
Butyl benzyl phthalate				0/42								

TABLE 1-2 (CONTINUED)

Compound	Carcinogenic Classification	Groundwater Concentrations* Maximum mg/l	Representative mg/l	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Cadmium	B1	0.01	0.00	16/42					3.56E-02	19	6.23E-03	16
Carbon disulfide				0/42								
4-Chloroaniline		0.07	0.00	1/17								
Chlorobenzene		21.00	0.83	19/42					3.00E+00	3	1.18E-01	4
Chloroform	B2			0/42								
2-Chlorophenol		0.05	0.00	2/39								
4-Chlorophenyl phenyl ether				0/42								
Chromium	A	0.08	0.01	12/42								
Chrysene	B2			0/42								
Copper		0.07	0.01	2/15					5.00E-02	18	4.28E-03	17
Cyanide		2.80	0.12	8/27								
Cyclohexane 3,3,5-trimethyl		1.30	0.14	1/9								
Dibenzo(a,h)anthracene	B2			0/42								
Dibenzofuran		0.01	0.00	1/17								
Dibromochloromethane				0/5								
1,2 Dichlorobenzene		3.25	0.22	9/42					1.69E-01	12	1.13E-02	12
1,3 Dichlorobenzene		0.10	0.00	5/42					5.19E-03	27	1.61E-04	27
1,4 Dichlorobenzene		0.47	0.02	7/42					2.44E-02	21	8.67E-04	21
1,1 Dichloroethane		0.01	0.00	1/42					2.84E-04	29	7.74E-06	29
1,2 Dichloroethane	B2	0.48	0.01	4/42	1.78E-03	12	4.49E-05	12	8.45E-03	25	2.13E-04	24
1,1 Dichloroethylene	C	0.02	0.00	2/42	5.70E-03	10	1.49E-04	10	8.53E-03	24	2.23E-04	23
1,2 trans Dichloroethylene		6.30	0.21	10/42					3.33E-01	10	1.09E-02	13
1,2 Dichloropropane				0/42								
Di-n-butyl phthalate		0.01	0.00	5/42					4.19E-04	28	3.81E-05	28
Di-n-octyl phthalate				0/42								
Diethylphthalate		0.21	0.01	4/42					5.61E-05	30	1.63E-06	30
1,2 Diphenylhydrazine	B2	2.10	0.07	3/37	2.75E-01	4	8.97E-03	4	7.01E-01	7	2.29E-02	9
Ethane 1,2-bis(2-chloroethoxy)		3.30	0.37	1/9								
Ethylbenzene		2.80	0.08	11/42					3.08E-02	20	9.19E-04	20
Fluoranthene		0.01	0.00	3/42								
Fluorene		0.01	0.00	1/42								
Furan,tetrahydrotetramethyl		0.56	0.06	1/9								
Hexachlorobenzene	B2	0.02	0.00	1/42	7.73E-03	8	1.68E-04	9	9.20E-03	22	2.00E-04	25
Hexachlorobutadiene	C			0/42								
Indeno(1,2,3-c,d)pyrene	C			0/42								
Iron		72.00	14.64	10/11								
Isophorone				0/42								
Lead		0.11	0.02	29/42					9.82E-02	16	1.89E-02	11

TABLE 1-2 (CONTINUED)

Compound	Carcinogenic Classification	Groundwater Concentrations* Maximum mg/l	Representative mg/l	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Manganese		15.00	1.95	25/27								
Mercury (inorganic)		0.00	0.00	1/31					9.20E-03	22	2.97E-04	22
Methanone, diphenyl		0.20	0.02	1/9								
4-Methyl 2-pentanone		0.37	0.01	3/42								
2-Methyl Phenol		0.03	0.00	3/17								
4-Methyl Phenol		0.23	0.03	3/17								
2-Methylnaphthalene		0.44	0.03	2/17								
Methylene chloride	B2	0.01	0.00	5/42					7.45E-06	31	5.52E-07	31
Naphthalene		0.72	0.02	8/42								
Nickel	A	0.10	0.01	4/15					4.26E-01	8	6.26E-02	6
N-nitrosodiphenylamine	B2	0.01	0.00	5/42								
Total Carcinogenic PAHs				0/42								
Pentachlorophenol		0.01	0.00	1/39								
Total PCBs	B2	1.10	0.04	5/30	1.58E+00	1	5.69E-02	1				
PCB-Aroclor 1248	B2	1.10	0.06	5/20								
PCB-Aroclor 1254	B2			0/20								
Phenanthrene	D	0.01	0.00	2/42								
Phenol		1.20	0.09	19/22					1.20E-01	13	8.95E-03	15
Phenol 4(1,1-dimethylethyl)		4.80	0.58	2/9								
Phenol 2,6,bis(1,1-di-methylethyl)		0.42	0.05	1/9								
Phenol 2,4,bis(1-methylethyl)		0.07	0.01	1/9								
Pyrene		0.00	0.00	2/42								
Silver		0.01	0.00	3/15					2.00E-01	11	4.00E-02	8
Sulfur		1.62	0.19	3/9								
1,1,2,2 Tetrachloroethane	C	3.80	0.13	4/42	1.80E-01	5	6.14E-03	5	1.73E+00	5	5.90E-02	7
Tetrachloroethylene	B2	0.75	0.02	2/42	6.22E-03	9	1.48E-04	11	7.22E-03	26	1.72E-04	26
Thallium		0.05	0.01	10/15								
1,2,4 Trichlorobenzene		0.46	0.01	1/42					9.84E-02	15	2.35E-03	19
1,1,2-Trichloroethane	C	0.03	0.00	1/42	2.78E-04	13	6.18E-06	14				
Trichloroethylene	B2	21.00	0.53	6/42	4.20E-02	6	1.05E-03	6	2.21E+01	1	5.52E-01	1
Trichlorofluoromethane				0/37								
Toluene		160.00	4.25	25/42					8.32E-01	6	2.21E-02	10
Total Xylenes		15.00	0.40	15/42								
Vinyl Chloride	A	1.00	0.04	4/42	4.29E-03	11	1.73E-04	8	8.77E-02	17	3.53E-03	18
Zinc		1.06	0.10	37/42					1.13E-01	14	1.02E-02	14

* All concentrations are automatically rounded off to the nearest 1/100 by the computer

TABLE 1-3

HAZARD IDENTIFICATION OF CONSTITUENTS BASED ON SURFACE SOIL DATA

Compound	Carcinogenic Classification	Surface Soil Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Acenapthene		2.30	0.10	5/36								
Acenaphthylene		0.52	0.04	3/36								
Acetone		14.00	0.56	15/37								
Alkane				-/0								
Anthracene		6.80	0.27	8/36								
Antimony				-/0								
Arsenic	A	18.00	4.77	23/36	3.65E-03	2	9.68E-04	2	1.62E-02	3	4.29E-03	2
Benzene	A	48.00	1.21	11/52	1.85E-05	7	4.67E-07	7	2.81E-04	11	7.08E-06	11
Benzene, acetic acid				-/0								
Benzene, acetonitrile				-/0								
Benzene, -1-chlor-2-methyl				-/0								
Benzene,1-(1,1 dimethylethyl)				-/0								
Benzene, 1-1' methylene bis				-/0								
Benzene, (methyl sulfonyl)				-/0								
Benzene 1,1-(oxy-bis(methylene))				-/0								
Benzene, 1,-sulfonyl bis				-/0								
Benzo(a) anthracene	B2	18.00	0.80	15/36	5.24E-04	5	2.32E-05	5				
Benzo(b) fluoranthene	B2	21.00	1.11	18/36								
Benzo(k) fluoranthene	D	21.00	1.10	18/36								
Benzo (g,h,i) perylene		9.10	0.40	11/36								
Benzo(a) pyrene	B2	14.00	0.70	16/36	3.19E-03	3	1.60E-04	3	1.86E-02	2	9.36E-04	5
Benzoic acid		1,100.00	60.65	9/21								
Benzo acid 4-chloro				-/0								
Benzoic acid, 4(-1,1-dimethylethyl)				-/0								
Benzoic acid, 3-methyl				-/0								
Benzyl alcohol		9.70	0.63	6/21								
Beryllium	B1			-/0								
Bicyclo-heptanone-trimethyl				-/0								
Bis(2-chloroethyl)ether	B2			0/41								
Bis(2-ethylhexyl)phthalate	B2	17.00	1.79	23/36	4.86E-07	12	5.12E-08	10				
Bromodichloromethane		0.00	0.00	1/52								
4 Bromophenyl phenyl ether				0/36								
2-Butanone		2.30	0.19	4/37					8.85E-07	22	7.39E-08	22
Butyl benzyl phthalate		0.23	0.01	2/36								

TABLE 1-3 (CONTINUED)

Compound	Carcinogenic Classification	Surface Soil Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Cadmium	B1	16.00	1.38	13/35					3.57E-03	6	3.09E-04	6
Carbon disulfide		4.40	0.12	6/37					9.33E-05	14	2.54E-06	14
4-Chloroaniline				0/21								
Chlorobenzene		23.00	0.66	14/52					1.64E-04	12	4.72E-06	13
Chloroform	B2	0.00	0.00	2/52	5.62E-09	16	2.81E-10	16				
2-Chlorophenol				0/21								
4-Chlorophenyl phenyl ether		0.76	0.02	1/36								
Chromium	A	2,880.00	138.60	47/47								
Chrysene	B2	15.00	0.75	15/36								
Copper				-/0								
Cyanide		34.80	2.43	22/35								
Cyclohexane 3,3,5-trimethyl				-/0								
Dibenzo(a,h)anthracene	B2	2.70	0.09	4/36	9.64E-04	4	3.38E-05	4				
Dibenzofuran		2.30	0.13	3/21								
Dibromochloromethane		0.00	0.00	1/52					1.82E-07	24	3.50E-09	25
1,2 Dichlorobenzene**		550.00	16.40	14/37					1.59E-04	7	8.29E-06	7
1,3 Dichlorobenzene		2.30	0.12	4/36					5.98E-06	20	2.99E-07	19
1,4 Dichlorobenzene		9.00	0.41	3/36					2.34E-05	15	1.07E-06	15
1,1 Dichloroethane				0/52								
1,2 Dichloroethane	B2	0.11	0.00	3/52	2.05E-08	15	4.09E-10	15	9.68E-08	26	1.94E-09	26
1,1 Dichloroethylene	C	0.01	0.00	3/52	9.92E-08	14	3.72E-09	14	1.49E-07	25	5.58E-09	24
1,2 trans Dichloroethylene		7.60	0.15	4/52					2.01E-05	16	3.90E-07	17
1,2 Dichloropropane				0/52								
Di-n-butyl phthalate		0.29	0.01	1/36					5.51E-07	23	1.54E-08	23
Di-n-octyl phthalate				0/36								
Diethylphthalate				0/36								
1,2 Diphenylhydrazine	B2			0/15								
Ethane 1,2-bis(2-chloroethoxy)				-/0								
Ethylbenzene		19.00	0.94	11/52					1.05E-05	17	5.17E-07	16
Fluoranthene		37.00	1.62	18/36								
Fluorene		3.00	0.12	6/36								
Furan,tetrahydrotetramethyl				-/0								
Hexachlorobenzene	B2	0.44	0.02	2/36	7.39E-06	8	2.62E-07	8	8.80E-06	18	3.12E-07	18
Hexachlorobutadiene	C	2.10	0.06	1/36	1.77E-06	10	4.91E-08	11				
Indeno(1,2,3-c,d)pyrene	C	9.90	0.42	10/36								
Iron				-/0								
Isophorone		3.10	0.09	1/36								
Lead		1,820.00	238.03	37/37					8.12E-02	1	1.06E-02	1

TABLE 1-3 (CONTINUED)

Compound	Carcinogenic Classification	Surface Soil Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Manganese		3,100.00	659.47	36/36								
Mercury (inorganic)		10.00	2.48	22/23					9.21E-03	4	2.29E-03	3
Methanone, diphenyl				-/0								
4-Methyl 2-pentanone		0.01	0.00	1/37								
2-Methyl Phenol		0.60	0.03	1/21								
4-Methyl Phenol		0.25	0.02	2/21								
2-Methylnaphthalene		3.70	0.20	2/21								
Methylene chloride	B2	130.00	2.94	37/52					5.98E-06	19	1.35E-07	20
Naphthalene		2.70	0.19	7/36								
Nickel	A			-/0								
N-nitrosodiphenylamine	B2	11.00	0.87	13/36								
Total Carcinogenic PAHs		80.60	3.87	18/36								
Pentachlorophenol		0.13	0.01	2/21								
Total PCBs	B2	480.00	21.39	20/30	3.46E-02	1	1.54E-03	1				
PCB-Aroclor 1248	B2	480.00	21.35	18/30								
PCB-Aroclor 1254	B2	0.64	0.04	5/30								
Phenanthrene	D	26.00	1.09	14/36								
Phenol				0/21								
Phenol 4(1,1-dimethylethyl)				-/0								
Phenol 2,6,bis(1,1-di-methylethyl)				-/0								
Phenol 2,4,bis(1-methylethyl)				-/0								
Pyrene		21.00	1.12	17/36								
Silver				-/0								
Sulfur				-/0								
1,1,2,2 Tetrachloroethane	C	24.00	0.47	4/52	5.69E-05	6	1.12E-06	6	5.45E-04	9	1.07E-05	9
Tetrachloroethylene	B2	8.10	0.22	10/37	3.35E-06	9	9.22E-08	9	3.90E-06	21	1.07E-07	21
Thallium				-/0								
1,2,4 Trichlorobenzene		14.00	0.61	6/36					1.50E-04	13	6.49E-06	12
1,1,2-Trichloroethane	C	0.54	0.01	1/52	2.78E-07	13	5.35E-09	13				
Trichloroethylene	B2	8.40	0.17	12/52	8.40E-07	11	1.65E-08	12	4.42E-04	10	8.71E-06	10
Trichlorofluoromethane												
Toluene		2,100.00	60.72	38/52					5.46E-04	8	1.58E-05	8
Total Xylenes		160.00	8.11	13/37								
Vinyl Chloride	A			0/52								
Zinc		1,530.00	197.74	38/38					8.15E-03	5	1.05E-03	4

* All concentrations are automatically rounded off to the nearest 1/100 by the computer

** When higher 1,2-Dichlorobenzene concentrations were tentatively identified during extra peak runs, that concentration was used to determine the maximum and representative concentrations.

TABLE 1-4

HAZARD IDENTIFICATION OF CONSTITUENTS BASED ON SUBSURFACE SOIL DATA

Compound	Carcinogenic Classification	Subsurface Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Acenapthene		3.50	0.22	6/34								
Acenaphthylene		0.43	0.02	2/34								
Acetone		15.00	1.12	28/43								
Anthracene		7.40	0.35	/34								
Alkane				-/0								
Antimony				-/0								
Arsenic	A	52.00	7.62	20/33	1.06E-02	1	1.55E-03	1	4.68E-02	2	6.86E-03	3
Benzene	A	33.00	1.50	13/50	1.27E-05	9	5.78E-07	8	1.93E-04	14	8.76E-06	14
Benzene, acetic acid				-/0								
Benzene, acetonitrile				-/0								
Benzene, -1-chlor-2-methyl				-/0								
Benzene,1-(1,1 dimethylethyl)				-/0								
Benzene, 1-1' methylene bis				-/0								
Benzene, (methyl sulfonyl)				-/0								
Benzene 1,1-(oxy-bis(methylene))				-/0								
Benzene, 1,-sulfonyl bis				-/0								
Benzo(a) anthracene	B2	19.00	0.81	6/34	5.53E-04	5	2.35E-05	5				
Benzo(b) fluoranthene	B2	27.00	1.29	7/28								
Benzo(k) fluoranthene	D	27.00	1.06	7/34								
Benzo (g,h,i) perylene		9.50	0.40	5/34								
Benzo(a) pyrene	B2	17.00	0.71	7/34	3.88E-03	2	1.62E-04	3	2.26E-02	4	9.44E-04	5
Benzoic acid		8,500.00	315.16	22/27								
Benzo acid 4-chloro				-/0								
Benzoic acid, 4(-1,1-dimethylethyl)				-/0								
Benzoic acid, 3-methyl				-/0								
Benzyl alcohol		51.00	1.90	2/27								
Beryllium	B1			-/0								
Bicyclo-heptanone-trimethyl				-/0								
Bis(2-chloroethyl)ether	B2			0/34								
Bis(2-ethylhexyl)phthalate	B2	690.00	24.83	23/34	1.97E-05	8	7.10E-07	7				
Bromodichloromethane				0/44								
4 Bromophenyl phenyl ether		1.70	0.05	1/34								
2-Butanone		5.80	0.53	13/43					2.23E-06	21	2.03E-07	21
Butyl benzyl phthalate		1.10	0.03	1/34								

TABLE 1-4 (CONTINUED)

Compound	Carcinogenic Classification	Subsurface Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Cadmium	B1	34.00	1.58	11/34					7.58E-03	6	3.52E-04	6
Carbon disulfide		0.79	0.03	10/43					1.67E-05	19	6.78E-07	17
4-Chloroaniline				0/27								
Chlorobenzene		160.00	5.19	22/50					1.14E-03	10	3.71E-05	10
Chloroform	B2			0/50								
2-Chlorophenol		0.95	0.04	1/27								
4-Chlorophenyl phenyl ether				0/34								
Chromium	A	7,250.00	439.36	45/45								
Chrysene	B2	17.00	0.72	6/34								
Copper				-/0								
Cyanide		62.30	2.91	21/34								
Cyclohexane 3,3,5-trimethyl				-/0								
Dibenzo(a,h)anthracene	B2	2.60	0.10	5/34	9.28E-04	4	3.71E-05	4				
Dibenzofuran		3.70	0.22	5/27								
Dibromochloromethane				0/44								
1,2 Dichlorobenzene**		710.00	21.07	13/36					1.85E-03	9	5.48E-05	9
1,3 Dichlorobenzene		33.00	1.17	5/34					8.58E-05	15	3.04E-06	15
1,4 Dichlorobenzene		130.00	4.49	7/34					3.38E-04	13	1.17E-05	12
1,1 Dichloroethane				0/50								
1,2 Dichloroethane	B2			0/50								
1,1 Dichloroethylene	C	0.00	0.00	1/50	2.48E-08	11	4.96E-10	11	3.72E-08	24	7.44E-10	24
1,2 trans Dichloroethylene		6.70	0.14	3/50					1.78E-05	18	3.79E-07	20
1,2 Dichloropropane				0/50								
Di-n-butyl phthalate		0.78	0.04	3/34					1.48E-06	23	7.22E-08	23
Di-n-octyl phthalate		0.30	0.01	1/34								
Diethylphthalate		0.25	0.01	1/34					3.35E-09	25	9.38E-11	25
1,2 Diphenylhydrazine	B2			0/13								
Ethane 1,2-bis(2-chloroethoxy)				-/0								
Ethylbenzene		27.00	1.11	16/50					1.49E-05	20	6.15E-07	18
Fluoranthene		33.00	1.58	11/34								
Fluorene		6.50	0.28	5/34								
Furan,tetrahydrotetramethyl				-/0								
Hexachlorobenzene	B2			0/34								
Hexachlorobutadiene	C			0/34								
Indeno(1,2,3-c,d)pyrene	C	12.00	0.48	5/34								
Iron				-/0								
Isophorone				0/34								
Lead		1,000.00	169.80	35/35					4.46E-02	3	7.57E-03	2

TABLE 1-4 (CONTINUED)

Compound	Carcinogenic Classification	Subsurface Concentrations* Maximum mg/kg	Representative mg/kg	Frequency	Carcinogenic IS Maximum	Rank	Representative	Rank	Non-Carcinogenic IS Maximum	Rank	Representative	Rank
Manganese		4,730.00	550.18	34/34								
Mercury (inorganic)		190.00	10.06	22/26					1.75E-01	1	9.27E-03	1
Methanone, diphenyl				-/0								
4-Methyl 2-pentanone				0/43								
2-Methyl Phenol		2.90	0.12	2/27								
4-Methyl Phenol		210.00	8.54	3/27								
2-Methylnaphthalene		3.60	0.35	7/27								
Methylene chloride	B2	33.00	2.09	41/50					1.52E-06	22	9.62E-08	22
Naphthalene		11.00	0.89	10/34								
Nickel	A			-/0								
N-nitrosodiphenylamine	B2	15.00	1.54	18/34								
Total Carcinogenic PAHs		94.60	3.88	7/34								
Pentachlorophenol				0/27								
Total PCBs	B2	38.00	3.77	18/31	2.74E-03	3	2.72E-04	2				
PCB-Aroclor 1248	B2	38.00	3.75	17/31								
PCB-Aroclor 1254	B2	0.45	0.02	5/31								
Phenanthrene	D	18.00	1.20	8/34								
Phenol		6.70	0.29	2/27					3.36E-05	16	1.47E-06	16
Phenol 4(1,1-dimethylethyl)				-/0								
Phenol 2,6,bis(1,1-di-methylethyl)				-/0								
Phenol 2,4,bis(1-methylethyl)				-/0								
Pyrene		42.00	1.74	10/34								
Silver				-/0								
Sulfur				-/0								
1,1,2,2 Tetrachloroethane	C	230.00	4.60	1/50	5.45E-04	6	1.09E-05	6	5.22E-03	7	1.04E-04	7
Tetrachloroethylene	B2	48.00	1.12	6/43	1.99E-05	7	4.63E-07	9	2.31E-05	17	5.38E-07	19
Thallium				-/0								
1,2,4 Trichlorobenzene		64.00	2.62	2/34					6.85E-04	11	2.80E-05	11
1,1,2-Trichloroethane	C			0/50								
Trichloroethylene	B2	80.00	1.62	5/50	8.00E-06	10	1.62E-07	10	4.21E-03	8	8.54E-05	8
Trichlorofluoromethane		0.07	0.01	1/13								
Toluene		1,600.00	39.33	37/50					4.16E-04	12	1.02E-05	13
Total Xylenes		120.00	4.90	16/43								
Vinyl Chloride	A			0/50								
Zinc		4,010.00	337.48	35/35					2.14E-02	5	1.80E-03	4

* All concentrations are automatically rounded off to the nearest 1/100 by the computer

** When higher 1,2-Dichlorobenzene concentrations were tentatively identified during extra peak runs, that concentration was used to determine the maximum and representative concentrations.

1.4 Selection of Indicator Compounds

Inspection of the analytical data at the UOP Site gives the picture of a site with many detected contaminants, only a few of which were found consistently. Thus high representative indicator scores were not the only factor considered important to the selection of Indicator Chemicals. Compounds with high scores that were detected infrequently were judged not to be significant health hazards at the site. Conversely, high frequency of detection in one or more media was considered to be sufficiently important to be the basis for choosing some compounds regardless of their low indicator scores (or in some cases, lack of indicator scores because of no published toxicity data).

Indicator compounds are listed in Table 1-5. Compounds chosen on the basis of high indicator score rank (based on maximum concentrations) were:

- arsenic,
- benzene,
- carcinogenic polynuclear aromatic hydrocarbons (PAH, including: benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, chrysene, dibenzo-[a,h]anthracene), and indeno(1,2,3-cd)pyrene,
- polychlorinated biphenyls (PCBs),
- chlorobenzene, and
- lead.

Some contaminants detected frequently at the UOP site either did not have toxicity constants published for them or were ranked low in the scoring system. Although the high-score compounds do in fact characterize the health risk at the site fairly comprehensively, two other substances for which no EPA toxicity constants are available and two low-ranking compounds were also included as "priority" health risks due to their frequency of detection. These compounds are:

TABLE 1-5

SUMMARY: INDICATOR CHEMICALS UOP SITE,

EAST RUTHERFORD, N.J.

Compound	Ground Water		Surface Soil		Subsurface Soils	
	IS Rank[d]	Frequency of Detection	IS Rank[d]	Frequency of Detection	IS Rank[d]	Frequency of Detection
Carcinogens						
Arsenic	2	25/42	2	23/36	1	20/33
Benzene	3	25/42	7	11/52	9	13/50
Bis(2 ethylhexyl) phthalate	14	11/42	12	23/36	8	23/34
Carcinogenic PAH	-	not found	4,3,5	16/36, 4/36, 15/36	4,2,5	5/34, 7/34, 6/34
Chromium	c	12/42	c	47/47	c	45/45
1,2-Diphenylhydrazine	4	3/37	-	not found	-	not found
PCB	1	5/30	1	20/30	3	18/31
1,1,2,2-Tetrachloroethane	5	4/42	6	4/52	6	1/50
Non Carcinogens						
Cadmium	19	16/42	6	13/35	6	11/34
Chlorobenzene	3	19/42	12	14/52	10	22/50
Cyanide	c	8/27	c	22/35	c	21/34
1,2-Dichlorobenzene	12	9/42	7	14/37	9	13/36
Lead	16	29/42	1	37/37	3	35/35
Mercury	22	1/31	4	22/23	1	22/26
Nickel	8	4/I5	-	not found	-	not found
Toluene	6	25/42	8	38/52	12	37/50
Zinc	14	37/42	5	38/38	5	35/35

a. Arsenic was present in soil at representative concentrations below New Jersey background concentrations.

b. Dibenzo[a,h]anthracene (soil ranks = 4,4), Benzo[a]pyrene (soil ranks = 3,2), and Benzo[a]anthracene (soil ranks = 5,5) were considered total "carcinogenic PAH" for the purposes of indicator compound selection.

c. Compounds do not have constants for use in the hazard calculation but will be considered due to the fact that these compounds were found more often than others.

d. IS rank based on maximum detected concentration.

- chromium (no toxicity constant)
- cyanides (no toxicity constant)
- bis(2-ethylhexyl)phthalate (low rank), and
- 1,2-dichlorobenzene (low rank).

In addition to the above Indicator Compounds, the NJDEP directed that the following chemicals also be treated as indicator compounds:

- Cadmium
- Mercury
- Nickel
- Zinc
- Toluene
- 1,2-diphenyhydrazine
- 1,1,2,2-tetrachloroethane

An assessment of surface soil data has been performed to evaluate the possible significance of Tentatively Identified Compounds (TICs). Only one compound, 1,1,2,3,4,4-hexachloro-1,3-Butadiene, had the input values necessary to evaluate its risk. This compound was detected in just 2 samples at very low estimated concentrations (max = 25 ug/kg) and has relatively low toxicity constants. Therefore, this compound does not meet any of the criteria for inclusion as an indicator compound and, if included, would not contribute to the total risk reported in the risk assessment.

Of the TICs found in soil, many are substituted chlorinated benzenes for which toxicity data are not available, and therefore cannot be evaluated for risk. However, related benzene compounds (benzene, chlorobenzene, and 1,2-dichlorobenzene) are indicator compounds and were evaluated. Not only are these compounds related, the ICs are also detected more frequently and at higher concentrations than the substituted chlorinated benzenes.

As a note of clarification, several compounds on the TIC list other than hexachlorobutadiene do have toxicity data; however, these compounds were accounted for as listed compounds in other analyses. For example, 1,2-dichlorobenzene (DCB) is included as a listed compound in the Base/Neutral Extractable suite but is also a TIC in the Volatile Organics suite. For conservatism, the higher concentrations found for DCB in the volatile organics suite are used in the data tables of this section.

The NJDEP has requested an assessment of the presence of 1,2-diphenylhydrazine in well 28I (August 22, 1988 letter, General Comment 1). This compound is included as an indicator compound and is addressed in Calculation Number 1 of Appendix A.

2. TOXICITY ASSESSMENT

The following section provides toxicity profiles and EPA estimates of the dose-responsiveness of the Indicator Chemicals at the UOP Site. For Indicator Chemicals that also occur naturally in the environment (metals and PAH), a determination of whether the concentrations at the site are elevated above local or national "background" (and thus represent an excess health risk) is also provided.

The dose-response assessment takes two forms. For non-carcinogenic substances, the underlying presumption is that a threshold for the effect exists. That is, there is a dose below which no effect will occur. Acceptable Intakes for Chronic exposures (AICs) are developed by EPA for non-carcinogenic compounds to provide reasonable certainty that the specified intake value is subthreshold and the risk is therefore practically zero.

Approximately 200 compounds have been reviewed by the EPA Carcinogen Assessment Group (CAG) pertaining to their carcinogenic potency. The underlying assumption for carcinogens is that there is no threshold for effect. Thus, there is no non-zero dose that is without some finite level of risk. The CAG has developed computerized methods that extrapolate observed dose-response relations to the low dose levels encountered in environmental situations. They incorporate both the no-threshold assumption and a further assumption that carcinogenic dose-response is linear at low doses. The result of the dose-response curve fitting computations is a "potency slope", which has units of reciprocal milligrams of compound per kilogram body weight per day ($[mg/kg/day]^{-1}$). Using the linearity assumption, a predicted intake needs only to be multiplied by a potency slope to give (unitless) risk values. The computed risk value should be viewed as an estimate of the excess chance of getting cancer above background cancer rates produced by intake of carcinogenic contaminants. In some cases, the CAG computation

produces a maximum likelihood estimate of the carcinogen dose-response relation, while in others, the 95% upper confidence bound on the dose-response relation is calculated. In the latter case, the cancer risk estimate for exposure at a site is an "upper-bound" estimate, the actual risk may, in fact, be lower.

2.1 Arsenic

The arsenic concentrations found in surface soil at the UOP site (4,770 µg/kg average, 18,000 µg/kg maximum) are within the limits of New Jersey background concentrations (800 - 73,800 µg/kg, (Harkov, et al., 1987). Even though exposure to and risk from arsenic do not exceed background, they will be included in the health risk assessments as directed by the NJDEP.

Arsenic is an irritant of skin, mucous membranes, and the gastrointestinal tract. Acute toxicity from ingestion results in vomiting, diarrhea, and cardiovascular effects. Acute exposure to airborne arsenic, adsorbed on particles, causes conjunctivitis and pharyngitis. Chronic exposure to high levels of arsenic are associated with fatigue, anemia, peripheral nerve injury, and hyperpigmentation or hyperkeratoses of the skin. Peripheral blood vessel effects which produce gangrene of extremeties ("Blackfoot") may also be caused by arsenic ingestion.

The interim drinking water standard, maximum contaminant level (MCL) and proposed recommended maximum concentration level (RMCL) is 50 ug/L.

Chronic inhalation of arsenic is associated with pulmonary cancer in producers of arsenical pesticides, and smelter workers. Ingestion of water with high inorganic arsenic levels, and taking arsenical medications have both been reported to be associated with cancer of the skin, although drinking water epidemiology studies in the U.S. have failed to confirm this finding. The CAG used the carcinogenicity data of Tseng, et al (1968) in a computer-fit model for dose-response

(the Weibull Distribution) which gives a potency slope of 1.5 $(mg/kg/day)^{-1}$. This value indicates that an increased risk of cancer of about 1.5 chances in 1000 is incurred by an individual ingesting 1 µg arsenic per kilogram body weight, daily, for life. Multiplying this value by the predicted intake of arsenic gives an estimate of risk from arsenic ingestion at the UOP Site. A similar extrapolation has been done to predict the cancer risk from inhalation exposures of arsenic. In this case data from a variety of epidemiologic reports on cancer in smelterworkers has been treated with an "absolute risk" linear model to give an inhalation potency slope of 1.8 $(mg/kg/day)^{-1}$ (EPA, 1988b).

Arsenic exists in two valence states. Naturally occurring arsenic is usually pentavalent and forms arsenate compounds. Arsenic that is introduced into the environment is usually trivalent and forms arsenites. Although arsenites are believed to be responsible for most toxic effects, the analytical data for the UOP Site has not been speciated. A conservative approach is to assume all arsenic detected is As (3+).

2.2 Benzene

Benzene and other light aromatic hydrocarbons (e.g., toluene, xylenes) are present in a variety of petroleum products including automotive fuels, fuel oils, lubricating oils, as well as wood and coal distillates. Benzene itself serves many purposes as a solvent, degreaser, fuel additive, and starting product for pharmaceuticals and synthetic chemicals.

Benzene has long been recognized to produce a variety of hematologic effects (effects on blood cells) in occupationally-exposed humans. This toxic effect of benzene is probably related to actions of the compound on the precursors of circulating blood cells that reside in the bone marrow. It has been a problem to determine the mechanism of this action because the toxic effect is difficult to produce in experimental animals. Humans have been shown to acquire anemia

(decreased red blood cells), leukopenia (decreased white cells) and thrombocytopenia (decreased platelets) on exposure to benzene. Chronic benzene exposure may lead to a decrease in all circulating cells (pancytopenia) or failure to manufacture blood cells altogether (aplastic anemia) (Goldstein, 1977).

Benzene has been reported to cause leukemia in workers exposed (Aksoy, et al, 1974; Infante, 1977a, b; Ott, 1978) by inhalation. For this reason, benzene is among the few substances given an "A" weight of evidence rating for carcinogenicity,* indicating the greatest certainty that the compound is a human carcinogen. The CAG has used this data in a linear dose-response model to obtain a cancer potency slope of 2.9×10^{-2} $(mg/kg/day)^{-1}$ for inhalation of benzene. When corrections are made to extrapolate the inhalation route of exposure to a presumed ingestion exposure the value is 2.9×10^{-2} $(mg/kg/day)^{-1}$. This value suggests that an individual ingesting 1 µg of benzene per kg body weight per day, for life, would have an excess risk of cancer of approximately 3 chances in 100,000 and inhalation of 1 µg/kg day would also produce a risk of approximately 3 chances in 100,000.

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*Only a limited number of chemical compounds have been demonstrated unequivocally to be human carcinogens. However, experimental and epidemiologic data are available that are suggestive of the carcinogenic activity of certain compounds. The quality and quantity of these data vary between compounds. EPA has developed a "weight-of-evidence" system that is intended to reflect the decreasing level of certainty that a compound is, in fact, a human carcinogen based on available data. The categories are:

1) A; human carcinogen - demonstrated human carcinogen
2) B-1; probable human carcinogen - suggested by limited studies in humans
3) B-2; probable human carcinogen - suggested by lifetime studies in animals
4) C; possible human carcinogen - suggested by limited studies in animals
5) D; no data or no demonstrated carcinogenic activity

2.3 Bis (2-ethylhexyl) Phthalate

Bis-(2-ethylhexyl) phthalate (BEHP) is primarily used as a plasticizer for resins such as polyvinyl chloride. Because plastic products are an intricate part of our life and because they are largely nonbiodegradable, additives like BEHP are widely present in our environment.

BEHP is the most persistent of the phthalate esters, breaking down slowly to monophthalate or phthalic acid. The fate and transport of BEHP in water is determined by its low solubility (400 µg/l (EPA, 1980a). It settles in sediment and is mobilized via entrainment mechanisms. The high octanol/water partition coefficient (log K_{ow} = 4.89) renders BEHP lipophilic (EPA, 1980b). This property contributes to the high bioconcentration factors seen in aquatic invertebrate and plant organisms: 107,670 in mosquito larvae (Culex) and 53,890 in algae (Oedogonium) (Metcalf et al., 1973). However, the bioconcentration factor for fish, such as guppies (Gambusia), calculated by the same authors, is much lower (130) signifying that some of the ingested BEHP is being metabolized and excreted.

The acute toxicity studies reveal that BEHP is a low order toxin. The range of rodent LD50s is from 14.2 g/kg to greater than 50 g/kg. The target organs appear to be the lungs and liver. Chronic and subchronic studies revealed testicular degeneration (Shaffer, et al 1945; Gray et al, 1977; NTP, 1980) and several studies observed decreased body weight gain and significant liver enlargement in animals that received oral doses ranging from 64000 µg/kg day to 2,000,000 µg/kg day (Gray, et al, 1977; Bell et al, 1978; Moody and Reddy, 1978). Chronic toxicity studies reviewed by the EPA (1980a) showed only dose-related liver enlargement at doses ranging from 20,000 µg/kg day to 400,000 µg/kg day. No adverse effects related to mortality, hematopoetic system, or fertility were observed in multigenerational studies done by Carpenter et al. (1953). The AIC for BEHP, published by the EPA's Office of Emergency and Remedial Response in the SPHEM, is 20 µg/kg.

Data has recently suggested that BEHP may be a liver carcinogen in rats fed 1.2% (12,000,000 ppb) BEHP in their diet and in mice fed 300,000 ppb BEHP (NTP, 1980). The EPA published a potency factor for BEHP, presumably based on the NTP Study, of 8.4×10^{-3} $(mg/kg/day)^{-1}$ for oral exposure (EPA 1988). No inhalation potency slope is available. No documentation regarding the methodology used to derive the potency slope accompanied this value.

2.4 Cadmium

Cadmium is a metal generally found in conjunction with zinc and lead ores. In the environment it typically exists as a salt of the +2 valence state or as the metal; it forms no stable organic compounds. Different cadmium salts have different water solubilities, with the oxide of cadmium being less soluble than the chloride. The abundance in the earth's crust is approximately 0.2 mg/kg. Man made/produced cadmium releases are generally associated with mining, smelting, manufacturing operations, and from the disposal of alkaline batteries containing cadmium (Doull, 1980; EPA 1981a).

Human exposure to cadmium is primarily through the ingestion of food, with vegetables typically containing less than 0.1 mg/kg, and up to 10 mg/kg shellfish, liver and kidneys. Consumption of food grown in contaminated areas results in exposures to cadmium. Absorption of cadmium is much higher in children than adults (EPA 1981a).

A great deal of data on the toxic effects of cadmium has been generated. The principal effects of chronic cadmium exposure are osteomalacia and osteoporosis (Itai Itai disease), and giomerular and tubular necrosis in the kidney. The Itai Itai ("ouch ouch") disease is endemic to the Jintsu River basin and other areas in Japan, which has been contaminated with mining wastes containing cadmium. The primary exposure is via ingestion of rice grown in the contaminated soils and water. Itai Itai victims display the osteomalacia and osteoporosis as

primary symptoms, as well as proteinuria, glycosuria, and aminoaciduria. Other chronic effects include immunosuppression, and decreases in measures of respiratory fitness (ventilation capacity, vital capacity, forced expiratory volume, etc.) (EPA 1981a).

A recent (1987a) review by the EPA of new carcinogenicity data indicates carcinogenesis is a health effect of concern. A 1985 epidemiological study of cadmium smelter workers demonstrated a significant association between exposure to cadmium and respiratory tract cancer. On the basis of these data, the CAG has classified cadmium as B1, a human carcinogen with adequate evidence for carcinogenicity in humans, and inadequate evidence in animals. Using these data, the CAG has computed the inhalation cancer potency of cadmium to be 6.1 $(mg/kg/day)^{-1}$ (EPA, 1987b). Cadmium has not been shown to be carcinogenic by ingestion and Acceptable Intake Chronic (AIC) values have been developed by EPA. These values are $1x10^{-3}$ (food) and $5x10^{-4}$ (water).

2.5 Carcinogenic PAH

PAHs are found in widely varying concentrations in surface and shallow soils all over the world. Since these compounds are formed primarily as a result of elevated temperature processes, there is widespread contribution of "naturally-occurring" PAHs from forest and prairie fires. Naturally-occurring PAHs are also formed at a much slower rate in natural crude oil products in the earth, which takes place at a temperature range of 100-150°C. The largest contribution of PAHs to the environment comes from man-made sources such as fossil fuel combustion and anthropogenic refuse, forest and agricultural fires. Particulate PAHs from these various sources can be atmospherics transported great distances and deposited on soil surfaces where, over time, they are leached into deeper soil layers.

The literature reports PAH concentrations in environmental soil samples of many origins. Youngblood and Blumer (1975) report PAH concentrations of 7,000 ug/kg (ppb) for coniferous forest soil in Maine, and 13,000 ug/kg in an oak forest in Massachusetts. They also report, in a 1977 publication, Swiss soils with a PAH concentration of 5,000-120,000 ug/kg from open country near a town, increasing to 21,000-300,000 ug/kg for samples taken near a highway in that same area.

Windsor and Hites (1979) looked at levels of ten individual PAHs in a variety of Nova Scotia soils. Totals of these concentrations were substantially lower than results from studies which quantify a larger number of PAH compounds. This study included a majority of the most toxic PAH species, however, and showed peat layers from a dense conifer forest in Nova Scotia at levels of only 240 ug/kg for the ten specific PAHs. Many other similar studies report concentrations with as much variability as the examples provided above. Highest values are generally associated with urban environments, specific industrial contribution or highway influence.

The pattern of PAHs, confined to Area 5 of the UOP Site, is characterized by elevated concentrations in the southern portion and relatively small concentrations across the remainder of the area. Due to the uneven distribution, the PAHs in the southern portion of Area 5 are probably not present as a result of some broad impact such as particulate PAH transfer from off-site traffic, industry or other combustion sources. The elevated PAH concentrations are at levels consistent with the possibility of a past burning of vegetation, disposal of a waste product (such as ash) which may have contained low concentrations of PAHs, or other similar sources. It is therefore necessary to include soil PAH among the compounds with potential for exposure above background levels at the UOP site. Smaller PAH concentrations across the remainder of Area 5 could have resulted from the off-site sources described above.

Production of cancer, either systemically or at a point of dermal contact is a toxic effect of a subset of PAH. Carcinogenic PAH generally tend to be high in molecular weight, have at least 3 aromatic rings (usually more), have low water solubility, are easily absorbed by the human and have very low acute toxicity.

EPA CAG has given a C to B2 rating of carcinogenicity to six high molecular weight PAH (no PAH is rated A or B-1). These compounds: benzo[a]anthracene, benzo[b]fluoranthene, benzo[a]pyrene, chrysene, dibenzo[a,h]anthracene and indeno(1,2,3-cd)pyrene have been detected in surface soil at the UOP site.

An adequate database for quantitatively determining the carcinogenic potency of each PAH detected at the site is lacking. In determining Ambient Water Quality Criteria, the EPA (1980b) used animal dose response data for benzo[a]pyrene to establish a criteria for all carcinogenic PAH (summed quantities). The validity of this approach is questionable because the carcinogenic potency of PAH is not only variable between compounds, but the potency of an individual compound may change according to the route of exposure and the presence of other compounds in the exposure mixture. It is nonetheless the only method available.

The EPA used the data of Neal and Rigdon (1967) to derive a potency slope for benzo(a)pyrene that is applied to all carcinogenic PAH. Neal and Rigdon (1967) gave mice feed containing between 1 and 250 ppm benzo[a]pyrene and found that more treated rats developed stomach tumors than the control group. The increased tumor incidence was dose dependent. After adjusting the doses to correct for presumed differences in mouse versus human metabolism, this data was used in a computer program which calculates the upper 95% confidence interval on the slope of a dose response line fitted to an equation modeling the assumed no threshold, multistage mechanism of chemical carcinogenesis. The potency slope derived is 11.5 $(mg/kg/day)^{-1}$ for ingestion exposures to benzo[a]pyrene. The potency slope indicates that an individual

consuming 1 µg benzo[a]pyrene per kg body weight, daily, for life, might have a risk of contracting cancer of about 1 chance in 100 over that of the non-exposed individual (note that this is an upper bound on the estimate, the actual risk is likely to be lower). The potency slope for inhalation exposures to PAH are based on the data of Thyssen et al., 1981. Using a similar dose-response extrapolation method, the EPA CAG determined the inhalation slope to be 6.11 $(mg/kg/day)^{-1}$. Because the dose-response relation is presumed to be linear, simply multiplying the predicted lifetime daily intake of carcinogenic PAH by the potency slope will give an upper bound estimate of excess cancer risk from exposure to constituents at the UOP Site.

2.6 Chlorobenzene

Although chlorobenzene (monochlorobenzene, MCB) is used as a solvent and chemical intermediate in American Industry, little toxicity literature is available for the compound. This may be due to the fact that few effects were seen in the sub-chronic toxicity tests of the compound that have been done in laboratory animals. Both Deichman (1981) and the U.S. EPA (1984c) cite several studies of oral exposure of animals to MCB where high doses produced non-specific changes of liver and kidney while lower doses were without effect. The U.S. EPA indicates that the maximum no-effect level found in these experiments was that seen in dogs in a study conducted by the Monsanto Chemical Company (Knapp, 1971). This dose, 27,000 µg/kg day, was divided by a 100-fold safety factor to arrive at a subchronic acceptable intake. The chronic acceptable intake (AIC) was set at 27 µg/kg day based on an additional 10-fold safety factor to correct for uncertainty involved in unstudied effects of extended exposures.

Toxic effects of MCB by the inhalation exposure route have been observed to be similar to those seen by the ingestion route. For this type of exposure, the U.S. EPA (1984c) used

the data of Dilley (1977) to set an acceptable inhalation intake. Dilley observed liver, kidney, and adrenocortical alterations in rats placed in an atmosphere containing 75 ppm MCB, seven hours per day, five days per week. This exposure converts to an intake of 53,000 µg/kg day. Because this was the "lowest effect level" rather than a "no-effect level", an acceptable intake was calculated by dividing the level by 1000. Thus the subchronic acceptable intake was calculated to be 53 µg/kg days and the AIC was 5×10^{-3} $(mg/kg/day)^{-1}$ (an additional 10-fold safety factor was added to correct for uncertainty involved in unstudied effects of extended exposures).

2.7 Chromium

Both maximum and average concentrations of soil chromium at the UOP site are in excess of background levels for salts of the metal for the State of New Jersey, according to draft risk assessment information from the Department of Environmental Protection. Potential exposures to chromium at the UOP site could thus be higher than ambient and will be assessed in the current report.

Chromium may exist in one of three oxidation states (Cr^{+2}, Cr^{+3}, or Cr^{+6}), as elemental chromium metal, or alloyed with other metals. Trivalent and hexavalent (Cr(VI)) chromium are predominant. Hexavalent chromium compounds such as chromic acid or chromate salts are substantially more toxic than trivalent compounds.

Although chromium was not speciated at the UOP site, a review of the literature indicated that the typical assumption that all chromium is chromium VI was not warranted. Instead the risk assessment assumes that 95% of total chromium is chromium (III) and that 5% of total chromium is chromium (VI). Justification for this assumption is provided in Appendix D which reviews the factors that affect the oxidation of chromium (III) to chromium (VI).

Chromium (VI) dusts and chromic acid are extremely irritating and have produced conjunctivitis, bronchitis, dermatitis, and ulcerations of eyes, respiratory tract, and skin. Ingestion of Cr(VI) has been reported to cause kidney toxicity and the effect has been reproduced in several experimental animal species.

Based on the drinking-water study of MacKenzie, et al (1958) in rats, the EPA set an AIC for ingestion for man at 5 µg/kg day. This value was derived by applying a 500 fold safety factor to the no-effect level of 2.5 mg/kg day observed by the investigators.

There is good epidemiological evidence that inhalation of certain Cr(VI) salts causes respiratory tract cancers. This issue is complicated, however, in that only relatively insoluble salts of Cr(VI) (e.g., $CaCrO_4$, $PbCrO_4$) are carcinogenic, while highly soluble Cr(VI) compounds are not. Carcinogenicity has not been demonstrated in man or animals exposed to chromium by other routes of exposure. It is possible that the distribution of inhaled Cr(VI) may differ from that of other routes of exposure. Chromium will be considered a carcinogen in inhalation exposures (but not ingestion) assessed in the present report. The potency slope for chromium is 41 $(mg/kg/day)^{-1}$, based on the studies of Mancuso (1975).

2.8 Cyanide

Cyanide levels in surface soil at the UOP site (34,800 µg/kg maximum, 2,430 µg/kg average) are in excess of National background according to NJDEP figures (background, 80 µg/kg). It is therefore necessary to assess the risk of potential exposures to cyanide in surface soil at the UOP site.

The term cyanides encompasses those inorganic or organic compounds which contain the -CN group. Examples include: cyanide ions that form complexes with metals, cyanates that

contain the -OCN radical, alkyl cyanates that trimerize to cyanurates, nitriles, and cyanohydrins. The toxicity of many of these substances is related to subsequent release of hydrogen cyanide (HCN) or the -CN radical. These components can be released as a result of photodecomposition, ionization, or dissociation (Dourdoroff, et al, 1966; EPA, 1980c).

Cyanides are used for a variety of applications. Cyanuric chloride based herbicides have experienced fast growth (Kirk-Othmer, 1978). Hydrogen cyanide (compressed gas) has been used a fumigant in ships, warehouses, and in greenhouses. Many industrial effluent wastes contain cyano-compounds including steel, plastics, synthetic fibers, and pharmaceutical and specialty chemicals, as well as the metallurgic industries (EPA, 1980c).

There are some naturally occurring substances that contain cyanide. Amygdalin and linamarin are examples of cyanogenic glycosides found in seeds of such plants as peaches, cherries, apples, and pears and in flax and lima beans, respectively. The starchy root of the cassava plant also contains a natural source of hydrogen cyanide.

The environmental fate and transport of cyanides will depend largely on their form. Cyanides are generally very water soluble. The mechanisms of loss in the aquatic environment are volatilization, microbial degradation, and sorption through particulate matter (EPA, 1985b). Sedimentation will occur with those substances that are less soluble.

The Ambient Water Quality Criterion calculated to protect saltwater aquatic organisms is as low as 1.0 µg/l (EPA, 1980c).

Cyanides are readily absorbed through the lungs, gastrointestinal tract and skin. Death from acute cyanide poisoning is the result of "cytotoxic anoxia", or cellular asphyxiation. It is one of the most rapidly acting toxins (Gilman, et al., 1980). Cyanide interferes with the iron component of cytochrome oxidase, a crucial terminal enzyme in the electron transport system.

The detoxification of cyanide is extremely efficient (Klaassen, et al., 1986). The extrapolated human detoxification rate has been calculated to be 0.017 mg/kg/minute (EPA, 1985b). As a result of the effective detoxification mechanism, chronic toxic effects of cyanides are rare. Many chronic studies have been performed in both rodents and dogs - all with negative findings (EPA, 1980c; EPA, 1985b). There do not appear to be any adverse health effects in rats resulting from long-term (2 years) low dose (76-190 mg/kg) cyanide exposure (Howard and Hanzal, 1955).

There are conflicting data regarding the teratogenicity of cyanides. Significant teratogenic effects observed in Golden Syrian hamsters in all concentration groups (78.5, 79.4, and 80.7 mg CN-/kg body weight/day) included increased fetal resorption and fetal abnormalities (Doherty, 1982). Tewe and Maner (1981a) designed an experiment with a low dose cassava meal (21 mg HCN/kg) before, during, and after pregnancy in order to study the effects of KCN on the reproductive performance of female Wistar rats. No significant differences were observed between the treated and the control groups. A similar study (Tewe and Maner 1981b) performed using pigs revealed similar negative results with the exception of significant differences found in fetal spleen-to-body and fetal heart-to-body ratios of the high-cyanide group (520.7 mg CN-/kg diet).

Noncarcinogenic effects have been quantified by the EPA (1985). The ten-day health advisory for a 10 kilogram child, drinking 1 liter of water per day is .16 µg CN/L. An uncertainty factor of 500 was used instead of the usual factor of 100 in order to account for the uncertainty involved in deriving a drinking water criterion from a dietary study. The same 10-day health advisory for an adult (weighing 70 kilograms and consuming 2 liters of water per day) is .560 µg CN/L. The same safety factor of 500 was applied. The lifetime health advisory is .750 µg/L. The acceptable daily intake (ADI) for a 70 kilogram adult was calculated to be 1500 µg CN-/day.

Although a safety factor of 500 was also applied in this derivation, it was for different reasons. First, an uncertainty of 100 was applied based on the National Academy of Sciences (NAS)/Office of Drinking Water (ODW) guidelines to accommodate the uncertainty of extrapolating an animal no-observed-effect-level (NOEL) for purposes of a human application. Then an additional factor of 5 was used to account for the dietary study to drinking water criterion conversion. This value (which translates to approximately 2×10^{-2} $(mg/kg/day)^{-1}$ has been accepted by EPA as the oral AIC. No inhalation value has been derived.

2.9 1,2-Diphenylhydrazine

1,2-Diphenylhydrazine is used and formed in several synthetic processes. It is used as the raw material in the manufacture of benzadine and in the production of other chemicals and dyes.

Very little toxicological information is available for 1,2-Diphenylhydrazine. Marhold et al. (1968) determined an oral LD_{50} value for male rats of 959 mg/kg. Chronic exposure to 1,2-Diphenylhydrazine has resulted in liver damage including carcinogenic tumors in mammals. The National Cancer Institute (NCI, 1978), after feeding rats and mice diphenylhydrazine for 78 weeks, found significant increases in heptacellular carcinoma and neoplastic nodules. In addition, Zymbal's gland squamous-cells or adrenal tumors were detected in male rats and neoplastic liver nodules or mammary carcinomas were found in female rats.

Based on this study, the USEPA has recognized diphenylhydrazine as a suspected human carcinogen. The USEPA has developed an ambient water quality criterion of 0.042 µg/l for an individual lifetime cancer risk of 10^{-6} (one cancer in one million people), based on a carcinogenic potency for humans of 0.8 $(mg/kg/day)^{-1}$ (USEPA, 1980). No other standards or guidelines for 1,2-diphenylhydrazine exposure have been developed.

2.10 Dichlorobenzene

There are three structural isomers of dichlorobenzene (DCB). 1,2-DCB, the Indicator Chemical chosen for the UOP site, is primarily used as a process solvent in the production of toluene diisocyanate and in the manufacture of dye-stuffs, herbicides, and degreasers (EPA, 1980d).

The high octanol/water coefficient of 1,2-DCB (log K_{ow} = 3.6) makes this substance lipophilic, lends the ability to cross biomembranes easily, and makes the compound likely to bioconcentrate in aquatic species. However, the bioconcentration factor for 1,2-DCB, is low at 89.

1,2-DCB has been classified as an eye and mucous membrane irritant, primary skin irritant, and a skin sensitizer (HAZARDLINE, 1987).

Varashavskaya (1967) determined the LD50 values for 1,2-DCB in a variety of laboratory animals. The target organs in these experiments appeared to be the liver, blood-forming system, the central nervous system (CNS), respiratory tract, and skin. At the highest dose of 1,2-DCB in a repeated dose study in rats, Hollingsworth (1958) found increased liver and kidney weights with some identifiable injury to the liver and decrease weight of the spleen, while at lower concentrations slight increases in liver and kidney weights were found. The highest no effect level in this experiment was 18,800 µg/kg day. The predominant subchronic effect reported by Varashavskaya (1967) was on the blood forming system. The highest non-detected-adverse-effect for 1,2-DCB was calculated by this author to be 1 µg/kg day. However, the EPA (1980) questioned this data because the end-points were not pathologic and there was little substantiation for the finding given in the report.

The 1988 update of SPHEM reports an oral AIC of $9x10^{-2}$ mg/kg/day for this compound and an inhalation AIC of $4x10^{-2}$ mg/kg/day. These AICs are used in this assessment.

2.11 Lead

Both the maximum and the average soil lead concentrations at the UOP site are higher than State and National background concentrations according to NJDEP information. Thus, the health risk of potential exposures to lead in soil at the UOP Site may be greater than ambient, and must be assessed.

Excessive or prolonged exposure to lead can cause both acute and chronic adverse health effects. Gastrointestinal colic and lead encephalopathy are the major acute systemic effects, while anemia, kidney disturbances, and neuromuscular dysfunction are characteristic of chronic exposure. Although chronic effects require repeated exposures, they generally occur at substantially lower doses than acute effects. Therefore, to develop the most protective limits, one must consider low-dose chronic effects.

Prolonged exposures to low levels of lead produce anemia. The anemic condition is due to the disruption of the enzyme systems involved in both the synethesis of hemoglobin and the maintenance of the integrity of the red blood cells. The lifespan of the circulating red blood cell is shortened, producing a microcytic (small cell), hypochromic (pale) anemia. To date, this sign appears to be the most sensitive and accurate indicator of lead intoxication. Subtle effects of lead on both the central and peripheral nervous systems have been reported. The velocity of electrical conduction in peripheral nerves is slowed by low concentrations of lead, but the mechanism of this effect is unknown. Low-level lead exposures in children have been reported to cause neurophyschological deficits, such as behavioral and delayed learning disorders (Needleman, et al. 1979), although such studies are controversial due to methodological issues related to measurement.

A problem arises in assessing lead exposures in that the toxic effects of lead are usually described as a function of blood lead content, rather than the conventional intake

levels. Algorithms have been developed that predict blood lead levels as a function of intake. This system is not compatible with the format for toxicity assessment developed in the SPHEM. The SPHEM suggests an oral AIC based on the level of intake that would occur from drinking water containing lead at the MCL (50 ug/l). Using standard assumptions concerning fluid ingestion and body weight the ingestion AIC is:

AIC = 50 ug/l X 2 L water consumed/day X 1/70 kg body weight x 1 mg/1000 ug = 1.4×10^{-3} mg/kg/day.

Likewise, an inhalation AIC may be derived from the Ambient Air Quality Standard for lead using standard assumptions:

AIC = 1.5 ug/m^3 X 20 m^3 air breathed/day X 1/70 kg body weight x 1 mg/1000 ug = 4.3×10^{-4} mg/kg/day.

These are not conventional AICs, and should only be viewed as screening values.

2.12 Mercury

Mercury has been used in the past for medicinal purposes: antiseptics, antisyphilitics, cathartics, and diuretics (Gosselin et al., 1984.) There are a number of occupations associated with mercury exposure, particularly through inhalation. These include mining, smelting, chloralkali production, and the manufacture of mercury-containing products such as batteries, measuring devices (thermometers) and paints. Mercury has also been used agriculturally as a seed and cereal protectant and as a fungicide.

Exposure to elemental (metallic) mercury causes behavioral effects and other nervous system damage. Inorganic mercury salts do not generally reach the brain, but will produce kidney damage. Divalent (mercuric) mercury is substantially more toxic in this regard than the monovalent (mercurous) form.

Organic mercury compounds are also toxic. The ionic forms of mercury can be methylated by microorganisms in detritus and sediments under bodies of water (Gosselin et al., 1984).

Acute mercury poisoning due to ingestion of ionizable mercurial salts begins with the corrosive nature of the compound. Cell death occurs immediately in the mouth and throat and then affects the tissues of the esophagus and stomach (Gosselin et al., 1984); pain and vomiting ensue. Death occurs within a few hours and is attributed to peripheral vascular collapse due to severe fluid and electrolyte losses (Gosselin et al., 1984). If death does not occur within a few hours, it can be delayed several days; this depends largely on the dose received. The kidneys are a target organ with tubular nephritis progressing to complete renal failure. Acute poisoning from inorganic mercury does not involve the central or peripheral nervous systems as does acute poisoning due to organic mercury or to chronic mercury (inorganic or organic).

The pharmokinetics and pharmacodynamics of mercury depends largely on its chemical form. Inhalation of elemental mercury vapor is problematic because it has such a high vapor pressure (18 mg/m^3 in a saturated atmosphere) (Klaassen et al., 1986). Preferential deposition occurs in the alveolar sacs based on the monoatomic state that is assumed by the vapor. The vapor is lipid-soluble, has increased retention time in the lung, and approximately 80% is absorbed by humans (Klaassen et al., 1986). This chemical form is <u>not</u> readily absorbed by the gastrointestinal tract. Organic mercury, however, is efficiently absorbed by the gastrointestinal tract based on its ability to traverse biological membranes. Distribution and metabolism of mercury are also dependent upon the chemical form. Both elemental and organic mercury degrade to divalent mercury, which is more toxic. The kidney is the target organ for the elemental form, whereas the central nervous system is the target organ for organic mercury.

Two widespread mercury poisonings associated with consuming tainted food have been reported. Methyl mercury bioaccumulated in fish of Minamata Bay in Japan after a typhoon

disturbed the Bay's bottom sediment in 1953 (Matsumoto et al., 1965). Consumption of contaminated fish by residents of Niigata and Minamata Bay, Japan caused 1,200 cases of Minamata disease including more than 100 fatalities (Tsubaki and Irukayama, 1977). Because methyl mercury can readily cross the placental barrier, the fetuses of many of the pregnant women suffered teratogenic effects or death (Matsumoto et al., 1965).

Another widespread methyl mercury poisoning occurred in Iraq when methyl-mercury-treated seed grains were used for bread flour and consumed. Clarkson et al. (1976) described 6,500 hospital admissions and 500 fatalities.

Symptoms of chronic mercury poisoning can be both neurological and psychological in nature as the central nervous system is the primary target organ. In cases of chronic exposure to organic mercury the route of entry does not influence the symptomology (Gosselin et al., 1984). Hand and finger tremors, slurred or scanning speech patterns, and drunken-stupor-like (atoxic) gait are some motor-control impairments that have been observed in chronic mercurial toxicity. Other neurological symptoms include visual disturbances. The peripheral nervous system may also be affected. A psychological syndrome known as erethism is known to occur (Gosselin et al., 1984); it is characterized by subtle or dramatic changes in behavior and personality including depression, fearfulness, restlessness, irritability, irascibility, timidity, indecision and early embarrassment. Advanced cases may also experience memory loss, hallucination, and mental deterioration.

There are acceptable intakes derived for both inorganic and organic mercury and compounds. The EPA has derived the same value for acceptable intake subchronic (AIS) and chronic (AIC) of 2.00×10^{-3} mg/kg/day for inorganic mercury. The inhalation-based AIS and AIC are 5.1×10^{-4} and 5.10×10^{-5} mg/kg/day for inorganic mercury, respectively. The oral AIS for organic mercury is 2.80×10^{-4} mg/kg/day, whereas the oral AIC is 3.00×10^{-4}. The inhalation AIS and AIC for organic mercury are both 1.00×10^{-4} mg/kg/day.

In a review of carcinogenic data for either inorganic mercury or methyl mercury, the EPA (1984) noted that none of the available data indicated "carcinogenic potential."

2.13 Nickel

Nickel in the ambient atmosphere typically exists as a constituent of suspended particulate matter (EPA 1985c). The greatest volume of nickel emitted into the atmosphere is the result of fossil fuel combustion (coal fired power stations for example). Other sources of nickel emissions are: primary production (nickel ore mining and smelting and nickel refining), incinerators, metallurgy (steel, nickel alloys and other smelters), chemical manufacturing, (nickel-cadmium batteries, and catalyst production), cement manufacturing, coke ovens, nickel recovery, asbestos mining/milling and cooling towers.

Ambient background levels of nickel in the atmosphere are very low (average of 0.008 $\mu g/m^3$). The predominant forms of airborne nickel appears to be nickel sulfate, complex oxides of nickel and other metals, nickel oxide, and to a much lesser extent, metallic nickel and nickel subsulfide. In ordinary circumstances, the contribution of ambient nickel in air to total nickel intake is negligible ranging from 1 µg per day via inhalation (in non-smokers) compared to 300 to 600 µg/day ingested in the diet and 3 to 15 µg/day inhaled as a result of smoking two packs of cigarettes per day.

Nickel occurs in soils both naturally and from man-made sources. Natural concentrations depend greatly on the elemental composition of rocks in the upper crust and range from 5 to 500 parts per million by weight (ppm) with an average of about 50 ppm. The most significant man-made sources are atmospheric deposition from smelting and refining operations, as discussed above, and direct application of sludge as both waste disposal and fertilizer. Nickel soil concentrations as much as 24,000 ppm by weight have been reported near metal refineries.

Nickel occurs in food by means of uptake via soils, particularly vegetables and by food processing. Processing can add nickel to food by leaching from nickel alloy-containing processing equipment and via flour milling and hydrogeneration of fats and oils using nickel catalysts.

The major adverse effects of nickel in humans are dermatitis, chemical pneumonitis, and lung and nasal cancers. These adverse effects occur under different circumstances and may be related to different nickel compounds

Nickel as a divalent ion will bind to proteins and nucleic acid and thus effect growth and enzyme action. This is particularly true for enzyme detoxification systems such as ATP-ase and the enzymes that mediate transmembrane transport. Nickel carconyl $Ni(CO)_4$ is a particularly toxic form of nickel and causes chest pain, dry coughing, hyperpnea, cyanosis, occasional gastrointestinal symptoms, sweating, visual disturbances and severe weakness. This is often followed by pulmonary hemorrhage, edema and cellular derangement, survivors may be left with pulmonary fibrosis.

In the work place, nickel dermatitis may result at high nickel concentrations. At lower concentrations some susceptible individuals develop eczema-like lesions. The threshold for these health effects are much greater than exposures which occur in the ambient environment.

Occupational studies on human exposure and animal studies indicate that certain nickel compounds appear to be carcinogens via inhalation. However, there is no evidence of carcinogenicity in mammals through ingestion or dermal exposure (EPA, 1985c).

An AIC exists for nickel for the oral route of exposure. The value is 2.00×10^{-2} mg/kg/day. The inhalation cancer potency factor, 1.19 $(mg/kg/day)^{-1}$, has been derived by the EPA. The EPA does not consider the oral route applicable to calculating cancer risks from the ingestion of elemental nickel.

2.14 PCBs

Information on human response to PCB exposure comes mainly from accounts of large scale unintended ingestion in Japan and Taiwan (Kuratsune et al., 1972; Hsu et al., 1985) and from data on occupationally exposed individuals (e.g., Smith et al., 1982). It should be noted that with this and all epidemiologic data, it is generally very difficult to separate toxic effects due to the compound being studied from those produced by contaminants also present.

Possible effects of reported PCB exposures include mucous membrane irritation (via the air exposure route), chloracne skin eruptions, hyperpigmentation of the skin, and abnormalities of the liver and immune system. These effects have been studied in laboratory animals, although the results have proven extremely variant among species (McConnell, 1985). Some animal bioassays have indicated that PCBs are carcinogenic, although others have failed to reveal this effect. Calandra (1975) found no cancers in rats treated with various Aroclors (100 ppm in the feed) for 24 months. The study done by the National Cancer Institute (NCI, 1978) revealed no increases in tumor incidence in rats fed 25, 50, or 100 ppm Aroclor 1254, and concluded that, under the conditions of the study, this mixture of PCBs could not be considered carcinogenic. However, Morgan et al. (1981) reevaluated the data and suggested that stomach tumors may have been elevated.

The animal study used by the EPA for determining risk to man is that of Kimbrough et al. (1975). In this study, female rats were given feed containing 100 ppm Aroclor 1260 or a control diet over a 21-month period (which represents approximately 80 percent of the animals' lifetime). Twenty-six of the 184 experimental animals were reported to have hepatocellular (liver) carcinoma versus 1 of 173 controls. Additional animals had neoplastic nodules, a lesion which may be a precancerous condition. Calandra (1975) reports that a separate pathologist's reevaluation of the Kimbrough data was

in disagreement with the evidence of carcinogenicity. These conflicts, coupled with equivocal findings in human clinical studies (both positive and negative findings have been made: see Bahn et al., 1977; Brown and Jones, 1981; Bertazzi, et al., 1981), indicate that there is a great deal of uncertainty concerning whether PCB produces cancer in man at all. However, the U.S. EPA argues that one positive animal study even in the face of negative studies is sufficient evidence to warrant the assessment of exposure to a compound as a possible human carcinogen. For the sake of conservatism, the potential carcinogenic response is addressed in this report.

The EPA has used the Kimbrough bioassay data in a model for cancer dose-response which presumes no threshold and linearity of response at low doses (EPA, 1980). There are aspects of the Kimbrough study that are notable for the present risk assessment. First, only one dose level (the dose was calculated to be 4.42 mg/kg body weight x day) was used. This means that the dose responsiveness of supposed PCB-induced carcinogenesis was not demonstrated in the study. This is a shortcoming of the study and probably contributes to uncertainty in the risk analysis. Second, the cancer incidence in the dosed animals was interpreted as 170/184, apparently because animals with neoplastic nodules were included in the animals considered positive for cancer. Thus, the risk estimate is not only for induced cancers but also for neoplastic nodules which may be precancerous states. Finally, this study was done on a different PCB mixture than was found at the UOP Site (Aroclor 1248). This adds uncertainty to the assessment but, as it has been suggested that lower chlorinated PCBs have demonstrated less, or no carcinogenicity relative to Aroclor 1260, using the CAG potency slope should be conservative (Kimbrough, 1987).

The upper 95% confidence bound on the slope of the dose-response line of the Aroclor 1260 data is 4.34 $(mg/kg/day)^{-1}$. No potency slope calculation has been made for PCB exposure by the inhalation route.

2.15 1,1,2,2-Tetrachloroethane

1,1,2,2-Tetrachloroethane, a chlorinated hydrocarbon, is produced in large quantities. It is a constituent of many commercial products, including paint, varnish, rust removers, weed killers and insecticides (Merck, 1983).

Because 1,1,2,2-tetrachloroethane has many industrial and commercial applications, numerous incidences of human exposure have been documented. Toxicological effects resulting from human exposure include dizziness, vomiting, malaise, headache, hand tremors, abdominal pain and death. Based on the toxicological information provided by animal studies to date, 1,1,2,2-tetrachloroethane is the second most toxic of the chrorinated ethanes; 1,2-dichloroethane is the most toxic of the chloroethanes studied (USEPA, 1980). Both acute and chronic exposures of 1,1,2,2-tetrachloroethane to animals have been studied. The results from a few of these studies are summarized below.

Smyth et al. (1969) determined an oral LD_{50} for rats to be 0.20 ml/kg. Acute inhalation exposures to 1,1,2,2-tetrachloroethane have produced anesthesia, fatty degeneration of the liver and tissue congestion and death in mice (Muller, 1932; Horiguchi, et al., 1962) and in rats (Horiguchi et al., 1962). Horiguchi et al. (1962) also observed increased vacuolization in the liver of monkeys after acute inhalation exposures. Intravenous or intraperitoneal injection of 1,1,2,2-tetrachloroethane was shown to cause weight loss, convulsions, fatty degeneration of the liver and kidney and death in guinea pigs (Muller, 1932).

Chronic inhalation exposures have also induced liver and kidney degeneration in rabbits (Navrotdkiy et al., 1971). Chronic exposure of rats and mice to 1,1,2,2-tetrachloroethane by gavage have resulted in an increased incidence of hepatocellular carcinoma in both male and female mice. The ambient water quality criterion for 1,1,2,2-tetrachloroethane is based on the results of a study on the effects of oral

exposure to female mice by the National Cancer Institute (NCI, 1978). This study also resulted in the induction of hepatocellular carcinoma.

The ambient water quality criterion for the ingestion of 1,1,2,2-tetrachloroethane contaminated water is 0.17 ug/l for a individual lifetime cancer risk of 10^{-6}, based on a cancer potency factor of 5.73×10^{-2} $(mg/kg/day)^{-1}$ (USEPA, 1980). However, the U.S. EPA uses a cancer potency factor of 2×10^{-1} $(mg/kg/day)^{-1}$ for risk characterization (U.S. EPA, 1986).

2.16 Toluene

Toluene (methylbenzene), an organic solvent formed during petroleum and coal tar distillation, is used in the manufacture of other chemicals and is found as a component of gasoline. In some media, toluene has short environmental half-lives. The air half-life is 1.3 days (Singh et al., 1981) and the water half-life is 4.1 hours (Macay and Yeun, 1983). It has a moderately low potential for adsorption and certain portions of spills may migrate into ground water (Wilson et al., 1981).

While toluene is a relatively common water contaminant, available studies have not indicated that it is highly toxic. Human studies have shown rapid absorption through the respiratory tract (Astrand et al., 1972). Gastrointestinal absorption information is limited to animal studies and is reported as relatively rapid (Pyykko et al., 1977). There is no available information on the oral exposure toxicity in humans, but very limited animal oral exposure studies indicate central nervous system (CNS) inhibition (Kimura et al., 1971).

Numerous human occupational studies of inhalation exposure to toluene have been done, and both acute and chronic exposures to varying air concentration of toluene have reported CNS toxicity (von Oettingen et al., 1942a,b,; Carpenter et al., 1944; Wilson, 1943; Munchinger, 1963; Hanninen et al., 1976).

Toluene is subject to abuse as a "recreational drug", and studies of chronic toluene abusers and occupational studies of chronically exposed workers have reported liver (Greenberg et al., 1942; Grabski, 1961) and renal function effects (Kroeger et al., 1980; Moss et al., 1980).

Pregnant animals exposed to toluene by ingestion and inhalation had decreased fetal weights (Hudak and Ungavry, 1978; Nawrot and Staples, 1979). Adequate data to evaluate the teratogenicity, mutagenicity or carcinogenicity are not available. Occupational standards have been set. U.S. EPA has not classified toluene as to carcinogenicity (Group D). The chronic oral risk reference dose (RfD) is 0.3 mg/kg/day (U.S. EPA, 1986) based on a study of inhalation exposure in rats. The chronic inhalation RfD is 1.0 mg/kg/day (PHRED, 1988).

2.17 Zinc

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis and carbohydrate metabolism. It is used in galvanizing processes. Ingestion of excessive amounts may cause fever, vomiting, stomach cramps and diarrhea. Metal-fume fever is caused by inhalation of zinc oxide fumes, but is not produced from zinc oxide dust. Contact with zinc salts can produce skin and eye irritation and inhalation of fumes, mists or dusts may irritate the respiratory and gastrointestinal tracts.

The EPA has calculated acceptable intakes for zinc and compounds. The acceptable intakes based on subchronic (AIS) and chronic (AIC) exposure are the same for the oral route of exposure: 2.1×10^{-1} mg/kg/day. The AIS for inhalation is 1.00×10^{-1} mg/kg/day, whereas the AIC for inhalation is 1.00×10^{-2} mg/kg/day. There are no data to support any carcinogenic effects. The EPA has designated zinc as a Group "D" compound, meaning not classified.

2.18 Summary

The carcinogen potency slopes and AIC values for Indicator Chemicals at the UOP Site, derived as described in this chapter, are compiled in Table 2-1.

TABLE 2-1

DOSE-RESPONSE VALUES FOR INDICATOR CHEMICALS

UOP SITE, EAST RUTHERFORD, NJ

Compound	CARCINOGENIC ASSESSMENT Potency Slope-Ingestion $(mg/kg\ day)^{-1}$	CARCINOGENIC ASSESSMENT Potency Slope-Inhalation $(mg/kg\ day)^{-1}$	OTHER TOXIC EFFECTS AIC-Ingestion (mg/kg day)	OTHER TOXIC EFFECTS AIC-Inhalation (mg/kg day)
Arsenic	$1.5x10^{0}$	$1.8x10^{0}$	$1x10^{-3}$	----
Benzene	$2.9x10^{-2}$	$2.9x10^{-2}$	----	----
BEHP	$6.84x10^{-3}$	----	$2x10^{-2}$	----
Carcinogenic PAH	$1.15x10^{+1}$	$6.11x10^{0}$	----	----
Cadmium	----	$6.1x10^{0}$	$1x10^{-3}$(food)	----
Chromium (III)	----	----	$1x10^{+0}$	$5.1x10^{-3}$
Chromium (VI)	----	$4.1x10^{+1}$	$5x10^{-3}$	----
PCB	$4.34x10^{+0}$	----	----	----
MCB	----	----	$2.7x10^{-2}$	$5.7x10^{-3}$
Cyanide	----	----	$2x10^{-2}$	----
1,2 DCB	----	----	$9x10^{-2}$	$4x10^{-2}$
Lead	----	----	$1.4x10^{-3}$	$4.3x10^{-4}$
1,2-Diphenylhydrazine	$8x10^{-1}$	$8x10^{-1}$	----	----
1,1,2,2 Tetrachloroethane	$2x10^{-1}$	$2x10^{-1}$	----	----
Mercury(a)	----	----	$2x10^{-3}$	----
Nickel	----	$1.19x10^{0}$	$2x10^{-2}$	$5.1x10^{-5}$
Toluene	----	----	$3x10^{-1}$	$1x10^{+0}$
Zinc	----	----	$2x10^{-1}$	$1x10^{-2}$

(a) AIC for mercury is for inorganic mercury.

3. IDENTIFICATION AND DEVELOPMENT OF EXPOSURE PATHWAYS

At any site humans may potentially be exposed to contaminants in air, water or solid media (soils, sediments or sludges); directly, or through the food chain. The route of intake may be by ingestion, inhalation, or dermal absorption. The following discussion indicates the direct exposure pathways pertinent for the UOP Site. Indirect pathways (food chain) will be evaluated in the ecological risk assessment.

3.1 Air

3.1.1 Volatile Emissions

The volatility of certain Indicator Chemicals at the UOP Site may make them available for exposure by the air route. Initially, two volatile compounds, benzene and chlorobenzene, which were found in surface soils at the UOP site were assessed. The NJDEP directed that 1,2-dichlorobenzene and toluene also be included in the risk assessment. However, "surface soils" at the UOP site were measured from 0 to 2 feet. The volatility of benzene and chlorobenzene and the other two compounds are such that it is unlikely they are truly present at the surface in any great concentration. Exposure via this route is estimated in Appendix C and is found to have extremely low levels of risk associated with all four compounds. Therefore, volatile emissions from soils need not be considered in the body of this assessment.

Volatile compounds have been detected in ground water. As ground water discharges to the surface at the various stream channels on site, volatilization can occur. Calculations of volatile emissions from the surface of soil or water bodies at several other sites with circumstances similar to the UOP Site show that dispersion and dilution processes make the off-site impact of volatilized material negligible. Consistent with this observation, the air monitoring performed during the

investigations at the UOP Site indicate that VOC concentrations in the air are very low (described in Section 5 of the RI). However, trespassers or visitors to the site as it currently exists may be exposed to relatively undiluted emissions. Similar exposures might occur in employees or visitors to businesses located at the site in the future. Health risks from this air pathway will be assessed for the subset of the population who are present on the UOP Site. The procedure and calculation used to estimate concentrations of volatiles in air following release from stream channels is described in Appendix A.

3.1.2 Particulate Emissions

For less volatile materials in surface soils, it is possible that entrainment might occur, such that individuals might be exposed by inhaling contaminated particulates. Again, it is likely that dispersion as well as sedimentation would make the health impact of entrained material insignificant off-site. However, like volatile emissions, this air pathway will need to be addressed for individuals who are present, now or in the future, on the UOP Site.

3.2 Surface Water

Persons may be exposed to surface waters as well as sediments in Area 4. The exposure pathways and potential risks associated with such exposures are presented in Appendix B.

3.3 Ground Water

Section 3.1 describes the exposure potential for ground water that discharges into the stream channels. The only other potential ground-water exposures are by direct contact with or consumption of contaminants in a potable ground-water supply. These are unlikely exposures for the UOP Site due to various

factors which include: high salinity and low-permeability of the contaminated shallow-aquifer, and easy access to the municipal water supply.

Table 3-1 shows salinity concentrations (NaCl) that are computed from a number of specific conductivity readings taken during the Phrase II Investigation. These salinity values range from 700 to 5700 mg/L for conductivity readings from the shallow aquifer (wells designated S and I). The salinity value in the deep wells (3D and 7D), at 300 mg/L is much lower than values found in the shallow aquifer. The most critical health risk component of sodium chloride is sodium. The MCL for sodium because of conflicting evidence surrounding its health effect (production of hypertension and other cardiovascular effects) is given as a range: 20 to 250 mg/L which corresponds to a range of 50 to 640 mg/L of sodium chloride. The shallow aquifer salinity values quoted above (700 to 5700 mg/L) exceed the high end of the MCL range; which renders the shallow aquifer an unsuitable potable water source. The deep aquifer salinity value falls within the range which makes it a more attractive although not ideal source of potable water.

Since the deep aquifer salinity is above the lower MCL limit of 50 mg/L, it is highly probable that future potable supply needs would be met by simply tapping into the municipal water supply.

In addition to the salinity problems, the low permeability of the shallow aquifer reported in the Remedial Investigation Report is a severe hindrance to its use as a water supply. The preference for using the deep aquifer (as a non-potable source) is demonstrated historically by the production wells which were used during plant operation and were all screened in the deep aquifer.

The shallow aquifer which contains contaminants at the site is not and will not be used as a potable water supply because of its high salinity, contributed from nearby saline surface waters. Measurements taken nearby from Berry's Creek by the Hackensack Meadowlands Development Commission during the

TABLE 3-1

GROUND-WATER SALINITY

Well	Specific Conductance (µmhos/cm)	Salinity* (mg/L)
1S	7500	5700
2S	2800	2000
2I	2010	1400
3S	1450	1000
3I	2500	1800
3D	450	300
4I	1200	900
5I	1250	900
6I	950	700
7S	3750	2700
7I	2500	1800
7D	350	300
8I	2250	1600
9I	3500	2500
10I	1100	800
11I	1850	1300
MW3	2000	1400
MW17	2500	1800

*Conductivity to Salinity Conversion is based on: Tiphane and St. Pierre, 1962, assuming a ground-water temperature of 12°C.

Source: Specific Conductivity Readings taken from: Phase II Investigation, Water and Soil Conditions, UOP Site E. Rutherford, NJ, May 1985 by Geraghty & Miller, Inc.

summers of 1983 through 1986 yielded an average salinity concentration of 4.4 parts per thousand. Water with a value above 3.5 parts per thousand is considered saline.

An additional consideration is the possibility that the shallow aquifer could contaminate the deep aquifer which is used several miles away from the site as a potable water supply. Section 4.5.3, "Site Hydrogeology", of the RI Report provides a lengthy explanation as to why water from the upper aquifer does not flow to the deep aquifer. The reasons are principally the presence of an upward hydraulic gradient and a thick impermeable clay layer between the two aquifers.

In conclusion, no exposure pathway exists for direct contact with or consumption of ground water.

3.4 Soils

The UOP Site is a flat, unused area, that is covered in parts by scrub brush, former building foundations, and dense Phragmites stands in the salt marsh area of the property. There are, however, some unpaved roadways and areas of unvegetated surface. The relative extent of these areas is apparent in Figure 3-1. It may be possible for individuals present at the site to make direct contact with surface soils in the limited area that is without barriers. Health risks from this pathway will be assessed for the subset of the population who visit or trespass on the UOP site currently or in making future use of the property. The additional exposure pathway for soils, entrainment of material from the surface and subsequent inhalation, has been described previously.

No exposure pathway currently exists for subsurface soils. In the event soils are disturbed during remediation or future construction at the site, this situation may change.

Southwest View

South View

Figure 3-1 Condition of Surface at the UOP Site
East Rutherford, NJ

3.5 Food Chain

Several of the constituents present in water and sediments at the UOP Site may be taken up by aquatic biota that could live in stream channels or Berry's Creek. It does not seem likely that any of these organisms would be taken for human consumption in the current condition of the water system. The potential for indirect exposure to contaminants via the food chain will be addressed in the ecological risk assessment.

3.6 Summary

Potential exposure pathways for the UOP Site are listed in Table 3-2. They are, in summary, inhalation of volatilized material from ground water discharging into stream channels, inhalation of entrained materials from surface soils, and intake of materials in soils with which direct contact might be made.

TABLE 3-2

EXPOSURE ASSESSMENT

UOP-SITE EAST RUTHERFORD, NJ

Media	Exposure Pathway	Exposure Point	Indicator Chemcials of Concern
Air	Inhalation of volatiles	Near stream channels	Benzene, MCB, Toluene, 1,2,-diphenylhydrazine, 1,1,2,2-tetrachloroethane
	Inhalation of entrained soils	Non-vegetated, unpaved areas	BEPH, PAH Chromium, PCB, Arsenic, Mercury, cyanide, 1,2-DCB, Lead, Zinc, Cadmium
Ground Water	Source of airborne volatilies	Stream channels	See air, volatiles
Soils	Ingestion	Soils	BEHP, PAH Chromium, PCB, Arsenic, Mercury, Cyanide, 1,2-DCB, Lead, Zinc, Cadmium
	Source of entrained materials	Non-vegetated, unpaved	See air, entrained soils

4. IDENTIFICATION AND CHARACTERIZATION OF POTENTIALLY EXPOSED POPULATIONS

4.1 Land Use

4.1.1 Current Land Use

The UOP Site is currently unused and bounded by commercial and industrial property, marshland, and a busy thoroughfare (Route 17). Approximately one-half mile to the west of Route 17, there is a residential area, and Henry P. Becton High School. The marshland portion of the site, to the east, has dense stands of Phragmites and typical marshland understory. Sixty-five bird species and several mammals and amphibians have been sited in the meadowlands area in the vicinity of the site (Geraghty & Miller, 1987).

The remainder of the site is discontinuously covered with building foundations, scrub-brush and aged blacktop roadways. Some unvegetated areas and unpaved roadways also exist. The extent of surface cover is depicted in photographs in Figure 3-1. There is evidence that individuals, perhaps youngsters, have been visiting the site. It appears that the roadways on the property have been used for motorcycling.

The UOP Site is drained by several stream channels (Area 4) that empty to Berry's Creek, a tributary of the Hackensack River. The surface water is an estuarine system. Berry's Creek currently appears to be a stressed ecological system.

4.1.2 Future Land Use

Future uses of the UOP Site are likely to be consistent with current land uses in the area. The site is part of a well defined area that, because of location, access and zoning, is generally used for similar types of activities throughout. This area is bounded by the following features: Paterson-Plank Road to the north, Route 17 to the west, Berry's Creek to the east, and Route 3 to the south.

Developed property in this area is predominantly occupied by large warehouses and small manufacturing facilities. Typically, a property has one or more buildings, large paved areas and in some instances lawns. The use of these properties is consistent with normal industrial uses in which the facilities are occupied by an adult work force during normal work hours and access by unauthorized individuals is controlled by either fences and/or security personnel.

A high percentage of the properties along Route 17 and Paterson-Plank Road are used by commercial retail businesses. Examples of these uses are: gasoline stations, a building supply store, an automobile dealership, a hotel, office parks, and restaurants. These land uses are characterized by buildings, large paved areas and often lawns in the case of restaurants and hotels. These uses have a large number of people who visit the site occasionally and an adult work force that is present continuously during normal business hours.

The above land uses are driven largely by zoning regulations. The site area west of the railroad tracks is within East Rutherford's jurisdiction and is zoned: I-2, General Industry and Business. The site area east of the railroad tracks is within the HMDC's jurisdiction and is zoned: Light Industrial, A. Communication with the HMDC (Nierstedt, 1987) regarding future development of the area reveals a strong HMDC commitment toward consistent zoning. Their policy is to allow special exception uses such as hotels and restaurants along Paterson-Plank Road and to ensure light industrial uses and office parks south of Paterson-Plank along Murray Hill Parkway. The HMDC is forceful in applying its policy; having recently disallowed a proposed shopping center along Paterson-Plank Road because of projected traffic congestion problems.

Discussions with private developers who are active in the general area indicate that a mixed use of the UOP property would be most economically advantageous. Mixed use would include possibly hotels and restaurants along Route 17 and

either warehousing or office use along Murray Hill Parkway; although office use may be more attractive than warehousing.

Undeveloped portions of the area generally are marshes that are wet and at low elevations. These areas usually remain as marshes due to regulatory obstacles to filling them for use as developable land. These areas are predominantly thickly vegetated, usually with marsh grass (phragmites). Access to these undeveloped areas is usually not restricted.

There are no residential properties in the area to the east of Route 17. Population trends in East Rutherford show recent declines: 1960-1970, 10% increase; 1970 to 1980, 8% decrease. In spite of presumed rapid growth in northern New Jersey, East Rutherford's population growth continues to be stagnant. The following population figures for East Rutherford were obtained from the Bergen County Department of Planning and Economic Development:

Year	Population
1980	7849
1987	7865

These figures for East Rutherford show negligible population growth in the 1980's. Furthermore, the UOP Site is located in an industrial area, is surrounded by wetlands, and there is no evidence of residential growth in the vicinity of the site. Future residential use is extremely unlikely and to evaluate such a scenario would be unrealistic and inappropriate. A possible action by UOP, as suggested by the NJDEP, would be to use a deed notification which summarizes the industrial practices at the site, the contamination, and the remediation that is proposed/implemented at the site. For these reasons, a residential site use scenario will not be incorporated into the Risk Assessment.

The UOP property could be developed for any of the uses described above (except residential) because: some of the

property borders Route 17 where retail businesses abound, much of the rest of the property is typical of properties that have warehousing and manufacturing facilities. Zoning encourages these uses and they are economically the most advantageous. The area between Murray Hill Parkway and Berry's Creek is predominantly marsh land and is expected to remain that way.

Berry's Creek is rated as Class FW2-NT/SE2 indicating that the waters should be capable of maintaining fish and other wildlife populations.

4.2 Potentially Exposed Populations

4.2.1 Off-Site Exposure - Current Site Use

It has previously been discussed (Section 3.1.1) that dispersion, dilution, and sedimentation of volatilized or entrained materials from the UOP Site would tend to minimize off-site impact of materials currently present at the UOP property. Ground water at the site is isolated from any useable aquifer. Thus, Areas 1, 1A, 2 and 5 of the UOP Site are unlikely to be causing any significant off-site impact in their current condition.

4.2.2 On-Site Exposure - Current Site Use

From the previous description of land use, it is apparent that only a subpopulation of the area inhabitants have potential for exposure to materials at the UOP Site. These would be individuals who occasionally trespass or legitimately visit the site. Of primary concern within this population is the possibility that young people frequent the area. The reasons for these concerns are:

- Young people may have a greater proclivity than adults for direct contact with surface materials at the site.

- Because young people are smaller, they may derive a greater body burden, on a per kilogram body weight basis, than adults when subjected to equivalent total exposures.

The exposure and risk assessment must therefore address the potential activities of young people who may trespass on the UOP Site. Beyond assessing the risk to these individuals, a properly designed exposure scenario for young people would also preclude the necessity for assessing the impact on other individuals involved in activities at the site. That is, adults with less contact with surface materials would be expected to derive less health risk from the constituents at the site. Thus, if remediation is designed to protect against the risks calculated for young people under the current site use scenario, it will be adequate for other visitors.

4.2.3 Off-Site Exposure - Future Site Use

It is conceivable that a recreational fishery might one day occur, if Berry's Creek is reclaimed. It is therefore pertinent to assess the risk of ingestion of aquatic biota that may take up Indicator Chemicals from the surface water or sediments at the site. The population presumed to be subject to this type of exposure would be anyone fishing in Berry's Creek. This route of exposure will be addressed in the Environmental Risk Assessment which is being performed for the site.

4.2.4 On-Site Exposure - Future Site Use

Potential future-use exposure scenarios that included residential, recreational or commercial use of the UOP property were considered in this risk assessment. Based on the research detailed in Section 4.1.2, residential and recreational uses were dismissed as highly improbable. Use of the property for

retail, warehousing, or office space is very likely and risks to humans occupying these facilities must be addressed. Probable receptors for this type of site use would be employees and visitors (customers). By virtue of their consistent, prolonged presence at the site, employee exposure is the most appropriate scenario to assess. Workforce populations are generally adult males and females.

In addition, the NJDEP has requested that a construction worker scenario be assessed. Therefore, a construction worker population will also be addressed, and a one-year facility construction project will be assumed.

5. ESTIMATION OF ENVIRONMENTAL CONCENTRATIONS

5.1 Air

5.1.1 Present Site Use

As mentioned in Chapter 3, contaminants could occur in the air as a result of entrainment of constituents that are present in surficial soils or by volatilization of constituents from surface water bodies at the site.

Because the site is relatively well covered with vegetation or foundations and pavement, levels of wind blown particulate should not be inordinate. However, there is evidence that motorcycles have been used at the property. Entrainment by motor vehicles is therefore a possibility. The subjects exposed to entrained materials, further, would be the individuals riding the vehicles that produced it. Therefore, a fairly dusty atmosphere should be presumed as part of a prudent exposure assessment. It should be cautioned that this may not be a prevalent condition of the site. Rather, it is a credible worst case situation. For the purposes of this report, a particulate concentration of 1,000 ug per cubic meter of air is presumed. This is a visibly dusty atmosphere and is in excess of entrainment concentrations used in similar soil exposure scenarios (Eschenroder, et al, 1986 - a higher value was used here because Eschenroder was considering a slower moving, albeit heavier vehicle, a tractor, causing entrainment). If it is presumed that the entire particulate concentration is from surface materials, the Indicator Chemical concentration in air would be:

Air Concentration (mg constituent/M^3) =
Soil concentration (mg constituent/kg soil)
x Dust Concentration (1000 µg soil/M^3)
x Correction Factor (10^{-9} kg/µg soil)

Because entrainment is expected to come from a large area of the site, the average soil concentrations are used in this calculation. Maximum concentrations are not appropriate for soil inhalation, since fugitive dust is not generated from soil in one spot, but from a large area. Therefore it is appropriate to use an average concentration that incorporates the large area. Ambient concentration of compounds calculated in this way are given in Table 5-1.

Volatile constituents may also be in the air as a result of emission from the surface of water body channels at the site. Concentrations of volatile Indicator Chemicals will be vastly different depending on the momentary meteorology, tide cycle (tides dilute and flush the compounds in surface water every six hours), and location of the receptor on the site. The risk assessment used a tiered approach to evaluate risks associated with volatile emissions from stream channels. The first tier used a "worst-case" screening model and the second tier uses a more refined and realistic model. If the screening model indicates that no potential for unacceptable adverse health exists, then further, more detailed modeling is not necessary. On the other hand, if the screening model predicts higher than allowable adverse health risks, than the refined model should be employed.

The screening model in this case assumed that the worst case situation is for an individual to be near the lengthwise "end" of a stream channel as the wind blows at low rates directly along the channel with stable meteorologic conditions prevailing during low tide. This maximizes the concentration of constituents in the source (thus maximizing emissions), maximizes the source size, and minimizes dilution due to wind and stability conditions. Again, the probability of this situation actually occurring, particularly with an individual present, has not been calculated but is believed to be very low.

Because potentially higher than allowable adverse health effects were predicted by the screening model, the risk

TABLE 5-1

INDICATOR CHEMICAL CONCENTRATIONS

FOR USE IN PRESENT SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

Compound	Surface Soil Conc-Avg[a] (mg/kg)	Surface Soil Conc-Max[a] (mg/kg)	Air Conc[b] (mg/m3)
Benzene	a	a	5.52×10^{-6} (v)
BEHP	1.79	17	1.79×10^{-6} (p)
Carcinogenic PAH	3.87	80.6	3.87×10^{-6} (p)
Chromium (III)	132	2740	1.32×10^{-4} (p)
Chromium (VI)	6.9	144	6.93×10^{-6} (p)
PCB	21.4	480	2.14×10^{-6} (p)
MCB	a	a	3.31×10^{-6} (v)
Cyanides	2.42	34.8	2.42×10^{-6} (p)
1,2 -DCB	16.40	550	1.64×10^{-5} (p)
Lead	238	1820	2.38×10^{-4} (p)
Mercury	2.48	10.0	2.48×10^{-6} (p)
Zinc	198	1530	1.98×10^{-4} (p)
Cadmium	1.38	16.0	1.38×10^{-6} (p)
Arsenic	c	18.0	c
Toluene	a	a	2.52×10^{-5} (v)
1,2,-Diphenylhydrazine	ND	ND	3.44×10^{-7} (v)
1,1,2,2-Tetrachloroethane	a	a	5.99×10^{-7} (v)

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a. The volatility of benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane, and MCB makes it unlikely that they are present in undisturbed surface soils. See Appendix C for detailed explanation.

b. Volatile concentrations (denoted by "v") calculated from the ISC model detailed in Appendix A. "p" is particulate

c. Arsenic levels were not above background at average concentrations found in the soil; they will only be considered at maximum levels.

assessment uses a more refined model that assumes an individual on the site is exposed to the maximum annual acreage concentration of volatile compounds resulting from emissions from the most contaminated segment of the stream channels. The person on the site, be that a tresspasser under current conditions or a worker under future conditions, is assumed to be situated at a distance of 15 meters from the edge of the stream channel. A description of a simplified screening "line source" model and the more detailed industrial source complex (ISC) model used to generate air concentration for this risk assessment is detailed in Appendix A. Concentration calculated using the ISC model are given in Table 5-1.

5.1.2 Future Site Use

Entrainment of contaminants for an office building or shop would be less of a problem than for the assumed present site condition. It is likely that much of the area would be covered by building foundations or paved parking or storage areas. Further, the human receptors in an office or shop would be indoors a large majority of the time. As a result, a less dusty atmosphere is assumed for the future site use scenario. The Ambient Air Quality Standard for Total Suspended Particulate (TSP) of 75 $\mu g/M^3$ would be a reasonable value. A variety of authors have measured or estimated, based on models, the protection against air contaminants afforded by being indoors (Eschenroder, et al, 1986; Roberts, et al, 1974; Sterling and Kobayashi, 1977). A fairly consistent ratio of concentration of contaminants in indoor dust vs. outside soil is 75%. This value will be used in the current risk assessment by assuming that outdoor dust is entirely comprised of surface soil, and taking 75% of that concentration as the indoor concentration. Thus, the air concentration of Indicator Chemicals in indoor air is:

Air Concentration (mg/M^3) =

Soil concentration (mg constituent/kg soil)
* 75 ug soil/M^3
* correction factor (10^{-9} kg/µg soil) * 0.75

Volatile emissions from the stream channels are estimated using the ISC model and assuming a receptor is located at the point 15 meters from the most contaminated channel with the maximum annual average concentration.

Particulate and volatile air contaminant concentrations for the future site use scenario are given in Table 5-2.

5.1.3 Construction Worker Scenario

For the construction worker scenario, separate assessments are performed for a 2-month excavation period and a 10-month construction period. For the a two-month excavation period, it is assumed that for particulates the entire particulate concentration is from surface and subsurface soils (weighted average), and the following equation was used to derive the air concentration:

Air concentration (µg constituent/m^3) =

Soil concentration (µg constituent/g soil)
x Dust concentration (1,000 µg soil/m^3)
x Correction Factor (10^{-6}g soil/µg soil)
x 0.75 (particulates of respirable size, see Section 7.1.1)

Volatile emissions from the sub-surface soils for this scenario are estimated using Model V, from Lyman et al., 1982 (see Appendix A, Calculation 2). For the remaining 10 months, surface soil concentrations are used, along with the following equation for particulates:

TABLE 5-2

INDICATOR CHEMICAL CONCENTRATIONS

FUTURE SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

Compound	Indoor Dust Concentration[a,c] (mg/kg)	Air Concentration[b] (mg/M3)
Benzene	c	5.52×10^{-6} (v)
BEHP	1.43	1.01×10^{-7} (p)
Carcinogenic PAH	3.10	2.18×10^{-7} (p)
Chromium (III)	105	7.41×10^{-6} (p)
Chromium (VI)	5.5	3.90×10^{-7} (p)
PCB	17.1	1.20×10^{-6} (p)
MCB	c	3.31×10^{-6} (v)
Cyanides	1.94	1.36×10^{-7} (p)
1,2-DCB	13.1	9.22×10^{-7} (p)
Lead	190	1.34×10^{-5} (p)
Mercury	1.98	1.39×10^{-7} (p)
Zinc	158	1.11×10^{-5} (p)
Cadmium	1.11	7.78×10^{-8} (p)
Arsenic	d	d
Toluene	c	2.52×10^{-5} (v)
1,2-Diphenylhydrazine	c	3.44×10^{-7} (v)
1,1,2,2-Tetrachloroethane	c	5.99×10^{-7} (v)

a. Assumed to be 80% of average outdoor soil concentration.

b. Particulate (p) contaminant or volatile (v) contaminant.

c. The volatility of benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane, and MCB makes it unlikely that they are present in surface soils.

d. Average soil arsenic levels were not above background; it will only be considered at maximum levels.

Air concentration (µg constituent/m^3)

Soil concentration (µg concentration/kg soil)
x Dust concentration (75 µg soil/m^3)
x Correction factor (10^{-6} g/µg soil)
x 0.75 (particulates of respirable size) (see Section 7.1.2)

Volatile air emissions for the 10-month scenario are estimated as in the future scenario. Air contaminant concentrations for the construction worker scenario are given in Tables 5-3 and 5-4.

5.2 Water

As mentioned in Chapter 3, surface water is directly relevant to environmental or human health impact. Exposure to surface water by humans is evaluated in Appendix B, and to biota in the ecological risk assessment.

5.3 Soil

5.3.1 Current Site Use

Surface soil concentrations are based on analytical data presented in the Remedial Investigation. Maximum and arithmetic means of soil concentrations are given in Table 5-1.

5.3.2 Future Site Use

The concentration of Indicator Chemicals in settled indoor dust is assumed to be approximately 80% of the average outdoor value. This value, theorized to be due to tracking of outdoor soil into a residence or business, has been used by Hawley (1985), although it is not clear how this author derived such a value. Nonetheless, given the similarity of this value to the

TABLE 5-3

INDICATOR CHEMICAL CONCENTRATIONS

FOR USE IN CONSTRUCTION WORKER SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

(10 MONTH EXPOSURE TO SURFACE SOILS)

Compound	Soil Conc.[a] Avg (mg/kg)	Soil Conc.[a] Max (mg/kg)	Air Conc.[b] (mg/kg)
Arsenic	c	18	c
Benzene	a	a	5.00×10^{-6} (v)
BEHP	1.79	17	1.01×10^{-7} (p)
Car. PAH	3.87	80.6	2.18×10^{-7} (p)
Cd	1.38	16	7.76×10^{-8} (p)
Cr (III)	131.67	2,736	7.41×10^{-6} (p)
Cr (IV)	6.93	144	3.90×10^{-7} (p)
PCB	21.39	480	1.20×10^{-6} (p)
MCB	a	a	3.30×10^{-6} (v)
Cyanide	2.43	34.8	1.37×10^{-7} (p)
1,2-DCB	16.40	550	9.22×10^{-7} (p)
Lead	238.0	1,820	1.34×10^{-5} (p)
1,1,2,2	a	a	6.00×10^{-7} (v)
Hg	2.48	10	1.39×10^{-7} (p)
Toluene	a	a	2.50×10^{-5} (v)
Zn	197.74	1,530	1.11×10^{-5} (p)

a. The volatility of benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane, and MCB makes it unlikely that they are present in undisturbed surface soils.

b. Volatile concentrations (denoted by "v") calculated from the ISC model detailed in Appendix A. "p" is particulate

c. Average soil arsenic levels were not above background; it will only be considered at maximum levels.

TABLE 5-4

INDICATOR CHEMICAL CONCENTRATIONS

FOR USE IN CONSTRUCTION WORKER SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

(2 MONTH EXPOSURE TO SURFACE & SUBSURFACE SOILS)

Compound	Soil Conc.[a] Avg (mg/kg)	Soil Conc.[a] Max (mg/kg)	Air Conc.[b] (mg/kg)
Arsenic	c	2,258	c
Benzene	0.47	12.76	1.20×10^{-3} (v)
BEHP	8.75	232.30	6.56×10^{-6} (p)
Car. PAH	1.49	32.49	1.12×10^{-6} (p)
Cd	0.51	8.43	3.84×10^{-7} (p)
Cr (III)	271.42	4,766.63	2.04×10^{-4} (p)
Cr (VI)	14.29	250.88	1.07×10^{-5} (p)
PCB	8.12	168.59	6.09×10^{-6} (p)
MCB	1.21	37.67	4.82×10^{-4} (p)
Cn	1.66	30.06	1.25×10^{-6} (v)
1,2-DCB	5.05	169.86	3.79×10^{-6} (p)
Pb	204.85	1,421.39	1.54×10^{-4} (p)
1,1,2,2	0.06	3.2	2.56×10^{-4} (v)
Hg	5.63	89.8	4.22×10^{-6} (p)
Toluene	37.48	1,383.33	1.99×10^{-3} (v)
Zn	264.74	2,719.04	1.99×10^{-4} (p)

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a. The volatility of benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane, and MCB makes it unlikely that they are present in undisturbed surface soils.

b. Volatile concentrations (denoted by "v") calculated from the ISC model detailed in Appendix A. "p" is particulate.

c. Average soil arsenic levels were not above background; it will only be considered at maximum levels.

ratio of indoor suspended dust vs. outdoor soil concentration and findings of other authors (TerHaar and Aronow, 1974) that soil and (unsuspended) house dust contaminants are often of similar concentration, the value will be used for the present assessment. Calculated concentrations are given in Table 5-2.

5.3.3 Construction Worker Scenario

Contaminant concentrations for the two month excavation period are a weighted average of surface and subsurface concentrations, based on analytical data presented in the RI. Concentrations for the remaining 10 months are from surface soils only. Maximum and arithmetic means of these concentrations are presented in Tables 5-3 and 5-4.

6. COMPARISON OF ENVIRONMENTAL CONCENTRATION TO RELEVANT AND APPLICABLE STANDARDS

Contaminants in the areas of the UOP site covered by this risk assessment are potentially present in air, soils, and ground water. A majority of the constituents that may be present in air have no criteria for permissible levels. There is, however an Ambient Air Quality Standard for lead. This value is 1.5 ug of lead per cubic meter of air. The predicted concentration of lead from the UOP site in air is an order of magnitude lower in concentration (0.07 ug/M^3, Table 5-1). There are no relevant or applicable standards for permissible concentrations of contaminants in soils, however New Jersey does have a set of non-promulgated soil cleanup objectives. For purposes of comparison, the soil cleanup objectives are tabulated with the indicator chemical soil concentrations in Table 6-1.

Ground water criteria are contained in NJAC 7:9-6.6. According to the text of NJAC 7:9 - 6.5, when these criteria are exceeded, a review process (incorporating an assessment of health and safety) is initiated to determine if groundwater restoration to NJAC 7:9-6.6 criteria levels is required. The GW-3 criteria and the indicator chemical concentration in ground water are presented in Table 6-2 for comparison purposes.

Based on total dissolved solids (TDS) estimates which could be inferred from conductivity readings in Table 3-1, the likely designation for the ground water in the shallow aquifer is GW-3 with TDS range of 500 to 10,000 mg/L (NJAC 7:9-6.6).

Given that NJAC 7:9-6.5 requires a health risk assessment of contaminated ground water and also that there are no relevant and applicable standards for air and soils, the significance of contamination will be assessed using standard health risk analysis procedures.

TABLE 6-1

COMPARISON OF INDICATOR CHEMICAL CONCENTRATION

IN SOIL AND NJ SOIL CLEANUP OBJECTIVES

UOP SITE, EAST RUTHERFORD, NJ

Compound	Surface Soil Conc-Avg (mg/kg)	Surface Soil Conc-Max (mg/kg)	Subsurface Soil Conc. Avg. (mg/kg)	Subsurface Soil Concentration Max. - (mg/kg)	NJ Soil Cleanup Objective mg/kg
Benzene	1.21	48.0	1.5	33	1[a]
BEHP	1.79	17.0	24.83	690	10[b]
Carcinogenic PAH	3.87	80.6	3.88	94.6	10[b]
Chromium, Total	138.9	2,880	439.4	7,250	100
PCB	21.4	480	3.77	38	1-5
MCB	0.66	23.0	5.19	160	1[a]
Cyanides	2.42	34.8	2.91	62.3	12
1,2-DCB	16.4	550	21.07	710	10[b]
Lead	238	1820	169.8	1,000	250
Mercury	2.48	10.0	10.06	190	1
Zinc	198	1530	337.48	4010	350
Cadmium	1.38	16.0	1.58	34	3
Arsenic	4.77	18.0	14.4	52	20
Toluene	60.7	2100	39.33	1,600	1[a]
1,2-Diphenylhydrazine	ND	ND	ND	ND	1[a]
1,1,2,2-Tetrachloroethane	0.47	24.0	4.6	230	1[a]

a. Total Volatile Organic Compound Objective
b. Total Base/Neutral Extractable Organic Compound Objective
c. NJ Soil Cleanup Objectives are Non-Promulgated Standards

TABLE 6-2

COMPARISON OF INDICATOR CHEMICAL CONCENTRATION

IN GROUND WATER AND NJAC 7:9-6.6

Compound	Ground-water Concentration, ug/l Average	Max	NJAC 7:9 - 6.6 Criterion, ug/l
Benzene	3,530	44,000	N/A
BEHP	10	200	N/A
Carcinogenic PAH	0	0	N/A
Chromium, Total	10	80	50[a]
PCB	40	1,100	0.001
MCB	830	21,000	N/A
Cyanides	120	2,800	200
1,2-DCB	220	3,250	N/A
Lead	20	110	50

N/A: No Criterion Listed

a: applies to Hexavalent Chromium

7. CALCULATION OF DOSE

Chemical intakes will be calculated with the aid of the exposure scenarios relevant to the pathways identified in Chapter 3. In order to make an estimate, some assumptions must be made concerning human activities that lead to the exposure. Included in these assumptions are the magnitude of intake of an environmental media (air, soil) and the frequency of the exposure event. The type of individual who may be at risk (e.g. child, adult worker) was identified in Chapter 4. The assumptions underlying the exposure estimate will be detailed in the following section. Intake values estimated for each scenario will be converted to units of milligrams Indicator Chemical per kilogram body weight per day (mg/kg/day) to make them compatible with the dose-response relations that were developed in Chapter 2. The intake values calculated for the current site use scenario in this section are compiled in Table 7-1. Intake values for the future site use scenario are given in Table 7-2, and those calculated for the construction worker are given in Tables 7-3 and 7-4.

7.1 Air

7.1.1 Present Site Use

This assessment uses the common assumption that individuals inhale approximately one cubic meter of air per hour during periods of light to moderate activity (SPHEM, EPA, 1986). For the current condition of the site, it is not likely that individuals would be frequent visitors to the site. Therefore, the intake of contaminants in air was calculated by presuming that an individual was on the site one hour per week twelve months out of each year and would inhale contaminants present in one M^3 of air in that period. Assuming that people visit the site twelve months per year and can be exposed to contaminants in air is an overestimate, however, the NJDEP

TABLE 7-1

INTAKE OF INDICATOR CHEMICALS[a]

PRESENT SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Ingestion of Soil[b] Average	Ingestion of Soil[b] Maximum	Inhalation	Absorption[b,c] Average	Absorption[b,c] Maximum
Benzene	b	b	1.61×10^{-9}	b	b
CHP	5.22×10^{-8}	4.96×10^{-7}	3.91×10^{-10}	5.07×10^{-10}	4.82×10^{-9}
PAH	1.13×10^{-7}	2.35×10^{-6}	8.46×10^{-10}	1.10×10^{-9}	2.28×10^{-8}
Chromium (III)	5.37×10^{-5}	1.12×10^{-3}	4.03×10^{-7}	c	c
Chromium (VI)	2.83×10^{-6}	5.88×10^{-5}	1.52×10^{-9}	c	c
DCB	6.24×10^{-7}	1.40×10^{-5}	4.68×10^{-9}	6.06×10^{-9}	1.36×10^{-7}
MCB	b	b	1.35×10^{-8}	b	b
Cyanides	9.90×10^{-7}	1.42×10^{-5}	7.42×10^{-9}	9.62×10^{-9}	1.38×10^{-7}
1,2-DCB	6.69×10^{-6}	2.24×10^{-4}	5.02×10^{-8}	6.50×10^{-8}	2.18×10^{-6}
Lead	9.72×10^{-5}	7.43×10^{-4}	7.29×10^{-7}	c	c
Mercury	1.01×10^{-6}	4.08×10^{-6}	7.60×10^{-9}	c	c
Zinc	8.07×10^{-5}	6.24×10^{-4}	6.05×10^{-7}	c	c
Cadmium	5.65×10^{-7}	6.53×10^{-6}	3.03×10^{-10}	c	c
Arsenic	d	5.25×10^{-10}	d	c	c
Toluene	b	b	1.03×10^{-7}	b	b
1,2-Diphenylhydrazine	b	b	1.00×10^{-10}	b	b
1,1,2,2-Tetrachloroethane	b	b	1.75×10^{-10}	b	b

a. All intake values in units of milligrams of contaminant per kilogram body weight per day (mg/kg day).

b. No benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane or MCB assumed to be in soil directly at the surface. See Appendix C for emission of volatiles from below the immediate surface.

c. Dermal absorption of chromium, arsenic, cadmium, mercury, zinc, and lead does not occur.

d. Only maximum concentration of arsenic exceeds background levels.

TABLE 7-2

INTAKE OF INDICATOR CHEMICALS[a]

FUTURE SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Ingestion of Dust[b]	Inhalation	Absorption[bc]
Benzene	b	2.25×10^{-7}	b
BEHP	4.38×10^{-9}	1.43×10^{-9}	1.32×10^{-9}
PAH	9.48×10^{-9}	3.08×10^{-9}	2.84×10^{-9}
Chromium (III)	6.45×10^{-7}	2.10×10^{-7}	c
Chromium (VI)	3.39×10^{-8}	5.52×10^{-9}	c
PCB	5.24×10^{-8}	1.70×10^{-8}	1.57×10^{-8}
MCB	b	2.70×10^{-7}	b
Cyanides	1.19×10^{-8}	3.86×10^{-9}	3.56×10^{-9}
1,2-DCB	8.03×10^{-8}	2.61×10^{-8}	2.41×10^{-8}
Lead	1.17×10^{-6}	3.79×10^{-7}	c
Mercury	1.21×10^{-8}	3.95×10^{-9}	c
Zinc	9.69×10^{-7}	3.15×10^{-7}	c
Cadmium	6.78×10^{-9}	1.10×10^{-9}	c
Arsenic	d	d	c
Toluene	b	2.06×10^{-7}	b
1,2-diphenylhydrazine	b	1.40×10^{-8}	b
1,1,2,2-Tetrachloroethane	b	2.44×10^{-8}	b

a. All intake values are in units of mg/kg/day

b. No benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane or MCB assumed to be in soil directly at the surface. See Appendix C for emission of volatiles from below the immediate surface.

c. Dermal adsorption of chromium, cadmium, mercury, zinc, arsenic, and lead does not occur

d. Only maximum concentration of arsenic exceeds background levels.

TABLE 7-3

INTAKE OF INDICATOR CHEMICALS[a]

CONSTRUCTION WORKER SCENARIO: 10 MONTH EXPOSURE

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Ingestion of Soil[b]		Inhalation	Absorption[b,c]	
	Average	Maximum		Average	Maximum
Arsenic	d	1.21×10^{-9}	d	c	c
Benzene	b	b	8.95×10^{-9}	b	b
BEHP	1.20×10^{-10}	1.14×10^{-9}	1.80×10^{-10}	6.65×10^{-10}	6.31×10^{-9}
Car. PAH	2.60×10^{-10}	5.41×10^{-9}	3.89×10^{-10}	1.44×10^{-9}	2.99×10^{-8}
Cd	6.48×10^{-9}	7.51×10^{-8}	1.39×10^{-10}	c	c
Cr (III)	6.18×10^{-7}	1.29×10^{-5}	9.28×10^{-7}	c	c
Cr (VI)	3.25×10^{-8}	6.76×10^{-7}	6.97×10^{-10}	c	c
PCB	1.44×10^{-9}	3.22×10^{-8}	2.15×10^{-9}	7.94×10^{-9}	1.78×10^{-7}
MCB	b	b	4.13×10^{-7}	b	b
Cyanide	1.14×10^{-8}	1.63×10^{-7}	1.71×10^{-8}	6.32×10^{-8}	9.04×10^{-7}
1,2-DCB	7.70×10^{-8}	2.58×10^{-6}	1.16×10^{-7}	4.26×10^{-7}	1.53×10^{-5}
Lead	1.12×10^{-6}	8.55×10^{-6}	1.68×10^{-6}	c	c
1,1,2,2-TCA	b	b	1.07×10^{-9}	b	b
Hg	1.16×10^{-8}	4.74×10^{-8}	1.75×10^{-8}	c	c
Toluene	b	b	3.13×10^{-6}	b	b
Zn	9.29×10^{-7}	7.19×10^{-6}	1.39×10^{-6}	c	c

a. All intake values in units of milligrams of contaminant per kilogram body weight per day.

b. No benzene, toluene, 1,2-diphenylhydrazine, 1,1,2,2-tetrachloroethane or MCB assumed to be in soil directly at the surface. See Appendix C for emission of volatiles from below the immediate surface.

c. Dermal absorption of chromium, arsenic, cadmium, mercury, zinc, and lead does not occur.

d. Only maximum level of arsenic exceeds background concentrations.

TABLE 7-4

INTAKE OF INDICATOR CHEMICALS (mg/kg/day)

CONSTRUCTION WORKER SCENARIO: 2 MONTH EXPOSURE

UOP SITE, RUTHERFORD, N.J.

Compound	Ingestion of Soil[b]		Inhalation	Absorption[b,c]	
	Average	Maximum		Average	Maximum
Arsenic	c	3.16×10^{-8}	c	c	b
Benzene	4.52×10^{-10}	9.95×10^{-9}	5.37×10^{-7}	3.00×10^{-11}	6.61×10^{-10}
BEHP	1.22×10^{-8}	3.25×10^{-7}	2.93×10^{-9}	8.12×10^{-10}	2.16×10^{-8}
Car. PAH	2.09×10^{-9}	4.54×10^{-8}	5.01×10^{-10}	1.39×10^{-10}	3.02×10^{-9}
Cd	5.01×10^{-8}	8.25×10^{-7}	1.72×10^{-10}	b	b
Cr (III)	2.66×10^{-5}	4.66×10^{-4}	6.37×10^{-6}	b	b
Cr (VI)	1.40×10^{-6}	2.45×10^{-5}	4.79×10^{-9}	b	b
PCB	1.14×10^{-8}	2.36×10^{-7}	2.73×10^{-9}	7.54×10^{-10}	1.56×10^{-8}
MCB	1.10×10^{-7}	3.38×10^{-6}	1.51×10^{-5}	7.27×10^{-9}	2.24×10^{-7}
Cyanide	1.62×10^{-7}	2.94×10^{-6}	3.90×10^{-8}	1.08×10^{-8}	1.95×10^{-7}
1,2-DCB	4.94×10^{-7}	1.66×10^{-5}	1.19×10^{-7}	3.28×10^{-8}	1.10×10^{-6}
Lead	2.00×10^{-5}	1.39×10^{-4}	4.81×10^{-6}	b	b
1,1,2,2-TCA	6.3×10^{-11}	3.1510^{-9}	1.15×10^{-7}	4.19×10^{-12}	2.09×10^{-10}
Toluene	1.40×10^{-6}	5.68×10^{-5}	6.23×10^{-5}	9.27×10^{-8}	3.77×10^{-6}
Hg	5.51×10^{-7}	8.79×10^{-6}	1.32×10^{-7}	b	b
Zn	2.59×10^{-5}	2.66×10^{-4}	6.22×10^{-6}	b	b

a. All intake values in units of milligrams of contaminant per kilogram body weight per day.

b. Dermal absorption of chromium, arsenic, cadmium, mercury, zinc, and lead does not occur.

c. Only maximum level of arsenic exceeds background concentrations.

has directed UOP to employ this assumption. While this is not a great period of time, it should be remembered that worst-case conditions were assumed in determining ambient concentrations of contaminants in air (i.e., all surface soil contamination available for entrainment, highest concentration of volatiles in ground-water wells discharging to surface water, point of maximum annual average concentration 15 meters distant from the channel). Thus, if exposure duration is greater than that presumed here, it is likely to be off-set by more moderate conditions controlling contaminant concentrations available for exposure. Intake is calculated as:

Intake (mg/day) = Concentration (mg/M^3) X Inhalation Rate (1 M^3/hour) X Exposure duration (1 hour/day X 1 day/7 days X 12 months/12 months)

for volatile materials. The intake is multiplied by 75% for particulates, indicating the portion of dust that is conservatively estimated to be of respirable size.

7.1.2 Future Site Use

The employee at a possible facility built on the UOP site will be assumed to breath air contaminated by entrained particulates 18.5 hours per week. This assumption is derived from study of the frequency of prevailing winds in the area of the UOP site (see Appendix A). If contaminated soil is tracked into the facility and subsequently entrained for 12 months per year (see comment on page 7-1 regarding NJDEP directive about 12 month exposure), the intake value is calculated as:

Intake (mg/day) = Concentration (mg/M^3) X Inhalation Rate (1 m^3/hour) X Exposure duration (18.5 hours/week x 1 week/7 days X 12 months/12 months) x 0.75

for particulates assuming 75% are of respirable size. Concentrations of volatized indicator compounds are calculated using the same formula but without the 0.75 adjustment for respirable size and assuming that a person is on site for 40 hours per week instead of 18.5. Forty hours is used because the ISC model estimates an annual average concentration that has built into it variable wind direction (see Appendix A).

The 18.5 hours per week exposure to entrained indicator chemicals in the future site use scenario is a reasonable value because not all dust entering the building originates on-site. To use 40 hours neglects the other considerations in the complete future site use scenario and assumes that all of the dust entering from the building exterior originated from contaminated site areas. This is overly conservative for two reasons:

1. Much of the soil tracked into the building would have originated from sources miles distant from the site.
2. Dust generated by windy conditions would be entrained over a considerable distance and the contribution from on-site soil would be but a fraction of the total.

7.1.3 Construction Workers

Construction workers would be on-site to build the facility, a potential future use for the site. It is assumed that construction workers would inhale $2m^3$ of air per hour, as their activity level is higher than the average individual. The construction worker is assumed to be on-site for 12 months (8 hours each day, five days per week for 50 weeks) with 2 months (10 weeks) of this time devoted to earthmoving and foundation work. During the earthmoving activities, a "worst-case" assumption is made that the air is visibly dusty (1000 µg particulates/m^3), and that the workers are exposed to both surface and subsurface soils (weighted average). The remaining 10 months assumes exposure to surface soils only with

particulate concentrations of 75 $\mu g/m^3$. Air concentrations used can be found in Tables 5-3 and 5-4. Other assumptions and equations are the same as those for 7.1.2. Intake values for the construction worker scenario can be found in Tables 7-3 and 7-4.

7.2 Soils

7.2.1 Current Site Use

Because the UOP Site is not currently fully secured, there is a potential that people might trespass and make direct contact with contaminated soils on-site. Consequently, an individual might have a systemic exposure as the result of inadvertent ingestion of materials clinging to hands or other articles which may be placed in the mouth, and by absorption of material through the skin.

The U.S. EPA has suggested that the primary individuals for whom soil ingestion should be of concern are children between the ages of two to six (EPA 1986). This particular group is not likely to frequent the UOP Site, given its location. Rather, older children or adolescents appear to be the group that should be of greatest concern. Several uncertainties exist in the determination of average daily intake in this group. They include uncertainties about how much soil young people of this age range would ingest, and at what part of the site exposure occurs, as well as the frequency of visits to the site.

Estimates of the amount of soil ingested by young children are based on little direct data and vary widely. Data on older children in the relevant age group for the current exposure assessment are even more scarce. The minimum soil ingestion reported for two to six year olds in the literature is 10 milligrams per day, based on presumed intake of soiled candies (Day, et al. 1975) while the highest is the upper portion of the range estimated by Kimbrough, et al. (1984); 10 grams per

day. The high end of predicted soil ingestion rates has been adjusted downward (EPA 1986) and it has been acknowledged by EPA that the high level of intake is probably only pertinent for children with pica. Recent studies using trace elements in fecal material as indicators of soil ingestion in children indicate that the low end of the estimated range is incorrect as well. Clausing, et al. (1987) reports that the mean soil ingestion of nursery school children is 100 milligrams. If one subtracts the portion of this quantity that is due to ingestion of house dust (45 milligrams, determined by studying hospitalized children who did not go outside), the mean soil ingestion of outdoor soil is 55 milligrams. Although for the purposes of this assessment, it is likely that the average intake of an older individual would be about one half of this, NJDEP has directed UOP to assume that older children ingest 100 mg of soil per day.

Concerning frequency of exposure, it was assumed that young people would visit the site infrequently, perhaps 1 hour per week, twelve months out of each year.

For a "worst-case" scenario, intake of compounds from surface soil ingestion at the UOP Site was calculated using the maximum concentration detected. A second intake was calculated using average surface soil concentrations. The contaminant intake calculations from soil ingestion are:

Ingestion Intake (mg/day) = Concentration (mg/kg) X Soil ingestion rate (100 mg/day) X Exposure duration (1 day/7 days x 12 months/12 months) X Correction factor (10^{-6} kg/mg)

Constituents bound to particles on soiled hands or arms may be absorbed through the skin. The magnitude of absorption is a function of:

1) The bioavailability of constituents on the soil, i.e., the relative tendency of material to leave its soil binding site and partition through human skin.
2) The location and surface area of the soiled skin (different areas of skin have different absorbing capacities).
3) The chemical/physical properties of the constituents.
4) The time that materials are in contact with the skin.

Inadequate data on all of these factors makes calculation of intake via absorption an extremely uncertain enterprise. For the purposes of this assessment, the parameters of Hawley (1985) were used, however, it should be emphasized that because much of Hawley's information is based on assumption, it is not possible to statistically analyze the uncertainty of the intakes predicted in the scenarios. A young person outdoors might soil hands and arms covering a surface area of 2280 cm^2 (hands and arms are 19% of the total surface area of an individual. This analysis uses 12000 cm^2, the surface area of a 35 kg, 5 foot tall individual, Diem and Lentner, 1971). The mass of soil clinging to the skin was assumed to be 1166 milligrams, based on the measured data of Lepow, et al. (1975) that there was approximately 11 milligrams of soil on the soiled hands of children (surface area, 21.5 cm^2) as determined by tape-stripping the material (2280 cm^2 x 11 mg/21.5 cm^2 = 1166 mg). Hawley assumes an absorption rate of 2 percent per 24 hours, based on observations of absorption rate of materials made by Poiger and Schlatter (1979) on TCDD absorption for adsorbents.

The equation describing the assumptions is:

$$\text{Absorption Intake} = \text{Soil Concentration (mg/kg)} \times 1166\ \text{mg} \times \text{Correction Factor } \left(\frac{10^{-6}\ \text{mg}}{\text{kg}}\right) \times \text{Absorption Rate } \left(\frac{.02}{24\ \text{hours}}\right) \times \text{Duration } \left(\frac{1\ \text{day}}{7\ \text{days}} \times \frac{1\ \text{hour}}{\text{day}} \times \frac{12\ \text{mos}}{12\ \text{mos}}\right)$$

7.2.2 Future Site Use

Hawley (1985) developed an ingestion rate for indoor dust in adults, based on assumptions about the surface area of skin that might be soiled by house dust and what would be removed and inadvertantly ingested during such activities as eating or smoking. This value, 0.6 mg/day, will be used for the present exposure assessment because it is likely that in an office or commercial setting, most ingestion would occur indoors. Assuming an individual works 5 days per week, and contaminant is tracked indoors to become part of indoor, unsuspended, dust for 12 months of each year, the intake equation would be:

Ingestion Intake (mg/day) = dust concentration (mg/kg) X ingestion rate (0.6 mg/day) X duration of exposure (5 days/7 days X 12 months/12 months) X correction factor (10^{-6} kg/mg)

Absorption in adults may be treated in much the same way as previously described for children, with the following variations:

1) only hands are soiled (this amounts to 900 cm^2 of surface area)

2) Using the assumption of Hawley, (1985), the density of indoor dust is less than soil, amounting to 0.06 mg/cm^2 clinging to skin.

3) The exposure duration is different. It is assumed that hands are soiled approximately half of the 8 hour work day, 5 days per week, and that contaminants are present in indoor dust 12 months of the year.

Incorporating these assumptions into an intake equation:

Absorption intake (mg/day) = dust concentration (mg/kg) X dust mass (900 cm^2 X .06 mg/cm^2) X absorption rate (0.02/24 hours) x duration of exposure (4 hours/day X 5 days/7 days X 12 months/ 12 months) X correction factor (10-6 mg/kg)

7.2.3 Construction Workers

The construction workers which could be on site to build a facility for future use may inadvertantly ingest soil while conducting their jobs. The construction worker is assumed to be exposed for 5 days per week for 50 weeks. Inadvertant ingestion is more likely to occur during excavation activities (50 day duration). The NJDEP has requested that an ingestion rate of 100 mg/day be used for children. However, it is more appropriate to use an inadvertant ingestion rate of 50 mg/day for adult construction workers. During excavation activities, exposure to subsurface and surface soils will be assumed. The weighted average was computed from data from these two media and are presented in Table 5-4. The dose obtained during the remaining 10 months of exposure will be estimated from surface soil concentrations at the indoor dust ingestion rate (0.6 mg/day) as in the future scenario, as mainly indoor

construction activities are assumed to occur during that period. Other parameters are identical to those presented in 7.2.1. In addition, workers may also intake chemicals via dermal absorption through the hands. Again, exposure will be assumed to be to subsurface and surface soils during the two excavation months, and to surface soils only during the remaining 10 months. The exposed surface area is 900 cm^2, and other assumptions and exposure parameters are listed above or in 7.2.1. Intake estimates for the construction worker scenario can be found in Tables 7-3 and 7-4.

7.3 Adjustments

To make the intake estimates compatible with potency slope or AIC values, an adjustment must be made for body weight of the exposed individual. SPHEM states that the average weight of an adult is 70 kg. Because the exposure scenario for current site use must take in to account the possibility that some individuals visiting the site are young, a lower body weight, 35 kg, was assumed. Dividing intake estimates by this value gives a weight-corrected intake. The standard 70 kg is used to weight-correct intake in the future use and construction worker scenarios.

A second adjustment must generally be made for carcinogenic materials. The potency slopes, developed in Chapter 2, estimate cancer risk from a lifetime of exposure. For less-than-lifetime exposures a downward correction is required to obtain average lifetime daily doses. For current site use, a lifetime correction factor of 5 years/70 year lifetime is used. For the future use scenario, it is assumed that a 35 year career is spent in the business housed at the site. Thus, the lifetime correction factor is 35 years/70 year lifetime. For the construction worker, one year exposure during construction of a facility is assumed. Thus, the lifetime correction factor is one year/70 year lifetime.

Intake values for the current site use scenario are given in Table 7-1 and values for the future use scenario are given in Table 7-2. Intake values for the construction worker scenario are given in Tables 7-3 and 7-4.

8. ESTIMATION OF RISK

8.1 Non-Carcinogens

Table 8-1 presents "margins of safety" for health risk from exposure to non-carcinogenic Indicator Chemicals under the assumptions of the present site use scenario. These values were developed by dividing the appropriate inhalation or ingestion AIC values by predicted intakes for the soil and air exposure routes. Values greater than one indicate levels of intake are lower than those expected to produce toxic effects. The concept of margin of safety is that as the calculated value becomes progressively greater than one, it reflects more certainty that the exposure is safe, even if errors in the exposure level or dose-response have been made.

Because the margin of safety is a product of division, addition of reciprocals is required to determine the total margin of safety of combined ingestion and inhalation exposure. That is, to calculate the total margin of safety, the following formula is used:

$$\text{Total Margin of Safety} = 1/[(1/\text{margin of safety for ingestion}) + (1/\text{margin of safety for inhalation})]$$

Margins of safety for non-carcinogenic health risk under the assumptions of the future use scenario are presented in Table 8-2, and margins of safety for the construction worker scenario are presented in Table 8-3.

In addition to margins of safety, the NJDEP has directed that hazard indices be calculated for each indicator chemical and then be summed to determine a site-wide hazard index (HI). Summing of the hazard indices for each compound to arrive at a site-wide hazard index, without regard for the toxicological endpoint and mechanism of action, is incorrect for this site. At a screening level, however, such a summation can indicate if any potential for adverse health effects exists. If summing the HI of all indicator compounds results in an HI of less than

TABLE 8-1

NON-CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

PRESENT SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Margin of Safety: Soil[b] Maximum	Margin of Safety: Soil[b] Average	Margin of Safety Air[c]	Margin of Safety: Total Maximum	Margin of Safety: Total Average
BEHP	40,000	380,000	51,100,000	39,900	377,000
Chromium (III)	895	186,000	12,700	836	7530
Chromium (VI)	85	1,770	3,370,000	85	1,770
MCB	e	e	370,000	370,000	370,000
Cyanides	1,390	20,000	2,690,000	1,390	19,900
1,2-DCB	397	13,300	797,000	397	13,100
Lead	2	14	590	2	14
Mercury	490	1,970	263,000	489	1,960
Zinc	320	2,480	16,500	314	2,150
Cadmium	153	1,770	3,300,000	153	1,770
Arsenic	1,910	f	f	1,900	f
Toluene	e	e	9,720,000	9,720,000	9,720,000

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a. Risks are given as margin-of-safety values (described in text). A value greater than 1 indicates no risk.

b. Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of above-detection-limit samples from surface soil.

c. Because entrained material is assumed to be generated from a large area of the site, a single intake value for dust was calculated using the arithmetic mean of the above-detection-limit surface soil samples.

d. Oral AIC used for inhalation exposures.

e. MCB and Toluene are assumed not present in surface soil.

f. Only maximum level of arsenic exceeds background levels.

TABLE 8-2

NON-CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

FUTURE SITE USE SCENARIO

Compound	Margin of Safety: Dust[b]	Margin of Safety: Air[b,c]	Margin of Safety: Total
BEHP	3,510,000	14,000,000	2,810,000
Chromium III	1,600,000	24,300	23,900
Chromium IV	147,000	924,000	127,000
MCB	d	18,500	18,500
Cyanides	1,300,000	5,180,000	1,040,000
1,2-DCB	862,000	1,530,000	551,000
Lead	1,200	1,130	583
Mercury	165,000	506,000	124,000
Zinc	207,000	31,800	27,500
Cadmium	148,000	907,000	127,000
Arsenic	e	e	e
Toluene	d	486,000	486,000

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a. Risks are given as margin of safety values (described in text). A value greater than 1 indicates no risk.

b. "Dust" risk calculated from sum of ingestion and absorption intake. Because the outdoor soil which ultimately contributes to indoor dust is assumed to be transported from a large area of the site, only one "average" value of contaminant intake via dust and air was calculated for this scenario.

c. Oral AIC used for inhalation exposures.

d. MCB and toluene are assumed to not be present and available for contact in surface soil.

e. Only maximum level of arsenic exceeds background concentrations.

TABLE 8-3

MARGINS OF SAFETY FOR INDICATOR CHEMICALS[a]

CONSTRUCTION WORKER SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

	Margin of Safety: Soil[b]				Margin of Safety: Air[c]		Margin of Safety: Total[e]	
	Maximum		Average					
Compound	10-Month	2-Month	10-Month	2-Month	10-Month	2-Month	Maximum	Average
MCB	f	8,330	f	25,700	12,100	48,400	322	322
Cyanides	18,700	6,380	268,000	115,000	1,170,000	513,000	4,690	65,800
1,2-DCB	5,330	5,080	179,000	171,000	346,000	377,000	2,560	57,800
Lead	164	10.1	1,250	69.8	256	89.4	8.3	33.1
Mercury	42,600	228	172,000	3,620	2,920	386	136	311
Zinc	27,800	752	215,000	7,720	7,180	1,610	470	1,120
Toluene	f	4,950	f	202,000	95,800	383,000	4,580	4,580
Chromium (III)	77,800	2,140	1,620,000	37,700	5,500	800	523	685
Chromium (VI)	7,390	204	154,000	3,580	d	d	198	3,500
Cadmium	13,300	1,210	154,000	5,880,000	d	d	1,110	150,000

[a]Risks are given as margins of safety. A value greater than 1 indicates no risk.

[b]Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of samples from surface soil (for 10 month) or the weighted average of the surface and subsurface soil samples (for 2 month).

[c]Because entrained material is assumed to be generated from a large area of the site, a single intake value for dust was calculated using the arithmetic mean of the surface soil samples (for 10 month) and the weighted average of the surface and subsurface soil samples (for 2 month).

[d]Chromium (VI) and cadmium are presumed to be carcinogenic via inhalation.

[e]Total 12-month risk to construction workers (10 month and 2 month scenarios combined).

[f]Volatiles are assumed not to be present and available for ingestion and dermal absorption from the surface soil.

one, then no potential for non-carcinogenic adverse health effects exists. If the result of the summation is greater than one, then a more detailed evaluation, one that sums only hazard indices of compounds with identical toxicological endpoints and mechanisms of action, is needed. Hazard indices for the current site-use scenario are presented in Table 8-4, and Table 8-5 presents hazard indices for the future site-use scenario. Hazard indices for the construction worker scenario are in Table 8-6.

8.2 Carcinogens

Table 8-7 indicates potential cancer risk from exposure to constituents under the assumptions of the present site use scenario. The values are unitless risk estimates (e.g. 2 X 10^{-5}, or 2 chances out of 100,000). As such they may be added directly to give the total cancer risk of each constituent from all exposure routes, and a total cancer risk from all carcinogens. The latter value has been calculated as required by the guidance, however, the scientific basis for the additivity of carcinogenic action is weak. Carcinogens may act by different mechanisms and on separate organ systems. Some carcinogens enhance each others activity while others tend to antagonize other compounds. Thus, the total carcinogenic risk calculation must be viewed with some skepticism. Table 8-8 indicates potential cancer risk under the assumptions of the future site use scenario. Potential cancer risks for the construction worker scenario are in Table 8-9.

TABLE 8-4

NON-CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

PRESENT SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Hazard Index Soil[b] Maximum	Hazard Index Soil[b] Average	Hazard Index Air[c]	Hazard Index Total Maximum	Hazard Index Total Average
BEHP	2.5×10^{-5}	2.6×10^{-6}	2.0×10^{-8}	2.5×10^{-5}	2.7×10^{-6}
Chromium III	1.1×10^{-3}	5.4×10^{-5}	7.9×10^{-5}	1.2×10^{-3}	1.3×10^{-4}
Chromium VI	1.2×10^{-2}	5.7×10^{-4}	3.0×10^{-7}	1.2×10^{-2}	5.7×10^{-4}
MCB	e	e	2.7×10^{-6}	2.7×10^{-6}	2.7×10^{-6}
Cyanides	7.2×10^{-4}	5.0×10^{-5}	3.7×10^{-7}	7.2×10^{-4}	5.0×10^{-5}
1,2-DCB	2.5×10^{-3}	7.5×10^{-5}	1.3×10^{-6}	2.5×10^{-3}	7.6×10^{-5}
Lead	5.3×10^{-1}	6.9×10^{-2}	1.7×10^{-3}	5.3×10^{-1}	7.1×10^{-2}
Mercury	2.0×10^{-3}	5.1×10^{-4}	3.8×10^{-6}	2.0×10^{-3}	5.1×10^{-4}
Zinc	3.1×10^{-3}	4.0×10^{-4}	6.1×10^{-5}	3.2×10^{-3}	4.6×10^{-4}
Cadmium	6.5×10^{-3}	5.7×10^{-4}	3.0×10^{-7}	6.5×10^{-3}	5.7×10^{-3}
Arsenic	5.3×10^{-4}	f	f	5.3×20^{-4}	f
Toluene	e	e	1.0×10^{-7}	1.0×10^{-7}	1.0×10^{-7}
Summed HI	5.6×10^{-1}	7.2×10^{-2}	1.8×10^{-3}	5.6×10^{-1}	7.4×10^{-2}

a. Risks are given as hazard indices. A value less than 1 indicates no risk.

b. Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of above-detection-limit samples from surface soil.

c. Because entrained material is assumed to be generated from a large area of the site, a single intake value for dust was calculated using the arithmetic mean of the above-detection-limit surface soil samples.

d. Oral AIC used for inhalation exposures.

e. MCB and toluene are assumed to not be present and available for contact in surface soil.

f. Only maximum concentration of arsenic exceeds background levels.

TABLE 8-5

NON-CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

FUTURE SITE USE SCENARIO

Compound	Hazard Index: Dust[b]	Hazard Index: Air [b,c]	Hazard Index: Total
BEHP	2.9×10^{-7}	7.1×10^{-8}	3.6×10^{-7}
Chromium III	6.5×10^{-7}	4.1×10^{-5}	4.2×10^{-5}
Chromium VI	6.8×10^{-6}	1.1×10^{-6}	7.9×10^{-6}
MCB	d	5.4×10^{-5}	5.4×10^{-5}
Cyanides	7.7×10^{-7}	1.9×10^{-7}	9.7×10^{-7}
1,2-DCB	1.2×10^{-6}	6.5×0^{-7}	1.8×10^{-6}
Lead	3.3×10^{-4}	8.8×10^{-4}	1.7×10^{-3}
Mercury	6.1×10^{-6}	2.0×10^{-6}	8.1×10^{-6}
Zinc	4.8×10^{-6}	3.2×10^{-5}	3.6×10^{-5}
Cadmium	6.8×10^{-6}	1.1×10^{-6}	7.9×10^{-6}
Arsenic	e	e	e
Toluene	d	2.06×10^{-6}	2.2×10^{-6}
Summed HI	8.6×10^{-4}	1.0×10^{-3}	1.9×10^{-3}

a. Risk are given as hazard indices. A value less than 1 indicates no risk.

b. "Dust" risk calculated from sum of ingestion and absorption intake. Because the outdoor soil which ultimately contributes to indoor dust is assumed to be transported from a large area of the site, only one "average" value of contaminant intake via dust and air was calculated for this scenario.

c. Oral AIC used for inhalation exposures.

d. MCB and toluene are assumed to not be present and available for contact in surface soil.

e. Only maximum concentration of arsenic exceeds background levels.

TABLE 8-6

HAZARD INDICES FOR INDICATOR CHEMICALS[a]

CONSTRUCTION WORKER SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

	Hazard Index: Soil[b]				Hazard Index: Air[c]		Hazard Index: Total[e]	
	Maximum		Average					
Compound	10-Month	2-Month	10-Month	2-Month	10-Month	2-Month	Maximum	Average
MCB	f	1.2×10^{-4}	f	3.89×10^{-6}	8.27×10^{-5}	3.02×10^{-3}	3.22×10^{-3}	3.10×10^{-3}
Cyanides	5.34×10^{-6}	1.57×10^{-4}	3.73×10^{-6}	8.66×10^{-6}	8.56×10^{-7}	1.95×10^{-6}	2.13×10^{-4}	1.52×10^{-5}
1,2-DCB	1.88×10^{-4}	1.97×10^{-4}	5.59×10^{-6}	2.89×10^{-6}	5.86×10^{-6}	2.97×10^{-6}	3.90×10^{-4}	1.73×10^{-5}
Lead	6.11×10^{-3}	9.93×10^{-2}	7.98×10^{-4}	1.43×10^{-2}	3.90×10^{-3}	1.12×10^{-2}	1.21×10^{-1}	3.02×10^{-2}
Mercury	2.35×10^{-5}	4.39×10^{-3}	5.82×10^{-6}	2.75×10^{-4}	3.43×10^{-4}	2.59×10^{-3}	7.35×10^{-3}	3.22×10^{-3}
Zinc	3.59×10^{-5}	1.33×10^{-3}	4.64×10^{-6}	1.3×10^{-4}	1.3×10^{-4}	6.22×10^{-4}	2.12×10^{-3}	8.95×10^{-4}
Toluene	f	2.02×10^{-4}	f	4.96×10^{-6}	1.04×10^{-5}	2.08×10^{-4}	2.23×10^{-4}	4.20×10^{-4}
Chromium (III)	1.29×10^{-5}	4.66×10^{-4}	6.18×10^{-7}	2.66×10^{-5}	1.82×10^{-4}	1.25×10^{-3}	1.91×10^{-3}	1.46×10^{-3}
Chromium (VI)	1.35×10^{-4}	4.91×10^{-3}	6.51×10^{-6}	2.8×10^{-4}	d	d	5.04×10^{-3}	2.86×10^{-4}
Cadmium	7.51×10^{-3}	8.25×10^{-4}	6.48×10^{-6}	1.7×10^{-7}	d	d	9.00×10^{-4}	6.65×10^{-6}
					Summed Hazard Index =		1.42×10^{-1}	3.94×10^{-2}

[a]Risks are given as hazard indices. A value less than 1 indicates no risk.

[b]Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of samples from surface soil (for 10 month) or the weighted average of the surface and subsurface soils (for 2 month).

[c]Because entrained material is assumed to be generated from a large area of the site, a single intake value for dust was calculated using the arithmetic mean of the surface soil samples (for 10 month) and the weighted average of the surface and subsurface soil samples (for 2 month).

[d]Chromium (VI) and cadmium are presumed to be carcinogenic via inhalation.

[e]Total 12-month risk to construction workers (10 month and 2 month scenarios combined).

[f]Volatiles are assumed not to be present and available for ingestion and dermal absorption from the surface soil.

TABLE 8-7

CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

PRESENT SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, N.J.

Compound	Risk: Soil[b]		Risk: Air[c]	Risk: Total	
	Maximum	Average		Maximum	Average
Benzene	d	d	4.7×10^{-11}	4.7×10^{-11}	4.7×10^{-11}
BEHP	4.2×10^{-9}	4.4×10^{-10}	3.3×10^{-12}	4.2×10^{-9}	4.5×10^{-10}
Chromium (VI)	e	e	6.2×10^{-8}	6.2×10^{-8}	6.2×10^{-8}
PAH	2.7×10^{-5}	1.3×10^{-6}	5.2×10^{-9}	2.7×10^{-5}	1.3×10^{-6}
PCB	6.1×10^{-5}	2.7×10^{-6}	2.0×10^{-8}	6.1×10^{-5}	2.8×10^{-6}
Arsenic	7.9×10^{-7}	g	g	7.9×10^{-7}	g
Cadmium	e	e	1.9×10^{-9}	1.9×10^{-9}	1.9×10^{-9}
1,2-diphenyl-hydrazine	d	d	8.0×10^{-11}	8.0×10^{-11}	8.0×10^{-11}
1,1,2,2-tetra-chloroethane	d	d	3.5×10^{-11}	3.5×10^{-11}	3.5×10^{-11}
			Total Cancer Risk:	8.95×10^{-5}	4.13×10^{-6}

---

a. Risk values should be regarded as excess chance of getting cancer, with unity being complete certainty. Thus 3×10^{-9} is three chances in 1,000,000,000.

b. Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of above-detection-limit samples from surface soil.

c. Because entrained material is assumed to be generated from a large area of the sige, a single intake value for dust was calculated using the arithmetic mean of the above-detection-limit surface soil samples.

d. Benzene, 1,2-diphenylhydrazine, and 1,1,2,2-tetrachloroethane were presumed not to be present in surface soil.

e. Chromium and cadmium are presumed to be non-carcinogenic by the oral route.

f. No potency slope is available for the inhalation route. The oral potency slope was used.

g. Only maximum arsenic level exceeds background concentrations.

TABLE 8-8

CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

FUTURE SITE USE SCENARIO

Compound	Risk: Dust[b]	Risk: Air	Risk: Total
Benzene	c.	6.6×10^{-9}	6.6×10^{-9}
BEHP	4.8×10^{-11}	1.2×10^{-11}	6.0×10^{-11}
Chromium (VI)	d.	2.3×10^{-7}	2.3×10^{-7}
PAH	1.4×10^{-7}	1.9×10^{-8}	1.6×10^{-7}
PCB	3.0×10^{-7}	7.4×10^{-8}	3.7×10^{-7}
Arsenic	f	f	f
Cadmium	d	6.7×10^{-9}	6.7×10^{-9}
1,2-diphenyl-hydrazine	c	1.1×10^{-8}	1.1×10^{-8}
1,1,2,2-tetra-chloroethane	c	4.9×10^{-9}	4.9×10^{-9}
	Total Cancer Risk:		7.9×10^{-7}

---

a. Risk values are excess chance of getting cancer.

b. "Dust" risk calculated from the sum of ingestion and absorption intake. Because the outdoor soil which ultimately contributes to indoor dust is assumed to be transported from a large area of the site, only one "average" value of contaminant intake via dust and air was calculated for this scenario.

c. Benzene 1,2 diphenylhdadrazine, and 1,1,2,2, tetrachloroethene are not present in surface soil.

d. Chromium and cadmium are not carcinogenic by the oral route and not absorbed, dermally.

e. Oral potency slope used for inhalation exposures.

f. Only maximum arsenic concentration exceeds background levels.

TABLE 8-9

CARCINOGENIC RISK FROM INDICATOR CHEMICALS[a]

CONSTRUCTION WORKER SCENARIO

UOP SITE, EAST RUTHERFORD, NJ

	Risk: Soil[b]				Risk: Air[c]		Risk: Total[e]	
	Maximum		Average					
Compound	10-Month	2-Month	10-Month	2-Month	10-Month	2-Month	Maximum	Average
Benzene	f	3.08×10^{-10}	f	1.40×10^{-11}	2.59×10^{-10}	1.56×10^{-8}	1.69×10^{-8}	1.58×10^{-8}
BEHP	6.26×10^{-11}	2.91×10^{-9}	6.59×10^{-12}	1.1×10^{-10}	1.51×10^{-12}	2.46×10^{-11}	3.0×10^{-9}	1.42×10^{-10}
PAHs	1.93×10^{-8}	5.52×10^{-7}	4.03×10^{-7}	2.54×10^{-8}	2.38×10^{-9}	3.06×10^{-9}	9.6×10^{-7}	5.02×10^{-8}
PCBs	9.13×10^{-7}	1.09×10^{-6}	4.07×10^{-8}	5.26×10^{-8}	9.34×10^{-9}	1.18×10^{-8}	2.03×10^{-6}	1.14×10^{-7}
Arsenic	1.81×10^{-9}	4.73×10^{-8}	g	g	g	g	5.24×10^{-8}	g
Chromium (VI)	d	d	d	d	2.68×10^{-8}	1.96×10^{-7}	2.25×10^{-7}	2.25×10^{-7}
Cadmium	d	d	d	d	8.47×10^{-10}	1.05×10^{-9}	1.89×10^{-9}	1.89×10^{-9}
1,1,2,2-TCA	f	6.72×10^{-10}	f	1.34×10^{-11}	2.15×10^{-10}	2.29×10^{-8}	2.38×10^{-8}	2.31×10^{-8}
					Total Cancer Risk =		3.30×10^{-6}	4.31×10^{-7}

[a]Risk values should be regarded as excess chance of getting cancer, with unity being complete certainty. Thus 3×10^{-9} is three chances in 1,000,000,000.

[b]Sum of ingestion and absorption intake. Maximum values calculated from maximum detected concentration of Indicator Chemical at the site. Average intake calculated using arithmetic mean of samples from surface soil (for 10 month) or the weighted average of the subsurface and surface soil (for 2 month).

[c]Because entrained material is assumed to be generated from a large area of the site, a single intake value for dust was calculated using the arithmetic mean of the surface soil samples (for 10 month) or the weighted average of the subsurface and surface soil (for 2 month).

[d]Chromium VI and cadmium are presumed to be carcinogenic via inhalation only.

[e]Total 12-month risk to construction workers (10 month and 2 month scenarios combined).

[f]Volatiles are assumed not to be present and available for ingestion and dermal absorption from the surface soil.

[g]Only maximum arsenic level exceeds background concentrations.

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9. PRESENTATION OF RISK

Risks from exposure to constituents at the UOP Site were presented in tabular form in Chapter 8 for present and future scenarios and for construction workers. Table 9-1 summarizes these risks. It can be seen from this table that no non-carcinogenic risk is expected for exposures of the magnitude developed in the exposure scenarios.

Total carcinogenic risk for the present use scenario ranges from approximately 9.0×10^{-5} to 4.1×10^{-6} depending on assumptions about the source for ingestion and absorption exposures. The total risk is primarily due to the presence of PAH and PCB in the soil; which contribute about 30% and 65%, respectively to the total risk. Direct contact with soil appears to be the pathway of importance for exposure and consequent risk.

Total carcinogenic risk for the future site use scenario is: 7.9×10^{-7}. Chromium (VI), PCB and PAH account for a greater than 90% of the total risk.

Total carcinogenic risk for the construction worker scenario ranges from 4.31×10^{-7} to 3.30×10^{-6}. As with present and future use scenarios, the majority of risk comes from exposure pathways involving contact with soil, and over 98% of the risk is associated with PAHs, PCBs and chromium (VI).

TABLE 9-1

RISK SUMMARY TABLE

UOP SITE, EAST RUTHERFORD, NJ

Scenario	Total Hazard Index[a]		Total Cancer Risk[b]	
	Maximum	Average	Maximum	Average
Present Site Use	0.56	0.074	8.95×10^{-5}	4.13×10^{-6}
Future Site Use	c	0.0019	c	7.9×10^{-7}
Construction Worker	0.142	0.039	3.3×10^{-6}	4.3×10^{-7}

[a] A value less than one indicates no risk.

[b] Risk values are excess chance of getting cancer.

[c] Because the outdoor soil which ultimately contributes to indoor dust is assumed to be transported from a large area of the site, only one "average" value of contaminant intake was calculated for this scenario.

10. UNCERTAINTY ANALYSIS

Uncertainties in the risk assessment derive from a variety of sources; including:

1) Variance in analytical measurement techniques and the quality of the results
2) Uncertainty related to the human activities giving rise to exposure
3) Dose-response extrapolation

10.1 Analytical Techniques

Variation in analytical results may produce an overestimate or underestimate of Indicator Chemical available for exposure.

For data with adequate QA/QC documentation, there is likely to be little uncertainty due to analytical error in this portion of the risk assessment.

10.2 Exposure Activities

There is extreme uncertainty in determining the types of human activity that produce exposure. Hypothesizing an exposure pathway that does not exist overestimates risk, while neglecting an existent pathway underestimates risk. It is ERT's experience that the ingestion pathway assessed in this report tends to reveal greater risk than some exposure paths not included. Thus, the current assessment should still give a conservative estimate of the risk of the site.

For the exposure pathway that has been chosen, uncertainty concerning frequency and duration of exposure may produce underestimates or overestimates of risk. Uncertainty concerning the location of exposure has produced overestimates of risk because the area of contamination was distributed

across the entire site for the exposure scenario rather than only in the detected areas.

10.3 Dose-Response Extrapolation

Uncertainty in extrapolating dose-response data from the laboratory or epidemiological study to environmental health risk assessments is large. It may tend to produce an overestimate or underestimate of risk. The EPA methodology for selecting AICs is reasonably conservative and should produce reasonable certainty that an exposure below the AIC will not cause an effect. The method, however, only relates to known effects of the compound.

The potency slope for carcinogenic PAH is based on a study of benzo(a) pyrene carcinogenicity and is an upper 95% confidence bound on the dose-response curve. As such, this risk estimator should be more likely to overpredict than underpredict risk. It appears that benzo(a) pyrene is a more potent carcinogen than other PAH being subjected to the same analysis in the current report. This should also tend to produce an overprediction of risk. PAHs that have not been included in the cancer assessment may have co-carcinogenic action (they are not carcinogenic themselves, but enhance cancer production of other PAH) or be anti-carcinogens. The other PAH in the material at the UOP site may have either of these actions and increase or decrease the risk from exposure to carcinogenic PAH. The potency slope for PCB is also an upper 95% confidence bound and should therefore be conservative. Uncertainty relative to the qualitative aspects of PCB toxicity was discussed previously, in Chapter 2.

11. DISCUSSION AND FINDINGS

Areas 1, 1A, 2, and 5 of the UOP site in East Rutherford, New Jersey have been found to contain organic and inorganic contaminants. For the most part, these constituents are distributed in the soils and ground water at the site in a random fashion (a possible exception to this trend is the presence of PAH and PCBs, which tend to be limited to Area 5). Further, detection of most compounds occurred infrequently (frequency of detection of Indicator Chemicals is compiled in Tables 1-2, 1-3 and 1-4). Indicator Chemicals, chosen by UOP based on high indicator score ranking or frequency of detection higher than most compounds, included:

- arsenic,
- benzene,
- bis(2-ethylhexyl)phthalate,
- carcinogenic polynuclear aromatic hydrocarbons (PAH, including benzo[a] anthracene, benzo[a]pyrene, benzo[b]fluoranthene, chrysene, and dibenzo-[a,h]anthracene),
- chromium
- cyanide
- 1,2-dichlorobenzene
- polychlorinated biphenyls (PCBs),
- mono chlorobenzene, and
- lead.

In addition, NJDEP directed that seven other compounds be included in the risk assessment because of their presumed potential to cause adverse health effects at the site.

Based on this directive, the following four were added to ground water:

- 1,2-diphenylhydrazine
- toluene

- 1,1,2,2-tetrachloroethane
- nickel;

and the following three were added to surface soils:

- mercury
- zinc
- cadmium.

The site has little impact on offsite receptors. Dusts or volatile emissions from the site are likely to be dispersed to very low concentrations before they reach offsite locations. Ground water does not communicate with offsite wells. The stream channels onsite empty to Berrys Creek and could provide a source for contamination of aquatic biota. The impact of these potential sources will be assumed in the forthcoming, environmental assessment.

Onsite receptors presently include individuals who trespass or are legitimate visitors to the site. As directed by NJDEP, an exposure scenario in which a young person was present at the site 1 hour per week, 12 months per year breathing volatile or entrained materials, ingesting 100 mg of surface soil, and absorbing constituents from soil clinging to hands and arms, was developed to assess the potential health risk to current site visitors.

The outcome of the risk assessment of the above described scenario indicates that non-carcinogenic toxic effects from constituents at the site are not likely to be significant. Predicted intakes of these materials are between 2 and 9,720,000 times lower than acceptable intakes (AICs) developed by the U.S.EPA. The overall cancer risk of the site was approximately 9.0×10^{-5} to 4.3×10^{-6}. The majority of the overall cancer risk is from carcinogenic PAH and PCBs. For both compounds, the soil ingestion route of exposure is primarily responsible for the risk level. The estimated carcinogenic risks are unrealistically high; at a minimum by

one order of magnitude. Assumptions that lead to an overestimate include:

- Assuming soil is available for trespassers for 12 months. This assumption does not account for times during the year that the site is snow covered, the ground is frozen, or the weather is inclement, thus preventing trespassers access to contaminants in soil.
- Assuming a person ingests 100 mg of soil during the brief period they are on site. Recent evidence indicates that young children, those most likely to ingest soil, only eat about 55 mg of outdoor soil per day.
- In addition, the risk assessment has assumed a very high concentration of dust in soil; a conservatively high proportion of respirable particulates in air; a volatile emission exposure scenario that has a very low probability of occurrence; and that indicator compounds do not degrade.

All of these assumptions lead to significant overestimates of risk. The results of the risk assessment should be used with this in mind.

Research into the zoning and land-use planning activities of authorities controlling the area of the UOP Site indicates that the future use of the property will almost certainly be commercial or industrial. An exposure scenario considering this type of land use revealed health risks slightly lower than that for the present use scenario. No non-carcinogenic indicator chemicals have significant health impact; Margins of Safety range from 583 to 2,840,000 for the future site use scenario. Total carcinogenic risk has been calculated to be 8.2×10^{-7}. In this case the risk is from chromium (VI), carcinogenic PAH, and PCBs. Ingestion exposures account for a majority of the risk.

Because most of the exposure pathways have the same source - surface soils - it appears that, if necessary, remedial action should address surface soil contamination of chromium, PAH, and PCBs. If necessary, remedial activities should reduce direct contact with these materials and prevent the possibility of entrainment.

For the purposes of remedial design, it is pertinent to develop a "design goal" that would reduce risk from the presumed exposure scenario to levels considered acceptable. Of course, "acceptable" is a value judgement that must be weighed against the following factors:

- Cost of remedial actions
  - Is the reduction in risk justified by the increase in cost? (A large cost increase for a modest risk reduction is not an appropriate use of resources.)
- Feasibility of remedial actions
  - Is the reduction in risk attainable by current technology?
  - Are the concentration goals measurable?
- Level of certainty that the exposure will occur.
  - Several conservative assumptions are built into each exposure scenario. As these conservative assumptions are compounded, there is less certainty that the scenario would actually occur. Some facilities are operated that have a virtual certainty of exposure. One example of these is a resource recovery facility that would have emissions of combustion by-products.
- Size of the population at risk
  - The size of the potentially affected population is important in determining the overall risk of exposure. Small impacted populations have a smaller total risk than large populations. In the example cited above, the resource recovery

facility emissions would be expected to expose a very large population. The uses of the UOP Site will result in a much smaller population that could potentially be exposed.

- Special attributes of the population at risk
  - Small children, nursing home residents, and hospital patients are examples of sensitive populations that might be protected to a lower risk level. These sensitive populations are not expected to be present or be future users of the UOP Site.

Under U.S. EPA Guidance under CERCLA (1985), design goals are to be developed for a range of risks from 10^{-4} to 10^{-7}. This range can be used with the other information available to risk managers, to select a design goal for the site. Because the cancer risk at low doses is presumed to be linear under EPA dose-response assessment methodology, the design goals for EPA criteria will merely be order of magnitude multiples. To calculate a design goal, one must determine the difference between the estimated risk and the risk goal and reduce the current soil concentration by that amount. In arithmetic form:

$$\text{Design Goal Concentration} = \frac{\text{Present Soil Concentration}}{(\text{current risk/risk goal})}.$$

Table 11-1 presents design goals at different acceptable risk levels for chromium, PAH, and PCBs for the current site use scenario. These calculations are made with either maximum risk or average risk estimates; the values are the same in either case. However, the design goals may be applied differently, depending on which exposure scenario, worst-case or "average", is selected as credible. If the worst-case ingestion exposure is considered likely for the site, a design goal represents the maximum value that should remain accessible anywhere on the surface of the site. If the random-contact scenario (which results in exposure to average soil conditions)

TABLE 11-1

HEALTH-BASED DESIGN GOALS

PRESENT SITE USE

UOP SITE, EAST RUTHERFORD, NEW JERSEY

Compound	Surface Soil Conc., mg/kg-Avg.	Surface Soil Conc., mg/ug.-Max	Goals for Surface Soils (mg/kg) Risk = 10^{-4}	Risk = 10^{-5}	Risk = 10^{-6}	Risk = 10^{-7}
Chromium VI	6.9	144	b.	b.	a.	a.
PAH	3.87	80.6	b.	a.	3.0	0.3
PCB	21.4	480	b.	a.	7.8	0.8

a. Existing average constituent concentration is less than the calculated design goal.

b. Existing maximum constituent concentration is less than the calculated design goal.

c: Indicator compounds not listed in the table do not have concentrations with greater than $1x10^{-7}$ risk.

is considered more likely, then a design goal represents the average value that should remain accessible on the surface of the site. Table 11-1 indicates that the existing maximum or average concentrations of Indicator Chemicals is less than the calculated design goal for risk levels equal to 10^{-4} and 10^{-5} for all Indicator chemicals, as well as the 10^{-6} and 10^{-7} for chromium (VI). This indicates that the site already meets these goals and no further site remediation is necessary to achieve these goals. Site remediation would be necessary to lower the risk to 10^{-6} or 10^{-7} remediation goals for PAH and PCB.

Table 11-2 presents design goals for chromium, PAH, and PCB based on the future use exposure scenario. Because exposure to Indicator Chemicals in this scenario results from average soil concentrations, the design goals derived in this table are goals for average soil conditions on the site. Here it is apparent that all Indicator chemicals meet the 10^{-4}, 10^{-5} and 10^{-6} design goals. Site remediation would only be necessary to lower the risks to the 10^{-7} level.

TABLE 11-2

HEALTH-BASED DESIGN GOALS

FUTURE SITE USE SCENARIO

UOP SITE, EAST RUTHERFORD, NEW JERSEY

Compound[c]	Surface Soil Conc., mg/kg-Avg.	Surface Soil Conc., mg/ug.-Max	Goals for Surface Soils (mg/kg) Risk = 10^{-4}	Risk = 10^{-5}	Risk = 10^{-6}	Risk = 10^{-7}
Chromium VI	6.9	144	b.	b.	a.	3.1
PAH	3.87	80.6	b.	b.	a.	2.4
PCB	21.4	480	b.	b.	a.	5.8

---

a: Existing average constituent concentration is less than the calculated design goal.

b: Existing maximum constituent concentration is less than the calculated design goal.

c: Indicator compounds not listed in the table do not have concentrations with greater than $1x10^{-7}$ risk.

REFERENCES

Aksoy, M., S. Erdem, and G. DiNicola (1974) Leukemia in shoe workers exposed chronically to benzene. Blood 44:837-841.

Bahn, A.K., P. Grover, I. Rosenwaike, K. O'Leary and J. Stellman (1977) PCBs and melanoma. New England J. Med. 296:108.

Bell, F.P. C.S. Patt, B. Brundage, P.J. Gillies, and W.A. Phillips (1987) Studies on lipid biosynthesis and cholesterol content of liver and serum lipoproteins in rats fed various phthalate esters. Lipids 13:66.

Bertazzi, P.A. et al (1981) Mortaility study of male and female workers exposed to PCBs. International Symposium on Prevention of Occupational Cancer. April 21-24.

Brown, D.P. and M. Jones (1981) Mortality and industrial hygiene study of workers exposed to polychloriated biphenyls. Arch Env. Health 6:120-129.

Calandra, J.C. (1975) Summary of toxicological studies on commercial PCBs. Proceeding of the National Conference on Polychlorinated Biphenyls. EPA 560/6-75-004.

Deichmann, W.B. (1981) Halogenated cyclic hydrocarbons in Patty's Industrial Hygiene and Toxicology G.D. Clayton and F.E. Clayton, editors, Wiley, New York. pp. 3604-3611.

Department of Health, Education and Welfare, Environmental Health Perspectives, Arsenic and Lead, Volume 19, August 1977.

Diem, K. and C. Lentner (1970) Document a Geigy. Scientific Tables, seventh edition. J. R. Geigy, Basle, Switzerland p. 537.

Dilley, J.V. (1977) Toxic evaluation of inhaled chlorobenzene. NIOSH contract No. 210-76-0126 (Cited in EPA, 1984).

Doherty, PA., V.H. Ferm, and R.P. Smith (1982) Congenital malformations induced by infusion of sodium cyanide in the golden hamster. Toxicol. appl. Pharmacol. 64:456.

Doudoroff, D. et al (1966) Acute toxicity to fish of solutions containing complex metal cyanides, in relation to concentration of molecular hydrocyanic acid. Trans. Am. Fish. Soc. 95:6.

EPA (1980a) An Exposure and Risk Assessment for Phthalate Esters. EPA 440/4-81-020.

EPA (1980b) Ambient Water Quality Criteria for Phthalate Esters. EPA 440/5-80-067.

EPA (1980c) Ambient Water Quality Criteria for Cyanides. EPA 440/5-80-037.

EPA (1980d) Ambient Water Quality Criteria for Dichlorobenzenes. EPA 440/5-80-39.

EPA (1980e) Ambient Water Quality Criteria for Polychlorinated Biphenyls. EPA 440/5-80-068 (October 1980).

EPA (1984a) Risk Analysis of TCDD Contaminated Soil. PEA 600/8-84-031.

EPA (1984b) Health Assessment Document for Inorganic Arsenic. EPA 600/8-83-021F.

EPA (1984c) Health Effects Assessment for Chlorobenzene. EPA 540/1-86/040.

EPA (1984d) Health Assessment Document for Chromium. EPA 600/

EPA (1985a) Guidance on Feasibility Studies Under CERCLA.

EPA (1985b) Drinking Water Criteria Document for Cyanides. EPA 600/x-84-192.

EPA (1986) Superfund Public Health Evaluation Manual EPA 540/1-86/060.

Eschenroder, A., R.J. Jaeger, J.J. Ospital, and C.P. Doyle (1986) Health risk analysis of human exposures to soil amended with sewage sludge contaminated with polychlorinated dibenzodioxins and dibenzofurans. Vet. Hum. Toxicol. 28:435-442.

Geraghty and Miller, Inc., Remedial Investigation Report, Areas 1, 1A, 2 and 5, UOP Site, East Rutherford, N.J., 1987.

Gilman, A.G., L.S. Goodman, and A. Gilman (1980) The Pharmacological Basis of Therapeutics. Sixth Edition. MacMillan, New York.

Goldstein, B.D. (1977) Hematotoxicity in humans. J. Toxicol. Env. Health (supplement) 2:69-105.

Gray, T., K.R. Butterworth, I.F. Gaunt, P. Grasso, and S.D. Gangoli (1977) Short-term toxicity study of di(2-ethylhexyl) phthalate in rats. Food Comet. Toxicol. 15:389.

Harkov, R., K. Kisselbach, T. Fields, and J. Hunter (1987) A summary of selected soil contaminants at background locations in New Jersey. Submitted to NJ ACAD Sci.

Hawley, J.K. (1985) Assessment of health risk from exposure to contaminated soil. Risk Analysis 5:289-302.

HAZARDLINE (computer database aquisition, 1987) Occupational Health Service, Inc. Secaucus, N.J.

Howard, J.W. and R.F. Janzal (1955) Chronic toxicity for rats of food treated with hydrogen cyanide. J. Agric. Food Chemistry 3:325.

Hollingsworth, R.L., et al (1958) Toxicity of O-chlorobenzene. Studies on animals and industrial experience. AMA Arch. Ind. Health. 17:180

Horiguchi, K., et al. 1962. Studies on the industrial tetrachloroethane poisoning (2). Osaka City Med. Journal 8:29.

Hsu, S-T, et al (1985) Discovery and epidemiology of PCB poisoning in Taiwan: A four-year followup. Environ. Health Perspect. 59,5

Infante, P.F., R.A. Rinsky, J.K. Wagoner, and R.J. Young (1977a) Leukemia in benzene workers. Lancet 2:76.

Infante, P.F., R.A. Rinsky, J.K. Wagoner, and R.J. Uoung (1977b) Benzene and leukemia. Lancet 2:867-869.

Kimbrough, R.D., et al (1975) Induction of Liver tumors in Sherman strain rats by polychlorinated biphenyl aroclor 1260. J. Nat Cancer Inst. 55, 144.

Kimbrough, R.D. (1987) Human health effects of polychlorinated biphenyls and polybrominated biphenyls. Ann Rev. Pharm. and Tox. 27:87-111.

Kirk-Othmer (1978) Encyclopedia of Chemical Technology, Third Edition. Volumes 8 and 13. Wiley and Sons, New York.

Klaassen, C.D., Amdur, M.O., and Doull, J. (1986) Cassarett and Doull's Toxicology, The Basic Science of Poisons. Third Edition. MacMillan, New York.

Knapp, W.K., W.M. Busey, W. Kundzins (1971) Subacute oral toxicity of monochlorobenzene in dogs and rats. toxicol. appl. Pharmacol 19:393.

Kuratsune, et al (1972) Epidemiology study on Yusho. Environ. Health Perspect. 1, 119.

Lepow, M.L., L. Bruckman, R.A. Rubino, S. Markowitz, M. Gillette, and J. Kapish (1975) Investigations into sources of lead in the environment of urban children. Env. Res. 10:415-426.

McConnell, E.E. (1985) Comparative toxicity of PCBs and related compounds in various species of animals. Environ. Health Perspect. 60:29.

MacKenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert, and F.L. Langham (1958) Chronic toxicty studies II. Hexavalent and trivalent chromium administered in drinking water to rates. AMA Arch. Ind. Health 18:232-234.

Mancuso, T.F. (1975) International Conference on Heavy Metals in the Environment. Toronto, Canada. (described in EPA, 1984d)

Marhold, J., Jr., et al. 1968. The possible complicity of diphenyline in the origin of tumors in the manufacture of benzidine. Neoplasma. 15:3.

The Merck Index (Merck). 1983. Windholz, M., S. Budavari, R.F. Blumetti, and E.S. Otterbein (editors), 10th edition.

Metcalf, R.L., G.M. Booth, C.K. Schuth, D.J. Hansen, and P. Lu (1973) Uptake and fate of di(2-ethylhexyl) phthalate in aquatic organisms and in a model ecosystem. Environ. Health Perspect. 4:175.

Moody, D.E. and J.K. Reddy (1978) Hepatic peroxisome (microbody) proliferation in rats fed plasticizers and related compounds. Toxicol. appl. Pharmacol. 45:497.

Morgan, R.W., J.M. Ward, and P.E. Hartman (1981) Aroclor 1254 induced intestinal metaplasia and adenocarcinoma in the glandular stomach of F344 rats. Cancer Res. 41:5052-5059.

Muller, L. 1932. Experimental contribution to tetrachloroethane poisoning. Archives Gewerbepathol. Gewebehg. 2:326 (Ger.)

National Cancer Institute. 1978. Bioassay of hydrazobenzene for possible carcinogenicity. Publication No. (NIH) 78-1342.

National Cancer Institute. 1978. Bioassay of 1,1,2,2-tetrachloroethane for possible carcinogenicity. National Institute of Health, National Cancer Institute. DHEW Publ. No. (NIH) 78-827. Public Health Serv. U.S. Dept. Health Edu. Welfare.

Navrotskiy, Vk., et al. 1971. Comparative assessment of the toxicity of a number of industrial poisons when inhaled in low concentrations for prolonged periods. Trudy S'ezda Gigenistov, Ukranixoi. 8:22y (Rus.)

Neal J. and R.J. Rigdon (1967) Gastric tumors in mice fed beno[a]pyrene: a quantitative study. tex. Rep Biol. Med. 25:553.

Nierstedt, William, staff engineer, Hackensack Meadowlands Development Commission, personal communication, May 15, 1987.

NCI (1978) Bioassay of Aroclor 1254 for Possible Carcinogenicity.

NTP (1980) Carcinogenesis bioassay of di(2-ethylhexyl) phthalate. Draft Report. DHHS publication number 81-1773.

Ott, M.G., J.C. Townsend, W.A. Fishbeck, and R.A. Langer (1978) Mortality among individuals occupationally exposed to benzene Arch. Env. Health. 33:3-10.

Roberts, T.M., W. Gizyn, and T.C. Hutchinson (1975) Lead contamination of air, soil, vegetation, and people in the vicinity of secondary lead smelters Conference on Trace Substances and Environmental Health 8:155-166.

Schaffer, C.B., C.P. Carpenter, and H.F. Smyth (1945) Acute and subchronic toxicity of di(2-ethylhexyl) phthalate with note upon its metabolism. J. Ind. Hyg. Toxicol. 27:130.

Smith, A.B. et al (1982) Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. Brit. J. Ind. Med., 39, 361.

Smyth, H.F. Jr., et al. 1969. Range-finding toxicity data: list VII. American Industrial Hygiene Association Journal. 30:470.

Sterling, T.d. and D.M. Kobayashi (1977) Exposure to pollutants in "enclosed" living spaces. Env. Res. 13:1-35.

TerHaar, G. and R. Aronow (1974) New information on lead in dirt and dust as related to the childhood lead problem. Environ. Health, Perspect. 7:83-89.

Tewe, O.O. and J.H. Maher (1981a) Long-term and carry-over effect of dietary inorganic cyanide (KCN) in the life cycle performance and metabolism of rats. Toxicol. appl. Pharmacol. 58:1-7.

Tewe, O.O. and J.H. Maher (1981b) Performance and pathophysiological changes in pregnant guinea pigs fedcassava diets containing different levels of cyanide. Res. Vet. Sci. 30:147.

Thyssen, J. J. Althoff, G. Kimmerle, and U. Mohr (1981) Inhalation studies with benzo[a]pyrene in Syrian Golden Hamsters. J. Nat Cancer Inst. 66:575-577.

Tiphane, Marcel and Jacques St. Pierce, Tables for Sea Water Salinity Determination By Electrolytic Conductivity, April 1962.

Tseng, W.P., H.M. Chu, S.W. HOw, J.M. Fong, C.S. Lin and S. Yeh (1968) Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Nat Canter Inst. 40:453-463.

U.S. Environmental Protection Agency. 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. PB81-117400.

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for Diphenylhydrazine. PB81-117731.

Varshaskaya, S.P. (1967) The hygienic standardization of mono- and dichlorbenzene. Nauch tr. Aspir. i Ordin. Pervyi Mosk. Med. Institut (Russian, described in EPA 1980d).

Windsor, Jr., J.G. and R.A. Hites, Polycyclic Aromatic Hydrocarbons in Gulf of Maine Sediments and Nova Scotia Soils, Geochimica et Cosmochimica Acta, Vol. 43, pp. 27-33, 1979.

Youngblood, W.W. and M. Blumer, Polycyclic Aromatic Hydrocarbons in the Environment: Homologus Series in Soils and Recent Marine Sediments. Geochim. Cosmochim. Acta, 39, 1303-1314, 1975.

APPENDIX A

VOLATILE EMISSION CALCULATIONS

CALCULATION 1: ATMOSPHERIC CONCENTRATION OF VOLATILES FROM UOP SITE STREAM CHANNELS

CALCULATION 2: AIR CONCENTRATIONS FROM VOC EMISSIONS FROM A BUILDING EXCAVATION

APPENDIX A

Calculation Number 1: Atmospheric Concentrations of Volatiles from UOP Site Stream Channels

A. SOURCE TERM

Assumption: The compounds benzene and chlorobenzene, which have been detected in ground water but not surface water, volatilize almost immediately upon being discharged to the surface during low tide periods. During periods when large volumes of tidal water exist in the channel, immediate volatilization may not be the case because in this state there is sufficient water for solubilization rather than volatilization and also because the distance that volatiles would have to diffuse to the air-water interface is large. Because volatile compounds have not been detected in stream channels an assumption must be made about the concentration and quantity of these compounds in ground water discharging and subsequently volatilizing. It was assumed that the highest concentration of benzene or chlorobenzene in wells 10I, 17I, 18I, or 28I (the wells nearest the stream channels) would be used for calculations. These values are 35,000 ppb (3.5×10^7 ug per cubic meter of water) and 21,000 ppb (2.1×10^7 ug per cubic meter of water) for benzene and chlorobenzene, respectively. Both values were detected in 1985 samples from well 17I. In addition to benzene and chlorobenzene, the NJDEP has required that the same analysis be carried through for 1,2-diphenylhydrazine, toluene, and 1,1,2,2-tetrachloroethane. The highest concentrations in the wells near the stream channels were:

- 1,2-diphenylhydrazine: 2200 ppb (2.2×10^6 $\mu g/m^3$)
- Toluene: 160,000 ppb (1.6×10^8 $\mu g/m^3$)
- 1,1,2,2-tetrachloroethane: 3,800 ppb (3.8×10^6 $\mu g/m^3$).

Assumption: The rate of volatilization may be determined by the slower of two processes: The rate of ground water discharge, which then almost immediately emits volatiles, or the rate of bulk diffusion of volatiles in the discharged water to the air-water interface before volatilization can occur.

The surface area of the stream channel presumed to be influenced by well 17I is approximately 50 m long, 3.5 m half-wide (contaminated water is discharging on one side of the stream channel and is assumed to affect only one half the channel), and 1.2 m deep (water table to bottom of channel).

Bulk transfer of volatiles is reported to be approximately 10^{-5} m/sec (Ref: D. MacKay (1985) Air-water exchange coefficients, in Environmental Exposure from Chemicals, Volume I Neely and Blau, editors. CRC Press, Boca Raton pp. 91-99). Thus the volume of water that could be cleared of volatiles if it had a surface area of 50 x 3.5 is:

$3.5\ m \times 50m \times 10^{-5}\ m/s = 1.75 \times 10^{-3}$ cubic meters per second

$= 0.63$ cubic meters per hour.

The flux rate of ground water (taken from Table 8 of the Remedial Investigation) to this channel is 4.3×10^{-4}m/day (1.8×10^{-5}m/hour). This flux is applicable to a 1.2m x 50m surface area of the stream channel. Thus, the volume discharge of ground water is:

$1.2\ m \times 50\ m \times 1.8 \times 10^{-5}$ m/hour = .0011 cubic meters per hour

Therefore, the rate at which materials can volatilize far outstrips the availability of ground water at the surface so that emission rates of volatile to the air is limited by ground water flux. It will therefore be assumed all four volatile compounds are immediately volatilized from ground water as it is discharged to the surface and the emission rate is controlled by discharge (flux) rate, producing the following relation for the specific UOP case:

E = Emission rate = flux x concentration

E (benzene) = $.0011 m^3/hr \times 3.5 \times 10^7\ \mu g/m^3$

$= 38{,}500\ \mu g/hr$

E (chlorobenzene) = .0011 m^3/hr x 2.1 x 10^7 $\mu g/m^3$
= 23,100 µg/hr
E (1,2-diphenylhydrazine) = 2,400 µg/hr.
E (Toluene) = 176,000 µg/hr.
E (1,1,2,2-tetrachloroethane) = 4,180 µg/hr.

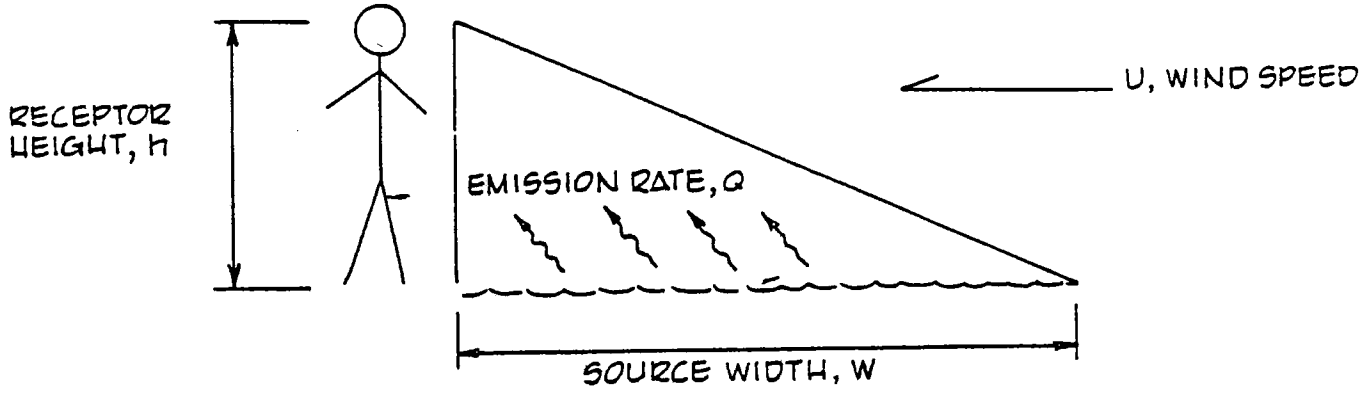
The air quality model, developed below, requires a source term, Q, in units of $\mu g/m^2$ sec. If one assumes that the E term above is being emitted in a homogeneous fashion from the 1.2 x 50 m surface of the stream bank:

Q = [E (µg/hr)/(1.2m x 50m)] x [1 hr/60 min x 1 min/60 sec]
Q (benzene) = 38,500 µg/hr x 1/$60m^2$ x 1 hr/3600 sec
= 1.78 x 10^{-1} $\mu g/m^2$ sec
Q (chlorobenzene) = 23,100 µg/hr x 1/$60m^2$ x 1 hr/3600 sec
= 1.07 x 10^{-1} $\mu g/m^2$ sec
Q (1,2-diphenylhydrazine) = 0.011 $\mu g/m^2$ sec.
Q (Toluene) = 0.81 $\mu g/m^2$ sec.
Q (1,1,2,2-tetrachloroethane) = 0.019 $\mu g/m^2$ sec.

B. AIR QUALITY SCREENING MODEL

A worst case situation for volatile emissions producing ambient air concentrations of volatile materials is for the emissions to be contained in a triangular area described by the maximum width, w, of the emission source and the height, h, of the receptors' breathing zone. This may be depicted graphically as in Figure A-1. The scenario is worst-case because:

1) The source contribution is maximized by using its maximum width.
2) The maximum height of mixing is presumed to be at the receptors breathing zone when, in fact, for certain conditions the height of mixing would be much higher and allow further dilution. For some conditions, the



$$\text{AMBIENT CONCENTRATION} = \frac{Q \cdot W}{U \cdot h}$$

Figure A-1 Graphic Representation of Air Quality Model

maximum height of mixing may pass beneath the receptors breathing zone.

3) No adjustment for lateral dispersion has been made (this is also why the model is run in 2 dimensions - it is essentially giving a concentration in a plane in which the individual is breathing). In the case of sources that extend for great distances in the lateral direction, the lack of adjustment is not extremely important, but for narrow sources the lack of lateral dispersion produces overestimate of concentration. In maximizing the source width at the UOP site, one places the receptor at the "end" of a stream channel where the lateral extent of the channel is in fact narrow (i.e. 7 m in the case of the location in question).

The ambient concentration, χ, in a model such as this is:

$$\chi = \frac{Q\ (\mu g/m^2\ sec)\ X\ W\ (m)}{u\ (m/sec)\ X\ h\ (m)}$$

where u = wind speed.

Because ambient concentration is inversely related to wind speed a low value was picked for this parameter. Using a 1 m/sec wind velocity, a 50 m source width (from the length value, above), and a receptor height of 2 m above the stream channel:

$$\chi\ (benzene) = ([1.78 \times 10^{-1}] \times 50)/(1 \times 2)$$
$$= 4.46\ \mu g/m^3$$

$$\chi\ (chlorobenzene) = ([1.07 \times 10^{-1}] \times 50)/(1 \times 2)$$
$$= 2.67\ \mu g/m^3$$

χ (1,2-diphenylhydrazine) = 0.275 $\mu g/m^3$

χ (toluene) = 20.3 $\mu g/m^3$

χ (1,1,2,2-tetrachloroethane) = 0.475 $\mu g/m^3$

In the case of long term exposures, such as would occur in a shop or office building placed on the site, correction must be made for distance from the stream channel, decreased concentration due to indoor exposure locale, and changing wind direction. The only parameter among these three that is easily obtainable is frequency of wind direction. From the wind rose of nearby Newark Airport (Figure A-2), it can be seen that the highest frequency of winds prevailing in any single direction is approximately 11%. This is approximately 18.5 hours (24 hours/day * 7 days * .11). Thus, the concentration of contaminants in air at a possible future facility built at the site may be regarded as less than the concentration calculated above, and the frequency of exposure for an immovable receptor will be no more than 18.5 hours per week.

C. REFINED AIR QUALITY MODEL

Review of the extremely conservative assumptions incorporated in the screening model and consideration of likely human exposure patterns led to development of a refined, more realistic air quality model and exposure scenario. This refined model calculates the annual average air concentration of chemicals emitted from the stream channel in the vicinity of a building which might be situated near the stream channel. This model corresponds with a long-term exposure scenario in which an individual works in or around a building for 40 hours per week over a 35-year career. The annual average concentration is the most appropriate concentration measure to use since it best reflects the long-term nature of a person's potential exposure at a given receptor point.

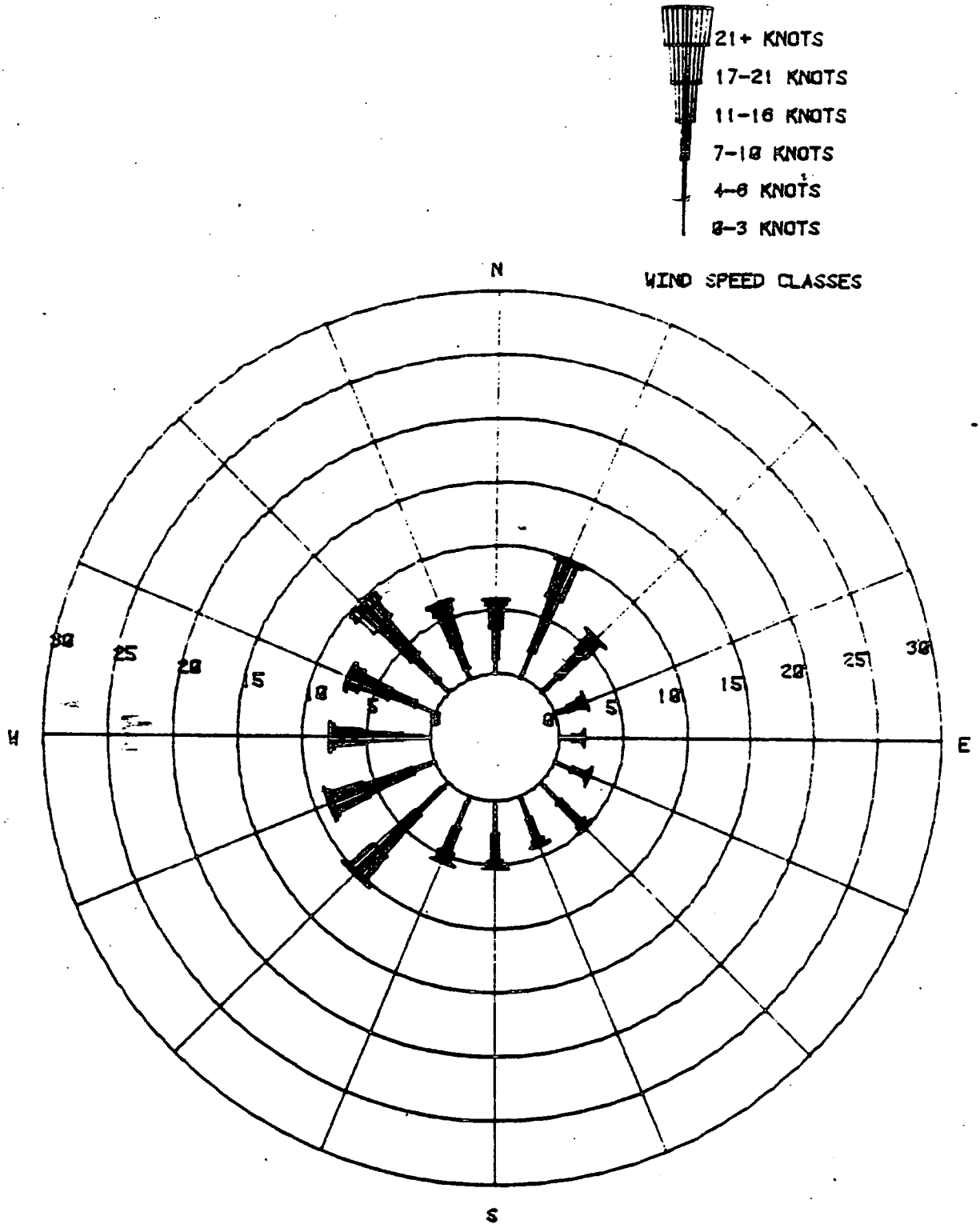


Figure A-2 Wind Rose for Newark, N.J. Airport

The volatile emissions from the stream channel were modeled using the U.S. EPA Industrial Source Complex (ISC) Model (EPA 1987) developed for continuous releases of this type. ISC is a computerized model that calculates ground-level or elevated airborne concentrations associated with continuous air emission sources. The model is comprised of mathematical formulas, based on field studies of atmospheric turbulence, that simulate the manner in which an emitted pollutant becomes distributed in the air as it travels downwind. This model is especially amenable to the present application because it has the ability to simulate the geometric configuration of a source, i.e. as a point, area or volume. The user specifies emission source parameters, such as area dimensions and emission rate receptor locations, and annual meteorological data, such as wind speed, wind direction, and atmospheric stability class frequencies. The stream channel is modeled as a rectangular area source, approximately 50 meters long by 7 meters wide, consistent with the screening model.

Climatological data from Newark International Airport collected from 1981 to 1985 were used for all ISC model runs. Newark climatology is appropriate because Newark is near the UOP Site and, therefore, subject to the same general climatological conditions.

The following model parameter values were assumed:

- Emission rates: the same chemical emissions used in the screening model were assumed here:
  E(benzene) = 38,500 µg/hr
  E(chlorobenzene) = 23,100 µg/hr
  E(toluene) = 176,000 µg/hr
  E(1,1,2,2 - tetrachloroethane) = 4,180 µg/hr

- Source area: the stream channel was defined as a 7m by 49m area, divided into contiguous 7m by 7m squares (ISC requires square area sources).

- Source orientation: the source area was aligned north-south lengthwise to reflect the orientation of the highest concentration stream channel adjacent to the UOP Site Area 1.

- Receptor grid: Sixteen (16) receptors were modeled surrounding the stream channel, each 50 feet (15 meters) away from the nearest of the stream channel, as shown in Figure A-3.

- Receptor height: Each receptor was assumed to be at a height of 1.5 meters (4.9 feet) above the ground surface to represent the breathing zone of a worker outside the building.

Table A-1 shows the modeled ambient air concentrations of the five chemicals of interest. Concentrations are shown at three locations: the highest impacted receptor, due east of the channel, and two locations where a new building would likely be located, due west and south of the channel.

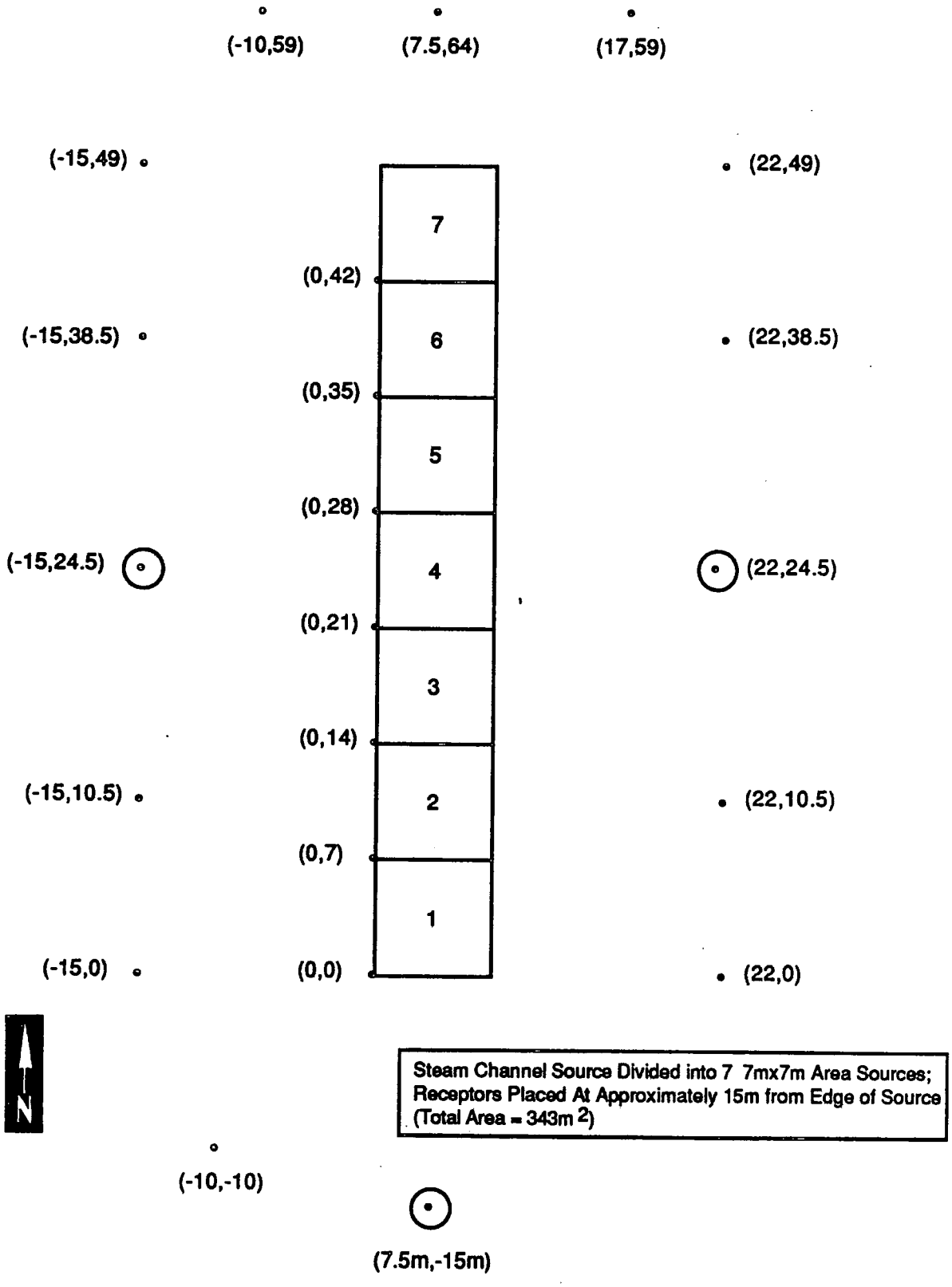


Figure A-3 Modeled Stream Channnel Area and Receptors

TABLE A-1

ISC MODELED AMBIENT AIR CONCENTRATIONS AT THEIR LOCATIONS NEAR THE UOP SITE STREAM CHANNEL

[Concentrations in $\mu g/m^3$]

Chemical	Receptor Location* East	West	South
Benzene	5.52×10^{-3}	2.96×10^{-3}	2.05×10^{-3}
Chlorobenzene	3.31×10^{-3}	1.78×10^{-3}	1.23×10^{-3}
1,2 - Diphenylhydrazine	3.44×10^{-4}	1.85×10^{-3}	1.28×10^{-4}
Toluene	2.52×10^{-2}	1.35×10^{-2}	9.37×10^{-3}
1,1,2,2 - Tetrachloroethane	5.99×10^{-4}	3.21×10^{-4}	2.23×10^{-4}

*Each receptor is located 15 meters from the nearest edge of the stream channel at an elevation of 1.5 meters above the ground.

Calculation Number 2: Air Concentrations for VOC Emissions from a Building Excavation

Purpose: To compute the air concentration and residence time for the compounds listed in Rev. 2. This calculation is for the construction worker scenario where soil contaminants are exposed to the air when a building site is excavated.

Assumption: Because the ground-water table is within a few feet of the ground surface, all buildings will be built on pads. This assumption is consistent with past building practices on the UOP site.

Method: Computation based on Model V pg. 16-18, Handbook of Chemical Property Estimation by W.J. Lyman, W.F. Reehl, D.H. Rosenblatt.

Set-up of Equations:

The flux:

$F = D_v R_o C_{ss}/d$ at $t>0$, $x = 0$

initial and boundary conditions; $c = c_s$ at $t>0$, $z = 0$

where c is concentration at the soil surface, c_s.

Therefore:

$F = D_v R_o C_{ss}/d$

where:

$R_o C_{ss} = C_{sa}$,

R_o = Ratio of concen. in air to concen. in soil (in most soils ratio is approximated by the ratio of Henry's Law to the soil adsorption coefficient.

D_v = the diffusion coefficient of the compound in the air, and

d = thickness of stagnant air layer in bottom of excavation; assumed to be 0.5 foot.

$$D_{V_{(air)}} = \frac{D_s}{E^{1/3}}$$

E = porosity = 0.40

D_s = diffusivity in soil = 4.3×10^{-5} ft^2/sec

$$D_v = \frac{4.3 \times 10^{-5}}{(0.40)^{1/3}} = 5.81 \times 10^{-5} \ ft^2/sec$$

$$R_o = \frac{K_H \times 41.6}{K_{oc} t_{oc}}$$

Benzene Computation:

Flux; $F = D_v \times R_o C_{ss}/d$

$= D_v \times C_{sa}/d$

where:

C_{sa} = Avg. C_{sa}

Avg. concentration of benzene =

(1-2' layer + 2-3' layer +3-4' layer)/3 = (857 µg/kg + 530 µkg + 530 µg/kg)/3 (concentration from Appendix C) = 639 ≃ 640 µg/kg

$$\text{Avg. } C_{sa} = 0.640 \text{ ppm} \times \frac{3.24 \text{ mg/m}^3}{1 \text{ ppm}}$$

$$= 2.07 \text{ mg/m}^3$$

$F = D_v \times C_{sa}/d$

$$F = 5.81 \times 10^{-5} \text{ ft}^2/\text{sec} \times 2.07 \text{ mg/m}^3 \times \frac{1}{1/2} \text{ ft} \times \frac{1 \text{ m}^3}{35.31 \text{ ft}^3}$$

$F = 6.81 \times 10^{-6}$ mg/sec ft^2

C_a - Air concentration at the surface of the soil

The effective emission rate q (mass/sec) out of sediment is q = FA where A is the surface area of contamination

$C_a = q/f$

where f is the cycling rate of air (vol./sec)

$q = FA$

where:

F = flux
A = area of foundation pad
= 200' x 50' (assumed based on typical remnant pads on site)
= 10,000 ft^2

Using the box-model:

$f = WVH$

where:

W = width = $\sqrt{\text{area}}$
V = wind verocity 3 MPH or 4 ft/sec
H = height of receptor

= 100 ft x 4 ft/sec x 5 ft
= 2000 ft^3/sec

$$Ca = \frac{FA}{f} = \frac{6.81 \times 10^{-6} \text{ mg/sec ft}^2 \times 10{,}000 \text{ ft}^2}{2000 \text{ ft}^3/\text{sec}}$$

$$= 3.4 \times 10^{-5} \frac{mg}{ft^3} \times 35.31 \frac{ft^3}{m^3}$$

$$= 1.20 \times 10^{-3} \text{ mg/m}^3$$

Calculation of Time to Dissipate Contaminant from Soil:

$$T = \frac{1}{F} d C_{ss} f_s$$

where:

F = flux
d = ft. thick of contaminated soil
P_s = avg. density of soil
= 1.9 g/cm^3
T = time in years
C_{ss} = avg. concentration of conta. soil ppm (from Appendix C)

$$T = \frac{\text{sec. ft}^2}{6.81\text{x}10^{-6}\ \text{mg}} \text{ x 4 ft x } \frac{1.81\ \text{ppm}}{1{,}000{,}000} \text{ x } \frac{1.93\ \text{g}}{\text{cm}^3} \text{ x 1,000 x}$$

$$\frac{1\ \text{cm}^3}{3.53\text{x}10^{-5}\ \text{ft}^3} \text{ x } \frac{1\ \text{yr}}{31{,}536{,}000\ \text{sec}} = 1.84 \text{ years}$$

Chlorobenzene Calculation:

Avg. C_{sa} concentration for chlorobenzene
= (60 + 236 + 236)/3
= 532 ppb/3
= 177 ppb = 0.177 ppm

$$\text{Avg } C_{sa} = 0.177 \text{ ppm} \times \frac{4.697 \text{ mg/m}^3}{1 \text{ ppm}}$$

$$= 0.83 \text{ mg/m}^3$$

$$F = D_v \times C_{sa}/d$$

$$= 5.81 \times 10^{-5} \text{ ft}^2/\text{sec} \times 0.83 \text{ mg/m}^3 \times \frac{1}{1/2 \text{ ft}} \times \frac{1 \text{ m}^3}{35.31 \text{ ft}^3}$$

$$= 2.73 \times 10^{-6} \text{ mg/sec ft}^2$$

C_a = Air concentration at the surface of the soil

$C_a = q/f$; $q = FA$

$f = WVH$
= 100 ft x 4 ft/sec x 5 ft
= 2000 ft^3/sec

$$C_a = \frac{FA}{f} = \frac{2.73 \times 10^{-6} \text{ mg/sec ft}^2 \times 10{,}000 \text{ ft}^2}{2000 \text{ ft}^3/\text{sec}}$$

$$= 1.36 \times 10^{-5} \frac{\text{mg}}{\text{ft}^3} \times \frac{35.31 \text{ ft}^3}{1 \text{ m}^3}$$

$$= 4.82 \times 10^{-4} \frac{mg}{m^3}$$

Calculation for time in years:

$$T = \frac{1}{F} d \, C_{ss} S_s$$

d = depth of contaminated soil

C_{ss} = Avg. concentration of contaminated soil = 11704 ppb/3

= 3.901 ppm

$$T = \frac{\text{sec. ft}^2}{2.73 \times 10^{-6} \text{ mg}} \times 4 \text{ ft} \times \frac{3.9 \text{ ppm}}{1{,}000} \times \frac{1.43 \text{ g}}{\text{cm}^3} \times \frac{\text{mg}}{\text{g}} \times$$

$$\frac{1 \text{ cm}^3}{3.53 \times 10^{-5} \text{ ft}^3} \times \frac{1}{31{,}536{,}000 \text{ sec.}}$$

= 9.9 years

1-2-Dichlorobenzene Calculation:

Avg. C_{sa} concentration for 1,2 Dichlorobenzene

= (30 + 93 + 93)/3

= 216/3

= 72 ppb = 0.072 ppm

Avg. C_{sa} = 0.072 ppb x 6.11 mg/m^3/1 ppm

= 0.44 mg/m^3

F = D_v C_{sa} (avg.)/d

= 5.81×10^{-5} $ft^2/sec \times 0.44$ $mg/m^3 \times \frac{1}{1/2 \text{ ft}} \times \frac{1m}{35.31 \text{ ft}^3}$

= 1.45×10^{-6} $mg/sec\text{-}ft^2$

C_a = Air concentration at the surface of the soil

$C_a = q/f$; $q = FA$

where:

F = flux

A = Area of foundation pad

= 200' x 50'

For box-model approach air cycling rate of an open area

F = 2000 ft^3/sec (as for previous compounds)

$$C_a = \frac{FA}{f} = \frac{1.45 \times 10^{-6} \text{ mg/sec ft}^2 \times 10{,}000 \text{ ft}^2}{2000 \text{ ft}^3/\text{sec}} \times \frac{35.31 \text{ ft}^3}{1m^3}$$

Calculation for time in years:

d = thickness of contaminated soil

C_{ss} = Avg. concentration of contaminated soil
= 46000/3 = 15333 ppb

$$T = \frac{1}{F} d\ C_{ss}\ \rho_s$$

$$= \frac{\text{sec-ft}^2}{1.45 \times 10^{-6}\ \text{mg}} \times 4\ \text{ft} \times \frac{15.33\ \text{ppm}}{10{,}000} \times \frac{1.93\ \text{g}}{\text{cm}^3} \times 1000\ \text{mg/g}$$

$$\times \frac{1\ \text{cm}^3}{3.53 \times 10^{-5}\ \text{ft}^3} \times \frac{1}{31536000\ \text{sec}}$$

= 73.3 years

Toluene Calculation:

Avg. C_{sa} concentration for toluene

= (1647 + 533 + 533)/3

= 2713/3

= 904 ppb = 0.904 ppm

Avg. C_{sa} = 0.904 ppm x 3.824 mg/m^3/1 ppm

= 3.45 mg/m^3

$F = D_v C_{sa} / d$

d = thickness of stagnant air (1/2 ft)

$F = 5.81 \times 10^{-5}$ $ft^2/sec \times 3.45$ $mg/m^3 \times \frac{1}{1/2 \text{ ft}}$

$= 1.13 \times 10^{-5}$ $mg/sec\text{-}ft^2$

C_a = Air concentration at the surface of the soil
C_a = q/f; q = FA
F = flux
A = area of foundation pad
= 200' x 50'

For box-model approach air cycling rate of an open area

f = 2000 ft^3/sec

$$C_a = \frac{FA}{f} = \frac{1.13 \times 10^{-5} \text{ mg/sec-ft}^2 \times 10{,}000 \text{ ft}^2}{2000 \text{ ft}^3/\text{sec}} \times \frac{35.31 \text{ ft}^3}{}$$

$= 1.99 \times 10^{-3}$ mg/m^3

Calculation for time in years:

$$T = \frac{1}{F^d} C_{ss(avg)} \rho_s$$

C_{ss} = Avg. concentration of cont. soil 200112/3 ppb
= 66704 ppb

$$T = \frac{\text{sec-ft}^2}{1.13 \times 10^{-5} \text{mg}} \times 4 \text{ ft} \times \frac{66.7 \text{ ppm}}{1000} \times \frac{1.93 \text{ g}}{\text{cm}^3} \times \frac{\text{mg}}{\text{g}}$$

$$\times \frac{1 \text{ cm}^3}{3.53 \times 10^{-5} \text{ ft}^3}$$

= 40.9 years

CALCULATION SUMMARY

Compound	Air Concentration, mg/m^3	Time in Years
Benzene	1.2×10^{-3}	1.8
Chlorobenzene	4.8×10^{-4}	9.9
1,2 dichlorobenzene	2.6×10^{-4}	73.3
Toluene	2.0×10^{-3}	40.9

APPENDIX B

HUMAN HEALTH RISK ASSESSMENT - UOP SITE, AREA 4

APPENDIX B

Human Health Risk Assessment - UOP Site, Area 4

B.1 Introduction

Ackerman's Creek, known as Area 4 of the UOP Site, is being incorporated into the Risk Assessment Report for the UOP Site as requested by the NJDEP. This area was not considered as part of the original scope of the draft risk assessment because this area (along with Area 3) was being evaluated on a separate approved schedule.

Ackerman's Creek consists of both natural and manmade interconnecting channels draining the associated wetlands which are dominated by Phragmites vegetation. The area is tidally influenced; Ackerman's Creek connects with Berry's Creek and ultimately the Hackensack River.

B.2 Ranking of Indicator Compounds

The hazard identification process identifies a subset of chemicals that are the most toxic, mobile, persistent, and prevalent chemicals present on the site and, therefore, are those which potentially pose the greatest adverse health effects due to exposure in Area 4. The selection of a subset of chemicals, called Indicator Chemicals, is necessary when there are more than 10 to 15 chemicals identified as on-site contaminants so that subsequent estimations of exposure point concentrations and chemical intakes are a more manageable task. This section describes the process of identifying the indicator chemicals from the compounds detected in Area 4. The selection process is analagous to that used for Areas 1, 1A, 2 and 5 (see Section 1.0).

Sediment samples and surface water samples were collected in the stream channels of Area 4 and analyzed for a variety of volatile organic compounds, semi-volatile organic compounds, PCBs and metals. The compounds detected in the sediments

(collected during Investigation Phases I, II and III) are listed in Table B-1. The compounds detected in the surface water samples (collected during Phase II) are listed in Table B-2. These tables also present the representative (mean) and maximum concentration of each contaminant detected and their frequency of detection (i.e., the number of samples with a detectable concentration compared with the number of samples collected) for each medium. The representative concentration for each contaminant in each medium was calculated as the arithmetic mean of all values for that contaminant. The concentration of chemicals in non-detect samples was assumed to be zero.

Table B-3 contains the toxicity values, the toxicological classification (potential carcinogen and/or noncarcinogen) and the U.S.EPAs rating value or weight-of-evidence score for each site contaminant. Tables B-4 and B-5 present the CT (Concentration X Toxicity Constant) values and IS (Indicator Scores) for the carcinogens detected in sediments and surface water, respectively. Tables B-6 and B-7 present the CT values and IS for noncarcinogens detected in sediment and surface water, respectively. Table B-8 lists the IS together with the ranking based on the IS and chemical rate and transport characteristics for both carcinogens and noncarcinogens detected in the sediment. Table B-9 list comparable information for surface water contaminants.

Based on the review of the information provided in these tables, it has been determined that the indictor chemicals for both the sediment and surface water for Area 4 are:

- Arsenic
- Benzene
- Carcinogenic polynuclear aromatic hydrocarbons (PAH):
  - benzo[a]anthracene
  - benzo[a]pyrene

TABLE B-1

MAXIMUM AND REPRESENTATIVE CONCENTRATION AND FREQUENCY OF CONTAMINANTS DETECTED IN AREA 4 SEDIMENTS

Chemical (CAS No.)	Koc Value											Soil (mg/kg)						
		Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos
ACENAPHTHENE (000083-32-9)	4.60E3	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-4.00E-1	3.60E-2	11	1	--	--	--	--
ACENAPHTHYLENE (000208-96-8)	2.50E3	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.10E0	1.00E-1	11	1	--	--	--	--
ANTHRACENE (000120-12-7)	1.40E4	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-6.00E-1	8.60E-2	11	0	--	--	--	--
BENZ(A)ANTHRACENE (000056-55-3)	1.38E6	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-4.00E0	4.52E-1	11	5	--	--	--	--
BENZO(B)FLUORANTHENE (000205-99-2)	5.50E5	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-6.00E0	6.99E-1	11	6	--	--	--	--
BENZO(K)FLUORANTHENE (000207-08-9)	5.50E5	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-6.00E0	6.99E-1	11	6	--	--	--	--
BIS(2-ETHYLHEXYL)PHTHALATE (000117-81-7)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-2.20E1	6.56E0	11	8	--	--	--	--
CHROMIUM VI AND COMPOUNDS (007440-47-3)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-4.78E4	6.28E3	31	31	--	--	--	--
CHRYSENE (000218-01-9)	2.00E5	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-2.80E0	3.96E-1	11	6	--	--	--	--
CYANIDES (000057-12-5)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.10E1	1.63E0	11	5	--	--	--	--
N-NITROSODIPHENYLAMINE (000156-10-5)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-2.00E0	1.82E-1	11	1	--	--	--	--
O-XYLENE (000095-47-6)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.50E1	3.85E0	4	2	--	--	--	--
M-XYLENE (000108-38-3)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-4.10E1	1.07E1	4	2	--	--	--	--
P-XYLENE (000106-42-3)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-9.80E0	2.45E0	4	1	--	--	--	--
IRON AND COMPOUNDS (015438-31-0)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-7.50E3	4.01E3	7	7	--	--	--	--
POLYCHLORINATED BIPHENYLS (001336-36-3)	5.30E5	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-5.68E2	1.21E2	27	22	--	--	--	--
FLUORANTHENE (000206-44-0)	3.80E4	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-2.40E0	4.05E-1	11	6	--	--	--	--
FLUORENE (000086-73-7)	7.30E3	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.10E0	1.00E-1	11	1	--	--	--	--
PHENANTHRENE (000085-01-8)	1.40E4	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-5.80E0	7.00E-1	11	3	--	--	--	--
PYRENE (000129-00-0)	3.80E4	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-2.20E0	3.77E-1	11	5	--	--	--	--
MAGNESIUM+ (000072-43-5)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
CHLOROTOLUENE, O- ( )	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.00E2	2.79E1	4	4	--	--	--	--
MANGANESE AND COMPOUNDS (007439-96-5)	--	--	--	--	--	--	--	--	--	--	--	--	--	0.00E0-1.30E4	2.56E3	11	11	--	--	--	--

TABLE B-1 (cont.)

Chemical (CAS No.)	Koc Value												Soil (mg/kg)							
		Range		Repres	Tot	Pos	Range		Repres	Tot	Pos	Range		Repres	Tot	Pos	Range		Repres	Tot	Pos
ETHYLBENZENE (000100-41-4)	1.10E3	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-1.30E1		3.41E0	4	2
1,3-DICHLOROBENZENE (000541-73-1)	1.70E3	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-1.40E1		9.73E-1	15	2
1,4-DICHLOROBENZENE (000106-46-7)	1.70E3	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-2.90E1		2.49E0	15	6
TOLUENE (000108-88-3)	3.00E2	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-1.10E3		3.96E1	4	4
1,2,4-TRICHLOROBENZENE (000120-82-1)	9.20E3	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-2.50E1		1.67E0	15	1
CHLOROBENZENE (000108-90-7)	3.30E2	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-6.90E0		2.68E0	4	2
1,2-DIPHENYLHYDRAZINE (000122-66-7)	4.18E2	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-7.80E0		1.50E0	11	3
PHENOL (000108-95-2)	1.42E1	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-3.80E1		5.92E0	11	10
BENZENE (000071-43-2)	8.30E1	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-3.80E1		1.26E1	4	4
1,2-DICHLOROBENZENE (000095-50-1)	1.70E3	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-1.60E3		1.24E2	15	12
CHROMIUM AND COMPOUNDS (007440-43-9)	--	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-4.30E0		1.68E0	11	11
BENZO(A)PYRENE (000050-32-8)	5.50E6	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-5.30E0		6.03E-1	11	6
ZINC AND COMPOUNDS (007440-66-6)	--	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-5.80E2		2.41E2	11	11
LEAD AND COMPOUNDS (INORGANIC) (007439-92-1)	--	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-1.30E2		6.90E1	11	11
ARSENIC AND COMPOUNDS (007440-38-2)	--	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-5.00E1		1.44E1	11	11
MERCURY AND COMPOUNDS (INORGANIC) (007439-97-6)	--	--		--	--	--	--		--	--	--	--		--	--	--	0.00E0-2.20E2		4.43E1	27	27

TABLE B-2

MAXIMUM AND REPRESENTATIVE CONCENTRATION AND FREQUENCY OF CONTAMINANTS DETECTED IN SURFACE WATER OF AREA 4

Chemical (CAS No.)	Koc Value	Surface water (mg/l)															
		Range	Repres	Tot Pos	Range	Repres	Tot Pos	Range	Repres	Tot Pos	Range	Repres	Tot Pos	Range	Repres	Tot Pos	
ACETONE (000067-64-1)	2.20E0	—	—	— —	—	—	— —	0.00E0-2.07E0	1.91E-1	11 2	—	—	— —	—	—	— —	
BIS(2-ETHYLHEXYL)PHTHALATE (000117-81-7)	—	—	—	— —	—	—	— —	0.00E0-8.00E-3	2.00E-3	11 3	—	—	— —	—	—	— —	
CHROMIUM VI AND COMPOUNDS (007440-47-3)	—	—	—	— —	—	—	— —	0.00E0-1.20E-2	2.00E-3	11 2	—	—	— —	—	—	— —	
CYANIDES (000057-12-5)	—	—	—	— —	—	—	— —	0.00E0-3.00E-2	3.00E-3	11 1	—	—	— —	—	—	— —	
CHLOROFORM (000067-66-3)	3.10E1	—	—	— —	—	—	— —	0.00E0-1.20E-2	1.00E-3	11 1	—	—	— —	—	—	— —	
IRON AND COMPOUNDS (015438-31-0)	—	—	—	— —	—	—	— —	0.00E0-7.20E-1	1.91E-1	5 3	—	—	— —	—	—	— —	
MAGNESIUM+ (000072-43-5)	—	—	—	— —	—	—	— —	0.00E0-3.10E0	9.44E-1	11 11	—	—	— —	—	—	— —	
1,1,1-TRICHLOROETHANE (000071-55-6)	1.52E2	—	—	— —	—	—	— —	0.00E0-8.00E-3	2.00E-3	11 4	—	—	— —	—	—	— —	
METHYL ETHYL KETONE (000078-93-3)	4.50E0	—	—	— —	—	—	— —	0.00E0-8.00E-3	1.00E-3	11 1	—	—	— —	—	—	— —	
TOLUENE (000108-88-3)	3.00E2	—	—	— —	—	—	— —	0.00E0-5.30E-2	5.00E-3	11 2	—	—	— —	—	—	— —	
DICHLOROMETHANE (000075-09-2)	8.80E0	—	—	— —	—	—	— —	0.00E0-4.70E-1	4.70E-2	11 4	—	—	— —	—	—	— —	
1,1-DICHLOROETHANE (000075-34-3)	30.00E0	—	—	— —	—	—	— —	0.00E0-1.50E-2	2.00E-3	11 2	—	—	— —	—	—	— —	
VINYL CHLORIDE (000075-01-4)	5.70E1	—	—	— —	—	—	— —	0.00E0-1.60E-2	1.00E-3	11 1	—	—	— —	—	—	— —	
1,2-DICHLOROPROPANE (000078-87-5)	51.00E0	—	—	— —	—	—	— —	0.00E0-9.00E-3	1.00E-3	11 1	—	—	— —	—	—	— —	
BENZENE (000071-43-2)	8.30E1	—	—	— —	—	—	— —	0.00E0-8.00E-3	1.00E-3	11 2	—	—	— —	—	—	— —	
CHLOROBENZENE (000108-90-7)	3.30E2	—	—	— —	—	—	— —	0.00E0-2.10E-2	3.00E-3	11 2	—	—	— —	—	—	— —	
1,1,2,2-TETRACHLOROETHANE (000079-34-5)	1.18E2	—	—	— —	—	—	— —	0.00E0-1.90E-2	2.00E-3	11 2	—	—	— —	—	—	— —	
1,2-DIPHENYLHYDRAZINE (000122-66-7)	4.18E2	—	—	— —	—	—	— —	0.00E0-4.50E-2	4.00E-3	11 1	—	—	— —	—	—	— —	
TRANS-1,2-DICHLOROETHYLENE (000540-59-0)	5.90E1	—	—	— —	—	—	— —	0.00E0-2.00E-1	2.90E-2	11 4	—	—	— —	—	—	— —	
1,2-DICHLOROBENZENE (000095-50-1)	1.70E3	—	—	— —	—	—	— —	0.00E0-1.05E0	1.17E-1	11 4	—	—	— —	—	—	— —	
ZINC AND COMPOUNDS (007440-66-6)	—	—	—	— —	—	—	— —	0.00E0-1.15E-1	7.20E-2	11 11	—	—	— —	—	—	— —	
CADMIUM AND COMPOUNDS (007440-43-9)	—	—	—	— —	—	—	— —	0.00E0-1.40E-2	4.00E-3	11 8	—	—	— —	—	—	— —	
ARSENIC AND COMPOUNDS (007440-38-2)	—	—	—	— —	—	—	— —	0.00E0-1.20E-2	1.00E-3	11 2	—	—	— —	—	—	— —	

TABLE B-2 (continued)

Chemical (CAS No.)	Koc Value	Surface water (mg/l)															
		Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos	Range	Repres	Tot	Pos
PHENOL (000108-95-2)	1.42E1	—	—	—	—	—	—	—	—	0.00E0-2.20E0	2.30E-1	11	11	—	—	—	—	—	—	—	—
TRICHLOROETHYLENE (000079-01-6)	1.26E2	—	—	—	—	—	—	—	—	0.00E0-2.30E-1	3.00E-2	11	4	—	—	—	—	—	—	—	—
LEAD AND COMPOUNDS (INORGANIC) (007439-92-1)	—	—	—	—	—	—	—	—	—	0.00E0-1.20E-1	5.80E-2	11	11	—	—	—	—	—	—	—	—

TABLE B-3

TOXICITY INFORMATION FOR CONTAMINANTS DETECTED IN AREA 4

Chemical	Toxicologic Class	Rating Value/EPA Category	H T	S T	A T
ACETONE	NC		0.00 E0	0.00 E0	0.00 E0
BIS(2-ETHYLHEXYL)PHTHALATE	PC	B2	5.71 E-4	2.86 E-8	5.71 E-3
	NC		0.00 E0	0.00 E0	0.00 E0
CHROMIUM IV AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
CYANIDES	NC		0.00 E0	0.00 E0	0.00 E0
CHLOROFORM	PC	B2	5.63 E-2	2.81 E-6	5.63 E-1
	NC		0.00 E0	0.00 E0	0.00 E0
IRON AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
MANGANESE AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
CHROMIUM III AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
1,1,1-TRICHLOROETHANE	NC	2 (oral) 2 (inhalation)	7.33 E-4	3.67 E-8	7.33 E-3
METHYL ETHYL KETONE	NC		7.75 E-3	3.87 E-7	7.75 E-2
DICHLOROMETHANE	PC	B2	0.00 E0	0.00 E0	0.00 E0
	NC	10 (oral) 10 (inhalation)	9.20 E-4	4.60 E-8	9.20 E-3
TOLUENE	NC	7 (oral) 7 (inhalation)	5.20 E-3	2.60 E-7	5.20 E-2
1,4-DICHLOROBENZENE	NC	4 (oral) 5 (inhalation)	5.19 E-2	2.60 E-6	3.61 E-1
1,1-DICHLOROETHANE	NC	7 (oral) 7 (inhalation)	2.58 E-2	1.29 E-6	2.58 E-1
BENZENE	PC	A	7.71 E-3	3.86 E-7	7.71 E-2
	NC	5 (oral) 10 (inhalation)	1.17 E-1	5.85 E-6	1.18 E2
1,2-DICHLOROPROPANE	NC	10 (oral) 10 (inhalation)	1.00 E-1	5.00 E-6	1.00 E0
VINYL CHLORIDE	PC	A	4.29 E-3	2.14 E-7	4.29 E-2
	NC	10 (oral) 10 (inhalation)	8.77 E-2	4.39 E-6	8.77 E-1
CHLOROBENZENE	NC	4 (oral) 1 (inhalation)	1.43 E-1	7.14 E-6	2.79 E-1
1,2-DICHLOROBENZENE	NC	4 (oral) 5 (inhalation)	5.19 E-2	2.60 E-6	3.61 E-1
TRANS-1,2-DICHLOROETHYLENE	NC	5 (oral) 5 (inhalation)	5.29 E-2	2.65 E-6	5.29 E-1
1,1,2,2-TETRACHLOROETHANE	PC	C	4.74 E-2	2.37 E-6	4.74 E-1
	NC	5 (oral) 5 (inhalation)	4.55 E-1	2.27 E-5	4.55 E0
ZINC AND COMPOUNDS	NC	8 (oral) 8 (inhalation)	1.07 E-1	5.33 E-6	1.07 E0
1,2-DIPHENYLHYDRAZINE	PC	B2	1.31 E-1	6.53 E-6	1.31 E0
	NC	10 (oral) 10 (inhalation)	3.34 E-1	1.67 E-5	3.34 E0
CADMIUM AND COMPOUNDS	PC	0	0.00 E0	0.00 E0	1.65 E1
	NC	10 (oral) 08 (inhalation)	4.45 E0	2.23 E-4	3.59 E2

TABLE B-3 (Continued)

Chemical	Toxicologic Class	Rating Value/EPA Category	W T	S T	R T
LEAD AND COMPOUNDS (INORGANIC)	NC	10 (oral) 10 (inhalation)	8.93 E-1	4.46 E-5	8.93 E0
TRICHLOROETHYLENE	PC	B2	2.00 E-3	1.00 E-7	2.00 E-2
	NC	5 (oral) 4 (inhalation)	1.65 E0	5.26 E-5	2.96 E1
ARSENIC AND COMPOUNDS	PC	A	4.07 E0	2.03 E-4	4.07 E1
	NC	9 (oral) 9 (inhalation)	1.80 E1	9.00 E-4	1.80 E2

TABLE B-3 (Continued)

Chemical	Toxicologic Class	Rating Value/EPA Category	H T	S T	A T
ACENAPHTHENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
ACENAPHTHYLENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
ANTHRACENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
BENZ(A)ANTHRACENE	PC	B2	5.81 E-1	2.91 E-5	5.81 E0
	NC		0.00 E0	0.00 E0	0.00 E0
BENZO(B)FLUORANTHENE	PC	B2	0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
BENZO(K)FLUORANTHENE	PC	D	0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
BIS(2-ETHYLHEXYL)PHTHALATE	PC	B2	5.71 E-4	2.86 E-8	5.71 E-3
	NC		0.00 E0	0.00 E0	0.00 E0
CHROMIUM IV AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
CHRYSENE	PC	B2	0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
CYANIDES	NC		0.00 E0	0.00 E0	0.00 E0
N-NITROSODIPHENYLAMINE					
O-XYLENE	NC		0.00 E0	0.00 E0	0.00 E0
P-XYLENE	NC		0.00 E0	0.00 E0	0.00 E0
MERCURY AND COMPOUNDS (ALKYL)	NC		0.00 E0	0.00 E0	0.00 E0
MANGANESE AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
POLYCHLORINATED BIPHENYLS	PC	B2	1.44 E0	7.21 E-5	1.44 E1
	NC		0.00 E0	0.00 E0	0.00 E0
FLUORANTHENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
FLUORENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
PHENANTHRENE	PC	D	0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
PYRENE	PC		0.00 E0	0.00 E0	0.00 E0
	NC		0.00 E0	0.00 E0	0.00 E0
CHROMIUM III AND COMPOUNDS	NC		0.00 E0	0.00 E0	0.00 E0
MERCURY AND COMPOUNDS (INORGANIC)	NC		0.00 E0	0.00 E0	0.00 E0
CHLOROTOLUENE, O-					
ETHYLBENZENE	NC	4 (oral) 4 (inhalation)	1.10 E-2	5.52 E-7	1.10 E-1
TOLUENE	NC	7 (oral) 7 (inhalation)	5.20 E-3	2.60 E-7	5.20 E-2
1,3-DICHLOROBENZENE	NC	4 (oral) 5 (inhalation)	5.19 E-2	2.60 E-6	3.61 E-1
1,4-DICHLOROBENZENE	NC	4 (oral) 5 (inhalation)	5.19 E-2	2.60 E-6	3.61 E-1
BENZENE	PC	A	7.71 E-3	3.86 E-7	7.71 E-2
	NC	5 (oral) 10 (inhalation)	1.17 E-1	5.85 E-6	1.18 E2

TABLE B-3 (Continued)

Chemical	Toxicologic Class	Rating Value/EPA Category	W T	S T	A T
CHLOROBENZENE	NC	4 (oral) 1 (inhalation)	1.43 E-1	7.14 E-6	2.79 E-1
1,2-DIPHENYLHYDRAZINE	PC	B1	1.31 E-1	6.53 E-6	1.31 E0
	NC	10 (oral) 10 (inhalation)	3.34 E-1	1.67 E-5	3.34 E0
PHENOL	NC	10 (inhalation)	1.00 E-1	5.02 E-6	2.49 E0
1,2,4-TRICHLOROBENZENE	NC	4 (oral) 1 (inhalation)	2.14 E-1	1.07 E-5	1.52 E0
CADMIUM AND COMPOUNDS	PC	0	0.00 E0	0.00 E0	1.65 E1
	NC	10 (oral) 08 (inhalation)	4.45 E0	2.23 E-4	3.59 E2
1,2-DICHLOROBENZENE	NC	4 (oral) 5 (inhalation)	5.19 E-2	2.60 E-6	3.61 E-1
ZINC AND COMPOUNDS	NC	8 (oral) 8 (inhalation)	1.07 E-1	5.33 E-6	1.07 E0
BENZO(A)PYRENE	PC	B2	4.55 E0	2.28 E-4	4.55 E1
	NC	8 (oral) 6 (inhalation)	2.67 E1	1.33 E-3	1.91 E1
LEAD AND COMPOUNDS (INORGANIC)	NC	10 (oral) 10 (inhalation)	8.93 E-1	4.46 E-5	8.93 E0
ARSENIC AND COMPOUNDS	PC	A	4.07 E0	2.03 E-4	4.07 E1
	NC	9 (oral) 9 (inhalation)	1.80 E1	9.00 E-4	1.80 E2

TABLE B-4

CT VALUES AND IS FOR CARCINOGENS DETECTED IN SEDIMENT IN AREA 4

Chemical	Soil CT				IS Value		Tentative Rank	
	Max	Repres	Max	Repres	Max	Repres	Max	Repres
ACENAPHTHENE	—	—	—	—	—	—	—	—
ACENAPHTHYLENE	—	—	—	—	—	—	—	—
ANTHRACENE	—	—	—	—	—	—	—	—
BENZ(A)ANTHRACENE	1.16 E-4	1.32 E-5	—	—	1.16 E-4	1.32 E-5	4	4
BENZO(B)FLUORANTHENE	—	—	—	—	—	—	—	—
BENZO(K)FLUORANTHENE	—	—	—	—	—	—	—	—
BIS(2-ETHYLHEXYL)PHTHALATE	6.29 E-7	1.88 E-7	—	—	6.29 E-7	1.88 E-7	7	7
CHRYSENE	—	—	—	—	—	—	—	—
POLYCHLORINATED BIPHENYLS	4.10 E-2	8.72 E-3	—	—	4.10 E-2	8.72 E-3	1	1
FLUORANTHENE	—	—	—	—	—	—	—	—
FLUORENE	—	—	—	—	—	—	—	—
PHENANTHRENE	—	—	—	—	—	—	—	—
PYRENE	—	—	—	—	—	—	—	—
1,2-DIPHENYLHYDRAZINE	5.09 E-5	9.79 E-6	—	—	5.09 E-5	9.79 E-6	5	5
BENZENE	1.47 E-5	4.86 E-6	—	—	1.47 E-5	4.86 E-6	6	6
CADMIUM AND COMPOUNDS	—	—	—	—	—	—	—	—
BENZO(A)PYRENE	1.21 E-3	1.37 E-4	—	—	1.21 E-3	1.37 E-4	3	3
ARSENIC AND COMPOUNDS	1.01 E-2	2.92 E-3	—	—	1.01 E-2	2.92 E-3	2	2

TABLE B-5

CT VALUES AND IS FOR CARCINOGENS DETECTED IN SURFACE WATER IN AREA 4

Chemical	Surface Water CT				IS Value				Tentative Rank			
	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
BIS(2-ETHYLHEXYL)PHTHALATE	—	—	—	—	4.57 E-6	1.14 E-6	—	—	—	—	4.57 E-6	1.14 E-6	8	8
CHLOROFORM	—	—	—	—	6.76 E-4	5.63 E-5	—	—	—	—	6.76 E-4	5.63 E-5	4	5
DICHLOROMETHANE	—	—	—	—	—	—	—	—	—	—	—	—	—	—
VINYL CHLORIDE	—	—	—	—	6.86 E-5	4.29 E-6	—	—	—	—	6.86 E-5	4.29 E-6	6	7
BENZENE	—	—	—	—	6.17 E-5	7.71 E-6	—	—	—	—	6.17 E-5	7.71 E-6	7	6
1,1,2,2-TETRACHLOROETHANE	—	—	—	—	9.01 E-4	9.48 E-5	—	—	—	—	9.01 E-4	9.48 E-5	3	3
1,2-DIPHENYLHYDRAZINE	—	—	—	—	5.90 E-3	5.24 E-4	—	—	—	—	5.90 E-3	5.24 E-4	2	2
CADMIUM AND COMPOUNDS	—	—	—	—	—	—	—	—	—	—	—	—	—	—
ARSENIC AND COMPOUNDS	—	—	—	—	4.88 E-2	4.07 E-3	—	—	—	—	4.88 E-2	4.07 E-3	1	1
TRICHLOROETHYLENE	—	—	—	—	4.60 E-4	6.00 E-5	—	—	—	—	4.60 E-4	6.00 E-5	5	4

TABLE B-6

CT VALUES AND IS FOR NONCARCINOGENS DETECTED IN SEDIMENT IN AREA 4

Chemical	Soil CT				IS Value				Tentative Rank			
	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
ACENAPHTHENE	--	--	--	--	--	--	--	--	--	--	--	--
ACENAPHTHYLENE	--	--	--	--	--	--	--	--	--	--	--	--
ANTHRACENE	--	--	--	--	--	--	--	--	--	--	--	--
BENZ(A)ANTHRACENE	--	--	--	--	--	--	--	--	--	--	--	--
BENZO(B)FLUORANTHENE	--	--	--	--	--	--	--	--	--	--	--	--
BENZO(K)FLUORANTHENE	--	--	--	--	--	--	--	--	--	--	--	--
BIS(2-ETHYLHEXYL)PHTHALATE	--	--	--	--	--	--	--	--	--	--	--	--
CHROMIUM VI AND COMPOUNDS	--	--	--	--	--	--	--	--	--	--	--	--
CHRYSENE	--	--	--	--	--	--	--	--	--	--	--	--
CYANIDES	--	--	--	--	--	--	--	--	--	--	--	--
N-NITROSODIPHENYLAMINE	--	--	--	--	--	--	--	--	--	--	--	--
O-XYLENE	--	--	--	--	--	--	--	--	--	--	--	--
M-XYLENE	--	--	--	--	--	--	--	--	--	--	--	--
P-XYLENE	--	--	--	--	--	--	--	--	--	--	--	--
IRON AND COMPOUNDS	--	--	--	--	--	--	--	--	--	--	--	--
POLYCHLORINATED BIPHENYLS	--	--	--	--	--	--	--	--	--	--	--	--
FLUORANTHENE	--	--	--	--	--	--	--	--	--	--	--	--
FLUORENE	--	--	--	--	--	--	--	--	--	--	--	--
PHENANTHRENE	--	--	--	--	--	--	--	--	--	--	--	--
PYRENE	--	--	--	--	--	--	--	--	--	--	--	--
MAGNESIUM+	--	--	--	--	--	--	--	--	--	--	--	--
CHLOROTOLUENE, O-	--	--	--	--	--	--	--	--	--	--	--	--
MANGANESE AND COMPOUNDS	--	--	--	--	--	--	--	--	--	--	--	--
ETHYLBENZENE	--	--	--	--	--	--	7.18 E-6	1.88 E-6	--	--	7.18 E-6	1.88 E-6	16	16
1,3-DICHLOROBENZENE	--	--	--	--	--	--	3.64 E-5	2.53 E-6	--	--	3.64 E-5	2.53 E-6	15	15
1,4-DICHLOROBENZENE	--	--	--	--	--	--	7.54 E-5	6.47 E-6	--	--	7.54 E-5	6.47 E-6	13	14
TOLUENE	--	--	--	--	--	--	2.86 E-4	1.03 E-5	--	--	2.86 E-4	1.03 E-5	8	13
1,2,4-TRICHLOROBENZENE	--	--	--	--	--	--	2.58 E-4	1.79 E-5	--	--	2.58 E-4	1.79 E-5	9	12
CHLOROBENZENE	--	--	--	--	--	--	4.93 E-5	1.91 E-5	--	--	4.93 E-5	1.91 E-5	14	11
1,2-DIPHENYLHYDRAZINE	--	--	--	--	--	--	1.30 E-4	2.50 E-5	--	--	1.30 E-4	2.50 E-5	12	10
PHENOL	--	--	--	--	--	--	1.91 E-4	2.97 E-5	--	--	1.91 E-4	2.97 E-5	11	9
BENZENE	--	--	--	--	--	--	2.22 E-4	7.37 E-5	--	--	2.22 E-4	7.37 E-5	10	8
1,2-DICHLOROBENZENE	--	--	--	--	--	--	4.16 E-3	3.22 E-4	--	--	4.16 E-3	3.22 E-4	5	7
CADMIUM AND COMPOUNDS	--	--	--	--	--	--	9.59 E-4	3.75 E-4	--	--	9.59 E-4	3.75 E-4	7	6
BENZO(A)PYRENE	--	--	--	--	--	--	7.05 E-3	8.02 E-4	--	--	7.05 E-3	8.02 E-4	3	5
ZINC AND COMPOUNDS	--	--	--	--	--	--	3.09 E-3	1.28 E-3	--	--	3.09 E-3	1.28 E-3	6	4
LEAD AND COMPOUNDS (INORGANIC)	--	--	--	--	--	--	5.80 E-3	3.08 E-3	--	--	5.80 E-3	3.08 E-3	4	3
ARSENIC AND COMPOUNDS	--	--	--	--	--	--	4.50 E-2	1.30 E-2	--	--	4.50 E-2	1.30 E-2	2	2
MERCURY AND COMPOUNDS (INORGANIC)	--	--	--	--	--	--	2.03 E-1	4.08 E-2	--	--	2.03 E-1	4.08 E-2	1	1

TABLE B-7

CT VALUES AND IS FOR NONCARCINOGENS DETECTED IN SURFACE WATER IN AREA 4

Chemical	Surface Water CT				CT		IS Value		Tentative Rank	
	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
ACETONE	—	—	—	—	—	—	—	—	—	—
BIS(2-ETHYLHEXYL)PHTHALATE	—	—	—	—	—	—	—	—	—	—
CHROMIUM VI AND COMPOUNDS	—	—	—	—	—	—	—	—	—	—
CYANIDES	—	—	—	—	—	—	—	—	—	—
CHLOROFORM	—	—	—	—	—	—	—	—	—	—
IRON AND COMPOUNDS	—	—	—	—	—	—	—	—	—	—
MAGNESIUM+	—	—	—	—	—	—	—	—	—	—
1,1,1-TRICHLOROETHANE	—	—	—	—	5.86 E-6	1.47 E-6	5.86 E-6	1.47 E-6	19	19
METHYL ETHYL KETONE	—	—	—	—	6.20 E-5	7.75 E-6	6.20 E-5	7.75 E-6	18	18
TOLUENE	—	—	—	—	2.76 E-4	2.60 E-5	2.76 E-4	2.60 E-5	17	17
DICHLOROMETHANE	—	—	—	—	4.32 E-4	4.32 E-5	4.32 E-4	4.32 E-5	15	16
1,1-DICHLOROETHANE	—	—	—	—	3.87 E-4	5.16 E-5	3.87 E-4	5.16 E-5	16	15
VINYL CHLORIDE	—	—	—	—	1.40 E-3	8.77 E-5	1.40 E-3	8.77 E-5	12	14
1,2-DICHLOROPROPANE	—	—	—	—	9.00 E-4	1.00 E-4	9.00 E-4	1.00 E-4	14	13
BENZENE	—	—	—	—	9.36 E-4	1.17 E-4	9.36 E-4	1.17 E-4	13	12
CHLOROBENZENE	—	—	—	—	3.00 E-3	4.29 E-4	3.00 E-3	4.29 E-4	11	11
1,1,2,2-TETRACHLOROETHANE	—	—	—	—	8.64 E-3	9.10 E-4	8.64 E-3	9.10 E-4	10	10
1,2-DIPHENYLHYDRAZINE	—	—	—	—	1.50 E-2	1.34 E-3	1.50 E-2	1.34 E-3	7	9
TRANS-1,2-DICHLOROETHYLENE	—	—	—	—	1.06 E-2	1.53 E-3	1.06 E-2	1.53 E-3	9	8
1,2-DICHLOROBENZENE	—	—	—	—	5.45 E-2	6.07 E-3	5.45 E-2	6.07 E-3	6	7
ZINC AND COMPOUNDS	—	—	—	—	1.23 E-2	7.70 E-3	1.23 E-2	7.70 E-3	8	6
CADMIUM AND COMPOUNDS	—	—	—	—	6.23 E-2	1.78 E-2	6.23 E-2	1.78 E-2	5	5
ARSENIC AND COMPOUNDS	—	—	—	—	2.16 E-1	1.80 E-2	2.16 E-1	1.80 E-2	3	4
PHENOL	—	—	—	—	2.20 E-1	2.30 E-2	2.20 E-1	2.30 E-2	2	3
TRICHLOROETHYLENE	—	—	—	—	2.42 E-1	3.15 E-2	2.42 E-1	3.15 E-2	1	2
LEAD AND COMPOUNDS (INORGANIC)	—	—	—	—	1.07 E-1	5.18 E-2	1.07 E-1	5.18 E-2	4	1

TABLE B-8

CHEMICAL RANKING[a] AND FATE AND TRANSPORT PROPERTIES FOR CONTAMINANTS DETECTED IN SEDIMENT IN AREA 4

Chemical	IS Values PC	IS Values NC	Ranking PC	Ranking NC	Water Solubility (mg/l)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm-m3/mole)	Koc	Half-Life (days) GW	SW	Soil	Air	IC
ACENAPHTHENE	--	--	--	--	3.42E0	1.55E-3	9.20E-5	4.60E3	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
ACENAPHTHYLENE	--	--	--	--	3.93E0	2.90E-2	1.48E-3	2.50E3	0.00E0 - 0.00E0	1.25E-1 - 0.00E0	0.00E0 - 0.00E0	5.50E0 - 0.00E0	
ANTHRACENE	--	--	--	--	4.50E-2	1.95E-4	1.02E-3	1.40E4	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
BENZ(A)ANTHRACENE	1.32E-5	--	4	--	5.70E-3	2.20E-8	1.16E-6	1.38E6	0.00E0 - 0.00E0	1.00E0 - 5.00E0	0.00E0 - 0.00E0	5.50E0 - 0.00E0	
BENZO(B)FLUORANTHENE	--	--	--	--	1.40E-2	5.00E-7	1.19E-5	5.50E5	0.00E0 - 0.00E0	1.00E0 - 2.00E0	0.00E0 - 0.00E0	5.50E0 - 0.00E0	
BENZO(K)FLUORANTHENE	--	--	--	--	4.30E-3	5.10E-7	3.94E-5	5.50E5	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
BIS(2-ETHYLHEXYL)PHTHALATE	1.88E-7	--	7	--	0.00E0	2.60E-7	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CHROMIUM VI AND COMPOUNDS	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CHRYSENE	--	--	--	--	1.80E-3	6.30E-9	1.05E-6	2.00E5	0.00E0 - 0.00E0	4.40E0 - 0.00E0	0.00E0 - 0.00E0	5.50E0 - 0.00E0	
CYANIDES	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	3.30E0 - 0.00E0	0.00E0 - 0.00E0	7.30E4 - 0.00E0	
N-NITROSODIPHENYLAMINE	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
O-XYLENE	--	--	--	--	1.75E2	1.00E1	0.00E0	0.00E0	0.00E0 - 0.00E0	1.50E0 - 9.00E0	0.00E0 - 0.00E0	0.58E0 - 0.00E0	
M-XYLENE	--	--	--	--	1.30E2	1.00E1	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
P-XYLENE	--	--	--	--	1.98E2	1.00E1	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
IRON AND COMPOUNDS	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
POLYCHLORINATED BIPHENYLS	8.72E-3	--	1	--	3.10E-2	7.70E-5	1.07E-3	5.30E5	0.00E0 - 0.00E0	2.00E0 -12.90E0	0.00E0 - 0.00E0	58.00E0 - 0.00E0	
FLUORANTHENE	--	--	--	--	2.06E-1	5.00E-6	6.46E-6	3.80E4	0.00E0 - 0.00E0	1.00E0 - 2.00E0	0.00E0 - 0.00E0	5.50E0 - 0.00E0	
FLUORENE	--	--	--	--	1.69E0	7.10E-4	6.42E-5	7.30E3	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
PHENANTHRENE	--	--	--	--	1.00E0	6.80E-4	1.59E-4	1.40E4	0.00E0 - 0.00E0	0.38E0 - 2.00E0	0.00E0 - 0.00E0	0.90E0 - 0.00E0	
PYRENE	--	--	--	--	1.32E-1	2.50E-6	5.04E-6	3.80E4	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.08E0 - 2.00E0	
MAGNESIUM+	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CHLOROTOLUENE, O-	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
MANGANESE AND COMPOUNDS	--	--	--	--	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
ETHYLBENZENE	--	1.88E-6	--	16	1.52E2	7.00E0	6.43E-3	1.10E3	0.00E0 - 0.00E0	1.50E0 - 7.50E0	0.00E0 - 0.00E0	1.46E0 - 0.00E0	
1,3-DICHLOROBENZENE	--	2.53E-6	--	15	1.23E2	2.28E0	3.59E-3	1.70E3	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
1,4-DICHLOROBENZENE	--	6.47E-6	--	14	7.98E1	1.18E0	2.89E-3	1.70E3	0.00E0 - 0.00E0	1.50E0 - 8.50E0	0.00E0 - 0.00E0	23.00E0 - 0.00E0	
TOLUENE	--	1.03E-5	--	13	5.35E2	2.81E1	6.37E-3	3.00E2	0.00E0 - 0.00E0	1.70E-1 - 0.00E0	0.00E0 - 0.00E0	1.30E0 - 0.00E0	
1,2,4-TRICHLOROBENZENE	--	1.79E-5	--	12	3.00E1	2.90E-1	2.31E-3	9.20E3	0.00E0 - 0.00E0	1.20E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CHLOROBENZENE	--	1.91E-5	--	11	4.66E2	1.17E1	3.72E-3	3.30E2	0.00E0 - 0.00E0	3.00E-1 - 0.00E0	0.00E0 - 0.00E0	3.50E0 - 0.00E0	
1,2-DIPHENYLHYDRAZINE	9.79E-6	2.58E-5	5	10	1.84E3	2.60E-5	3.42E-9	4.18E2	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
PHENOL	--	2.97E-5	--	9	9.30E4	3.41E-1	4.54E-7	1.42E1	0.00E0 - 0.00E0	6.20E-1 - 9.00E0	0.00E0 - 0.00E0	6.20E-1 - 9.00E0	
BENZENE	4.86E-6	7.37E-5	6	8	1.75E3	9.52E1	5.59E-3	8.30E1	0.00E0 - 0.00E0	1.00E0 - 6.00E0	0.00E0 - 0.00E0	6.00E0 - 0.00E0	
1,2-DICHLOROBENZENE	--	3.22E-4	--	7	1.00E2	1.00E0	1.93E-3	1.70E3	0.00E0 - 0.00E0	1.50E0 - 8.50E0	0.00E0 - 0.00E0	2.60E1 - 0.00E0	
CADMIUM AND COMPOUNDS	--	3.75E-4	--	6	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 - 0.00E0	
BENZO(A)PYRENE	1.37E-4	8.02E-4	3	5	1.20E-3	5.60E-9	1.55E-6	5.50E6	0.00E0 - 0.00E0	0.40E0 - 0.00E0	4.20E2 - 4.88E2	1.00E0 - 6.00E0	
ZINC AND COMPOUNDS	--	1.28E-3	--	4	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 -20.00E0	
LEAD AND COMPOUNDS (INORGANIC)	--	3.08E-3	--	3	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 - 0.00E0	
ARSENIC AND COMPOUNDS	2.92E-3	1.30E-2	2	2	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	5.00E0 - 0.00E0	
MERCURY AND COMPOUNDS (INORGANIC)	--	4.08E-2	--	1	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	

[a]Ranking based on representative concentrations.

TABLE B-9

CHEMICAL RANKING[a] AND FATE AND TRANSPORT PROPERTIES FOR CONTAMINANTS DETECTED IN SURFACE WATER IN AREA 4

Chemical	IS Values PC	IS Values NC	Ranking PC	Ranking NC	Water Solubility (mg/l)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm-m3/mole)	Koc	Half-Life (days) GW	SW	Soil	Air	IC
ACETONE	—	—	—	—	1.00E6	2.70E2	2.06E-5	2.20E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
BIS(2-ETHYLHEXYL)PHTHALATE	1.14E-6	—	8	—	0.00E0	2.00E-7	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CHROMIUM VI AND COMPOUNDS	—	—	—	—	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
CYANIDES	—	—	—	—	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	3.30E0 - 0.80E0	0.00E0 - 0.00E0	7.30E4 - 0.00E0	
CHLOROFORM	5.63E-5	—	5	—	8.20E3	1.51E2	2.87E-3	3.10E1	0.00E0 - 0.00E0	0.30E0 -30.00E0	0.00E0 - 0.00E0	80.00E0 - 0.00E0	
IRON AND COMPOUNDS	—	—	—	—	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
MAGNESIUM+	—	—	—	—	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
1,1,1-TRICHLOROETHANE	—	1.47E-6	—	19	1.50E3	1.23E2	1.44E-2	1.52E2	0.00E0 - 0.00E0	1.40E-1 - 7.00E0	0.00E0 - 0.00E0	8.03E2 - 1.75E3	
METHYL ETHYL KETONE	—	7.75E-6	—	18	2.68E5	7.75E1	2.74E-5	4.50E0	0.00E0 - 0.00E0	16.00E0 - 0.00E0	0.00E0 - 0.00E0	0.58E0 - 0.00E0	
TOLUENE	—	2.60E-5	—	17	5.35E2	2.81E1	6.37E-3	3.00E2	0.00E0 - 0.00E0	1.70E-1 - 0.00E0	0.00E0 - 0.00E0	1.30E0 - 0.00E0	
DICHLOROMETHANE	—	4.32E-5	—	16	2.00E4	3.62E2	2.03E-3	8.80E0	0.00E0 - 0.00E0	1.20E0 - 5.80E0	0.00E0 - 0.00E0	5.32E1 - 0.00E0	
1,1-DICHLOROETHANE	—	5.16E-5	—	15	5.50E3	1.82E2	4.31E-3	30.00E0	0.00E0 - 0.00E0	1.00E0 - 5.00E0	0.00E0 - 0.00E0	4.50E1 - 0.00E0	
VINYL CHLORIDE	4.29E-6	8.77E-5	7	14	2.67E3	2.66E3	8.19E-2	5.70E1	0.00E0 - 0.00E0	1.00E0 - 5.00E0	0.00E0 - 0.00E0	1.20E0 - 0.00E0	
1,2-DICHLOROPROPANE	—	1.00E-4	—	13	2.70E3	4.20E1	2.31E-3	51.00E0	0.00E0 - 0.00E0	1.40E0 - 7.70E0	0.00E0 - 0.00E0	80.00E0 - 0.00E0	
BENZENE	7.71E-6	1.17E-4	6	12	1.75E3	9.52E1	5.59E-3	8.30E1	0.00E0 - 0.00E0	1.00E0 - 6.00E0	0.00E0 - 0.00E0	6.00E0 - 0.00E0	
CHLOROBENZENE	—	4.29E-4	—	11	4.66E2	1.17E1	3.72E-3	3.30E2	0.00E0 - 0.00E0	3.00E-1 - 0.00E0	0.00E0 - 0.00E0	3.50E0 - 0.00E0	
1,1,2,2-TETRACHLOROETHANE	9.48E-5	9.10E-4	3	10	2.90E3	5.00E0	3.81E-4	1.18E2	0.00E0 - 0.00E0	0.04E0 - 0.00E0	0.00E0 - 0.00E0	5.84E2 - 0.00E0	
1,2-DIPHENYLHYDRAZINE	5.24E-4	1.34E-3	2	9	1.84E3	2.60E-5	3.42E-9	4.18E2	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	0.00E0 - 0.00E0	
TRANS-1,2-DICHLOROETHYLENE	—	1.53E-3	—	8	6.30E3	3.24E2	6.56E-3	5.90E1	0.00E0 - 0.00E0	1.00E0 - 6.00E0	0.00E0 - 0.00E0	2.10E0 - 0.00E0	
1,2-DICHLOROBENZENE	—	6.07E-3	—	7	1.00E2	1.00E0	1.93E-3	1.70E3	0.00E0 - 0.00E0	1.50E0 - 8.50E0	0.00E0 - 0.00E0	2.60E1 - 0.00E0	
ZINC AND COMPOUNDS	—	7.70E-3	—	6	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 -20.00E0	
CADMIUM AND COMPOUNDS	—	1.78E-2	—	5	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 - 0.00E0	
ARSENIC AND COMPOUNDS	4.07E-3	1.80E-2	1	4	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	5.00E0 - 0.00E0	
PHENOL	—	2.30E-2	—	3	9.30E4	3.41E-1	4.54E-7	1.42E1	0.00E0 - 0.00E0	6.20E-1 - 9.00E0	0.00E0 - 0.00E0	6.20E-1 - 9.00E0	
TRICHLOROETHYLENE	6.00E-5	3.15E-2	4	2	1.10E3	5.79E1	9.10E-3	1.26E2	0.00E0 - 0.00E0	1.00E0 - 9.00E1	0.00E0 - 0.00E0	3.70E0 - 0.00E0	
LEAD AND COMPOUNDS (INORGANIC)	—	5.18E-2	—	1	0.00E0	0.00E0	0.00E0	0.00E0	0.00E0 - 0.00E0	persistant	0.00E0 - 0.00E0	4.80E0 - 0.00E0	

[a]Ranking based on representative concentrations.

- benzo[b]fluoranthene
- chrysene
- dibenzo-[a,h]anthracene
- indeno(1,2,3-cd)pyrene

- Chromium
- Polychlorinated biphenyls
- 1,2-Dichlorobenzene
- Lead
- Zinc
- 1,2-Diphenylhydrazine
- 1,1,2,2-Tetrachloroethane

These contaminants are also among those chosen for Areas 1, 1A, 2 and 5.

B.3 Toxicity Assessment

The compounds which have been chosen for evaluation are a subset of those evaluated for other areas of the site. As such, the toxicity profiles may be found in the main body of the report (see Section 2).

B.4 Identification and Development of Exposure Pathways

At any site humans may potentially be exposed to contaminants in a variety of media such as soil, sediment, or water. The potential receptor may be exposed directly by ingestion, inhalation, or dermal absorption. Indirect exposure by consumption of contaminated biota is another potential exposure pathway. Potential indirect exposures will be evaluated in the ecological risk assessment being conducted for the site. In the stream channels, direct exposure to sediments and surface water will be examined. Inhalation of volatile organics from the stream channels is assessed in the body of the report (see 3.1, 5.1, 7.1).

B.4.1 Sediments

The organically rich sediments of the stream channels can act as sinks for a variety of contaminants. Hydrophobic compounds sorb strongly to the fine sediment particles. Many of the Indicator Compounds have been detected in the sediments. Persons who enter the wetland area may be potentially exposed to contaminants in the sediments via dermal absorption.

B.4.2 Surface Water

A number of Indicator Compounds have been found in the stream channel surface waters (see B.2). Generally, a given IC is found only once or occasionally, and in relatively low concentrations. The water in the stream channels is brackish and flushed with the tidal cycle. Persons encountering the water may be exposed by dermal contact. The potential for bioaccumulation of these contaminants into possible food items will be evaluated in the ecological risk assessment for the site. The surface water samples should adequately reflect exposure concentrations and are used to assess dermal contact.

B.5 Characterization of Potentially Exposed Populations

Potentially exposed populations have been identified in the body of the risk assessment report (see 4.0). Area 4 may be treated similarly. The potential for off-site exposure may exist via indirect pathways, and this will be evaluated in the ecological risk assessment. The potential for on-site exposure is basically restricted to young people and construction workers, and of these only young people are likely to be in Area 4. The Area 4 evaluation, therefore will address the potential activities of young people who may trespass on the UOP Site in the wetland area. Adults would be expected to derive lesser body burdens on a per kilogram basis than

adolescents (4.2.4), so the evaluation of the young persons precludes the necessity for assessing the impact on other individuals which may encounter the wetlands. It should be noted that although persons have been seen trespassing occasionally on the upland portions of the UOP site, no one has been observed in Area 4 during site visits.

B.6 Comparison of Environmental Concentration to Relevant and Applicable Standards

Contaminants in Area 4 are present in surface water and sediments. There are no relevant or applicable standards for permissable concentrations of contaminants in sediments. However, New Jersey does have a set of non-promulgated soil cleanup objectives. For purposes of comparison, the soil cleanup objectives and the indicator chemical concentrations in sediment are presented in Table B-10.

There are ambient water quality criteria for surface water. Those criteria apply to protection of aquatic species and protection of human health from exposure through drinking water or the food chain. The Ackerman's Creek surface waters are brackish and not a drinking water source. Environmental and food chain considerations, including a comparison of environmental concentration to relevant and applicable standards, will be addressed in the environmental risk assessment.

B.7 Calculation of Dose

Chemical intakes will be estimated with the aid of the exposure scenario relevant to the pathways as identified in B.4. In order to estimate exposure, some assumptions must be made concerning human activities that could lead to the exposure. Included in these assumptions are the magnitude of intake of contaminants from an environmental media, in this case surface water and sediment, and the frequency of the

TABLE B-10

COMPARISON OF INDICATOR CHEMICAL CONCENTRATIONS

IN SOIL AND NJ SOIL CLEANUP OBJECTIVES

UOP SITE, EAST RUTHERFORD, NJ

Compound	Sediment Conc. (mg/kg) Average	Max.	NJ Soil Cleanup[c] Objective (mg/kg)
Arsenic	12.3	50	20
Benzene	5.15	8.20	1
Carcinogenic PAH	1.17	6	10
Chromium	5000	24,500	100
PCB	160	568	1-5
1,2-DCB	191	1600	10[b]
Lead	67.4	130	250
Zinc	214	580	350
1,2-Diphenylhydrazine	5.5	7.8	10[b]
1,1,2,2-Tetrachloroethane	ND	ND	1[a]

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[a] Total Volatile Organic Compound Objective

[b] Total Base/Neutral Organic Compound Objective

[c] NJ Soil Cleanup Objectives are non-promulgated standards

exposure event. The type of individuals potentially at risk, young people, are discussed in Sections B.5 and 4.2.2. The exposure scenarios will be outlined below, and the underlying assumptions will be referenced in the main body of the risk assessment or stated explicitly below. Dose estimates will be converted to units of milligrams Indicator Chemical per kilogram body weight per day (mg/kg/day) to make them compatible with the dose-response relationships developed in Chapter 2.

B.7.1 Sediment

There is a possibility that young people might trespass and make direct contact with contaminated sediments in Area 4. Direct absorption of the material through the skin is the likely mechanism. Inadvertent ingestion of sediments is not likely to occur, and would be at extremely low levels compared to the potential for soil ingestion on dusty, terrestrial areas. Therefore, its contribution to total dose compared with dermal absorption would be insignificant, and it is not considered here. Assumptions used to evaluate the exposure of young people to contaminants in sediments are identical to those for soils (as delineated in 7.2.1) except as noted below.

On-site observations suggest that young people would visit Area 4 even less frequently than other areas of the site. It was assumed that a person might be exposed 1 day per week, one hour per day, during the summer months (June, July, and August) for a total of 13 exposure events per year.

For a "worst-case" scenario, the dose of compounds from dermal exposure to sediments at the UOP Site was calculated using the maximum concentration detected. A representative dose was calculated using the average sediment concentrations. The contaminant dose calculations from sediment absorption are:

Body dose (mg/kg/day) = Sediment concentration (ug/g) X Amount contacted (g/event) X Absorption Factor X 1/Body weight (kg) X Number of events/year X 1 year/365 Days X 1 mg/1000 ug

where:

- Amount contacted (g/event) = Amount sediment deposited (g/cm^2) X Exposed skin surface area (cm^2/event) = 0.0005 g/cm^2 X 4,140 cm^2/event = 2.07 g/event, which assumes that a person's arms to the elbows and a person's legs to the knees are exposed (see also 7.2).
- Dermal Absorption Factor = 0.02/24 hr X 1 hr = 0.00083 (see 7.2).
- Body weight = 35 kg (see 7.3).
- Number of events per year = 13 (one day per week in June, July, and August).

In the case of potential carcinogens, the exposure dose must be expressed on a per lifetime basis. A lifetime was assumed to be 70 years, and the exposure duration for the young person was assumed to be 5 years (see 7.3). Therefore, the above equation is further modified for carcinogens by multiplying by (5 years/70 year lifetime).

B.7.2 Surface Water

Dermal exposure to contaminants in surface water would be likely to occur concurrently with exposure to sediments, so the same exposure assumptions were used as noted above (B.7.1). Again, maximum concentrations were used for a "worst-case" scenario, and average concentrations were used for a more realistic exposure scenario. The body dose via direct dermal contact with surface water is estimated as:

Body Dose (mg/kg/day) = Surface water concentration (mg/L) X 1/Body weight (kg) X Number of hours/event X Exposed surface area (cm^2) X 1 L/1000 cm^3 X Permeability constant (cm/hr).

where:

- Body weight = 35 kg.
- Exposed surface area = 4,140 cm^2 (Anderson et al., 1985).
- Permeability constant = 8 x 10^{-4} cm/hr (U.S. EPA, 1988). This is the permeability constant for water, which must be used since chemical specific data are not available for the ICs. (See the U.S. EPA Superfund Exposure Assessment Manual, Appendix A, for more information). Using the constant for water assumes a solute transport mechanism.

For carcinogenic compounds the adjustment for lifetime exposure must be made as noted above (B.7.1 and 7.3).

B.8 Estimation of Risk

B.8.1 Non-Carcinogens

Noncarcinogenic risk estimates are determined by dividing body dose levels (calculated as described in Section B.7) for each noncarcinogen by the AIC (see Table 2-1). This results in a ratio or Hazard Index (HI). If the HI is less than or equal to one, the associated body dose level is likely to be without significant lifetime risk to human populations. If the ratio is greater than one, the predicted body dose level could potentially result in adverse health effects, although this is by no means a certainty as the relevant standards or guidelines

have conservative safety factors incorporated into their values, and are not exact numbers.

B.8.2 Carcinogens

Incremental carcinogenic risk estimates are determined by multiplying the body dose levels (calculated as described in Section B.7) by cancer potency slopes. To put these incremental risk levels into perspective, they should be evaluated against a reference (no-effect) level. However, for carcinogens, it is current USEPA policy that cancer induction is a non-threshold phenomenon. Therefore, any exposure poses some probability of causing cancer, and a "safe" (i.e. no-effect) level cannot be determined. USEPA guidelines indicate that the target total individual carcinogenic risk resulting from exposures at a Superfund site may range anywhere from 10^{-4} (that is, a dose corresponding to one excess cancer case in a population of 10,000) to 10^{-7} (one excess cancer case in a population of 10,000,000). Remedial alternatives being considered should be able to reduce total potential carcinogenic risks to individuals to levels within this range (U.S.EPA, 1986).

B.9 Presentation of Risks and Uncertainties

Risks from exposure to contaminants in sediments and surface water are presented in Tables B-11 through B-18. It can be seen from these tables that non-carcinogenic risk is insignificant in both media. The summary hazard indices (HI) for sediments range from 0.00024 (representative, Table B-11) to 0.0012 (maximum, Table B-13) and for surface water the HI ranges from 0.00015 (representative, Table B-15) to 0.00037 (maximum, Table B-17).

Total carcinogenic risk for each media was also very low. The total risk for sediments ranges from 7×10^{-8} (representative, Table B-12) to 3×10^{-7}, (maximum,

TABLE B-11

DIRECT CONTACT WITH SEDIMENT, NONCARCINOGENIC EFFECTS, REPRESENTATIVE CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Body Dose (mg/kg/day)} = \text{Soil Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{Number of events per year}}{365 \text{ days}} \times \frac{1 \text{ mg}}{1000 \text{ ug}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Amount of Soil Ingested (g/event)	Dermal TK Factor (%/hr)	Ingestion TK Factor (%)	Body Weight (kg)	No. of Events per year	Ingestion Body Dose (mg/kg/day)	Direct Contact Body Dose (mg/kg/day)	Standard/ Guideline (mg/kg/day)	Hazard Index
Arsenic	14.3640	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	2.51E-08	1.00E-03	2.51E-05
Chromium+3	5969.705	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	1.04E-05	1.00E+00	1.04E-05
Chromium+6	314.195	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	5.49E-07	5.00E-03	1.10E-04
1,2-Dichlorobenzene	123.7910	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	2.16E-07	9.00E-02	2.40E-06
Lead	69.0000	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	1.21E-07	1.40E-03	8.62E-05
Zinc	240.6360	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	4.21E-07	2.00E-01	2.10E-06
									SUMMARY HAZARD INDEX		2.36E-04

TABLE B-12

DIRECT CONTACT WITH SEDIMENT, CARCINOGENIC EFFECTS, REPRESENTATIVE CONCENTRATION

This table calculates estimated body doses and incremental cancer risks.

The equations to calculate body dose level and incremental cancer risks are:

$$\text{Body Dose (ug/kg/day)} = \frac{\text{Soil Concentration (ug/g)} \times \text{Amount Contacted (g/day)} \times \text{Fraction Absorbed} \times 1}{\text{Body Weight (kg)}} \times \frac{\text{Frequency of Contact}}{\text{365 days}} \times \frac{\text{No. of Years of Exposure}}{\text{70 yr. Lifetime}}$$

Incremental Cancer Risk = Body Dose x CAG Potency estimate

Coapound	Concentration (ug/g)	Amount of Soil Contacted (g/day)	Fraction Absorbed (%/lhr)	Body Weight (kg)	Frequency of Contact (days)	Fraction of Year Exposed	Years of Exposure (years)	Fraction of Lifetime Exposed	Body Dose (ug/kg/day)	Micrograms per milligram	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate
Arsenic	14.364	2.07	0.00083	35	13	0.04	5	0.07	1.79E-06	1000	1.5	2.69E-09
Benzene	12.625	2.07	0.00083	35	13	0.04	5	0.07	1.58E-06	1000	0.029	4.57E-11
PAHs	1.064	2.07	0.00083	35	13	0.04	5	0.07	1.33E-07	1000	11.5	1.53E-09
PCBs	121.365	2.07	0.00083	35	13	0.04	5	0.07	1.52E-05	1000	4.34	6.58E-08
1,2-Diphenylhydrazine	1.5	2.07	0.00083	35	13	0.04	5	0.07	1.87E-07	1000	0.8	1.50E-10
											TOTAL =	7.02E-08

TABLE B-13

DIRECT CONTACT WITH SEDIMENT, NONCARCINOGENIC EFFECTS, MAXIMUM CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Body Dose (mg/kg/day)} = \text{Soil Concentration (ug/g)} \times [(\text{Amount Contacted (g/event)} \times \text{Dermal TK Factor}) + (\text{Amount Ingested (g/event)} \times \text{Ingestion TK Factor})] \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{Number of events per year}}{365 \text{ days}} \times \frac{1 \text{ mg}}{1000 \text{ ug}}$$

$$\text{Hazard Index} = \frac{\text{Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/event)	Amount of Soil Ingested (g/event)	Dermal TK Factor (%/hr)	Ingestion TK Factor (%)	Body Weight (kg)	No. of Events per year	Ingestion Body Dose (mg/kg/day)	Direct Contact Body Dose (mg/kg/day)	Standard/ Guideline (mg/kg/day)	Hazard Index
Arsenic	50.0000	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	8.74E-08	1.00E-03	8.74E-05
Chromium+3	45410	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	7.94E-05	1.00E+00	7.94E-05
Chromium+6	2390	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	4.18E-06	5.00E-03	8.36E-04
1,2-Dichlorobenzene	1600.0000	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	2.80E-06	9.00E-02	3.11E-05
Lead	130.0000	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	2.27E-07	1.40E-03	1.62E-04
Zinc	580.0000	2.1	0.0	8.30E-04	1.00	35	13	0.00E+00	1.01E-06	2.00E-01	5.07E-06
									SUMMARY HAZARD INDEX		1.20E-03

TABLE B-14

DIRECT CONTACT WITH SEDIMENT, CARCINOGENIC EFFECTS, MAXIMUM CONCENTRATION

This table calculates estimated body doses and incremental cancer risks.

The equations to calculate body dose level and incremental cancer risks are:

$$\text{Body Dose (ug/kg/day)} = \text{Soil Concentration (ug/g)} \times \text{Amount Contacted (g/day)} \times \text{Fraction Absorbed} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{\text{Frequency of Contact}}{\text{365 days}} \times \frac{\text{No. of Years of Exposure}}{\text{70 yr. Lifetime}}$$

Incremental Cancer Risk = Body Dose x CAG Potency estimate

Compound	Concentration (ug/g)	Amount of Soil Contacted (g/day)	Fraction Absorbed (%/1hr)	Body Weight (kg)	Frequency of Contact (days)	Fraction of Year Exposed	Years of Exposure (years)	Fraction of Lifetime Exposed	Body Dose (ug/kg/day)	Micrograms per milligram	CAG Potency Estimate (mg/kg/day)-1	Risk Estimate
Arsenic	50	2.07	0.00083	35	13	0.04	5	0.07	6.24E-06	1000	1.5	9.37E-09
Benzene	38	2.07	0.00083	35	13	0.04	5	0.07	4.75E-06	1000	0.029	1.38E-10
PAHs	12.1	2.07	0.00083	35	13	0.04	5	0.07	1.51E-06	1000	11.5	1.74E-08
PCBs	568	2.07	0.00083	35	13	0.04	5	0.07	7.09E-05	1000	4.34	3.08E-07
1,2-Diphenylhydrazine	7.8	2.07	0.00083	35	13	0.04	5	0.07	9.74E-07	1000	0.8	7.79E-10
											TOTAL =	3.36E-07

TABLE B-15

DIRECT CONTACT WITH SURFACE WATER, NONCARCINOGENIC EFFECTS, REPRESENTATIVE CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Direct Contact Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \text{No. of Events (365 days)} \times \frac{1}{\text{Body Weight (kg)}} \times \text{Dermal TK Factor (\%)} \times \frac{\text{No. of Hours}}{\text{Event}} \times \text{Exposed Surface Area (cm\^2)} \times \frac{1\ \text{L}}{1000\ \text{cm\^3}} \times \text{Permeability Constant (cm/hr)} \times \frac{1\ \text{mg}}{1000\ \text{ug}}$$

$$\text{Ingestion Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \text{No. of Events (365 days)} \times \text{Amount Ingested (L/Event)} \times \text{Ingestion TK Factor (\%)} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{1\ \text{mg}}{1000\ \text{ug}}$$

$$\text{Hazard Index} = \frac{\text{Ingestion Body Dose (mg/kg/day)} + \text{Direct Contact Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/L)	No. of Events (365 days)	Body Weight (kg)	Dermal TK Factor (%/1hr)	No. of Hours (per Event)	Exposed Surface Area (cm^2)	Permeability Constant (cm/hr)	Direct Contact Body Dose (mg/kg/day)	Ingestion TK Factor (%)	Amount Ingested (L per Event)	Ingestion Body Dose (mg/kg/day)	Standard/ Guideline (mg/kg/day)	Hazard Index
Arsenic	1.0	13	35	1.00E+00	1.0	4140	8.00E-04	3.37E-09	1.00	0.0	0.00E+00	1.00E-03	3.37E-06
chromium+3	1.9	13	35	1.00E+00	1.0	4140	8.00E-04	6.40E-09	1.00	0.0	0.00E+00	1.00E+00	6.40E-09
Chromium+6	0.1	13	35	1.00E+00	1.0	4140	8.00E-04	3.37E-10	1.00	0.0	0.00E+00	5.00E-03	6.74E-08
1,2-Dichlorobenzene	117.0	13	35	1.00E+00	1.0	4140	8.00E-04	3.94E-07	1.00	0.0	0.00E+00	9.00E-02	4.38E-06
Lead	58.0	13	35	1.00E+00	1.0	4140	8.00E-04	1.95E-07	1.00	0.0	0.00E+00	1.40E-03	1.40E-04
Zinc	72.0	13	35	1.00E+00	1.0	4140	8.00E-04	2.43E-07	1.00	0.0	0.00E+00	2.00E-01	1.21E-06
												SUMMARY HAZARD INDEX	1.47E-04

TABLE B-16

DIRECT CONTACT WITH SURFACE WATER, CARCINOGENIC EFFECTS, REPRESENTATIVE CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Direct Contact Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \frac{\text{No. of Events}}{365\ \text{days}} \times \frac{1}{\text{Body Weight (kg)}} \times \text{Dermal TK Factor (\%)} \times \frac{\text{No. of Hours}}{\text{Event}} \times \text{Exposed Surface Area (cm}^2\text{)} \times \frac{1\ \text{L}}{1000\ \text{cm}^3} \times \text{Permeability Constant (cm/hr)} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{\text{No. of Years Exposed}}{70\ \text{year lifetime}}$$

$$\text{Ingestion Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \frac{\text{No. of Events}}{365\ \text{days}} \times \text{Amount Ingested (L/Event)} \times \text{Ingestion TK Factor (\%)} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{\text{No. of Years Exposed}}{70\ \text{year lifetime}}$$

$$\text{Incremental Risk} = \left(\text{Ingestion Body Dose (mg/kg/day)} + \text{Direct Contact Body Dose (mg/kg/day)}\right) \times \text{CAG Potency Estimate (mg/kg/day)}^{-1}$$

Compound	Concentration (ug/L)	No. of Events (365 days)	No. of Years Exposed (70 yr lifetime)	Body Weight (kg)	Dermal TK Factor (%)	No. of Hours (per Event)	Exposed Surface Area (cm^2)	Permeability Constant (cm/hr)	Direct Contact Body Dose (mg/kg/day)	Ingestion TK Factor (%)	Amount Ingested (L per Event)	Ingestion Body Dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)^-1	Incremental Risk
Arsenic	1.0	13	5	35	1.00	1.0	4140	8.00E-04	2.41E-10	1.00	0.0	0.00E+00	1.50E+00	3.61E-10
Benzene	1.0	13	5	35	1.00	1.0	4140	8.00E-04	2.41E-10	1.00	0.0	0.00E+00	2.90E-02	6.98E-12
PAHs	0.0	13	5	35	1.00	1.0	4140	8.00E-04	0.00E+00	1.00	0.0	0.00E+00	1.15E+01	0.00E+00
1,2-Diphenylhydrazine	4.0	13	5	35	1.00	1.0	4140	8.00E-04	9.63E-10	1.00	0.0	0.00E+00	8.00E-01	7.70E-10
1,1,2,2-Tetrachloroethane	2.0	13	5	35	1.00	1.0	4140	8.00E-04	4.81E-10	1.00	0.0	0.00E+00	2.00E-01	9.63E-11
													SUMMARY HAZARD INDEX	1.23E-09

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TABLE B-17

DIRECT CONTACT WITH SURFACE WATER, NONCARCINOGENIC EFFECTS, MAXIMUM CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Direct Contact Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \text{No. of Events (365 days)} \times \frac{1}{\text{Body Weight (kg)}} \times \text{Dermal TK Factor (\%)} \times \frac{\text{No. of Hours}}{\text{Event}} \times \text{Exposed Surface Area (cm}^2\text{)} \times \frac{1\text{ L}}{1000\text{ cm}^3} \times \text{Permeability Constant (cm/hr)} \times \frac{1\text{ mg}}{1000\text{ ug}}$$

$$\text{Ingestion Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \text{No. of Events (365 days)} \times \text{Amount Ingested (L/Event)} \times \text{Ingestion TK Factor (\%)} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{1\text{ mg}}{1000\text{ ug}}$$

$$\text{Hazard Index} = \frac{\text{Ingestion Body Dose (mg/kg/day)} + \text{Direct Contact Body Dose (mg/kg/day)}}{\text{Standard or Guideline (mg/kg/day)}}$$

Compound	Concentration (ug/L)	No. of Events (365 days)	Body Weight (kg)	Dermal TK Factor (%/hr)	No. of Hours (per Event)	Exposed Surface Area (cm^2)	Permeability Constant (cm/hr)	Direct Contact Body Dose (mg/kg/day)	Ingestion TK Factor (%)	Amount Ingested (L per Event)	Ingestion Body Dose (mg/kg/day)	Standard/ Guideline (mg/kg/day)	Hazard Index
Arsenic	12.0	13	35	1.00E+00	1.0	4140	8.00E-04	4.04E-08	1.00	0.0	0.00E+00	1.00E-03	4.04E-05
chromium+3	11.4	13	35	1.00E+00	1.0	4140	8.00E-04	3.84E-08	1.00	0.0	0.00E+00	1.00E+00	3.84E-08
Chromium+6	0.6	13	35	1.00E+00	1.0	4140	8.00E-04	2.02E-09	1.00	0.0	0.00E+00	5.00E-03	4.04E-07
1,2-Dichlorobenzene	1050.0	13	35	1.00E+00	1.0	4140	8.00E-04	3.54E-06	1.00	0.0	0.00E+00	9.00E-02	3.93E-05
Lead	120.0	13	35	1.00E+00	1.0	4140	8.00E-04	4.04E-07	1.00	0.0	0.00E+00	1.40E-03	2.89E-04
Zinc	115.0	13	35	1.00E+00	1.0	4140	8.00E-04	3.88E-07	1.00	0.0	0.00E+00	2.00E-01	1.94E-06
											SUMMARY HAZARD INDEX		3.69E-04

TABLE B-18

DIRECT CONTACT WITH SURFACE WATER, CARCINOGENIC EFFECTS, MAXIMUM CONCENTRATION

This table calculates estimated body doses and risk ratios.

The equations to calculate body dose level and risk ratio are:

$$\text{Direct Contact Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \frac{\text{No. of Events}}{365\ \text{days}} \times \frac{1}{\text{Body Weight (kg)}} \times \text{Dermal TK Factor (\%)} \times \frac{\text{No. of Hours}}{\text{Event}} \times \text{Exposed Surface Area (cm}^2\text{)} \times \frac{1\ \text{L}}{1000\ \text{cm}^3} \times \text{Permeability Constant (cm/hr)} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{\text{No. of Years Exposed}}{70\ \text{year lifetime}}$$

$$\text{Ingestion Body Dose (mg/kg/day)} = \text{Concentration (ug/L)} \times \frac{\text{No. of Events}}{365\ \text{days}} \times \text{Amount Ingested (L/Event)} \times \text{Ingestion TK Factor (\%)} \times \frac{1}{\text{Body Weight (kg)}} \times \frac{1\ \text{mg}}{1000\ \text{ug}} \times \frac{\text{No. of Years Exposed}}{70\ \text{year lifetime}}$$

$$\text{Incremental Risk} = \left[\text{Ingestion Body Dose (mg/kg/day)} + \text{Direct Contact Body Dose (mg/kg/day)}\right] \times \text{CAG Potency Estimate (mg/kg/day)}^{-1}$$

Compound	Concentration (ug/L)	No. of Events (365 days)	No. of Years Exposed (70 yr lifetime)	Body Weight (kg)	Dermal TK Factor (%)	No. of Hours (per Event)	Exposed Surface Area (cm^2)	Permeability Constant (cm/hr)	Direct Contact Body Dose (mg/kg/day)	Ingestion TK Factor (%)	Amount Ingested (L per Event)	Ingestion Body Dose (mg/kg/day)	CAG Potency Estimate (mg/kg/day)^-1	Incremental Risk
Arsenic	12.0	13	5	35	1.00	1.0	4140	8.00E-04	2.89E-09	1.00	0.0	0.00E+00	1.50E+00	4.33E-09
Benzene	8.0	13	5	35	1.00	1.0	4140	8.00E-04	1.93E-09	1.00	0.0	0.00E+00	2.90E-02	5.59E-11
PAHs	0.0	13	5	35	1.00	1.0	4140	8.00E-04	0.00E+00	1.00	0.0	0.00E+00	1.15E+01	0.00E+00
1,2-Diphenylhydrazine	45.0	13	5	35	1.00	1.0	4140	8.00E-04	1.08E-08	1.00	0.0	0.00E+00	8.00E-01	8.67E-09
1,1,2,2-Tetrachloroethane	19.0	13	5	35	1.00	1.0	4140	8.00E-04	4.57E-09	1.00	0.0	0.00E+00	2.00E-01	9.15E-10
												SUMMARY HAZARD INDEX		1.40E-08

Table B-14) and for surface water ranges from 1 x 10^{-9} (representative, Table B-16) to 1 x 10^{-8} (maximum, Table B-18). All carcinogenic risks are therefore below the U.S. EPA target risk range for site clean-up considerations. It can be concluded that direct contact with sediments and surface water in the Area 4 stream channels would not pose a threat to human health under reasonable worst case exposure conditions. All risks associated with Area 4 are summarized in Table B-19.

Uncertainties in the risk assessment process come from a variety of sources. These were discussed in detail in Section 10 of this report.

TABLE B-19

AREA 4 RISK SUMMARY TABLE

UOP SITE, EAST RUTHERFORD, NJ

Medium	Total Hazard Index[a]		Total Cancer Risk[b]	
	Maximum	Average	Maximum	Average
Sediments	0.0012	0.00024	3×10^{-7}	7×10^{-8}
Surface Water	0.00037	0.00015	1×10^{-8}	1×10^{-9}

[a] A value less than one indicates no risk.

[b] Risk values are excess chance of getting cancer.

APPENDIX C
DEMONSTRATION OF NEGLIGIBLE RISK DUE TO VOLATILIZATION OF SOIL CONTAMINANTS INTO THE AIR

APPENDIX C
DEMONSTRATION OF NEGLIGIBLE RISK DUE TO VOLATILIZATION OF SOIL CONTAMINANTS INTO THE AIR

In the ERT response (date Jun 6, 1988) to NJDEP comments (dated March 10, 1988) on Revision 1 of the Risk Assessment Report, it was demonstrated by calculation that the human health risk from benzene diffusing from soil into the air and subsequently being inhaled is negligible. In a later communication (date August 22, 1988) the NJDEP requested that the calculation be included in this report. This calculation has been expanded to include the indicator compounds; chlorobenzene, 1,2-dichlorobenzene and toluene in addition to benzene. The complete calculation follows.

PART 1: COMPUTATION OF VOLATILE CONCENTRATIONS IN AIR ABOVE CONTAMINATED SOIL

The volatilization of contaminants from soil into air occurs primarily by diffusion in the vapor phase if the contaminants preferentially partition into air rather than water or if the soil is dry. Vapor phase diffusion can be calculated using the following method.

Given a difference in the contaminant concentration in the soil pore air space at a depth x and the surface of the soil, a flux of gas will diffuse in the direction from high to low concentration. The flux is dependent on the diffusivity D for a particular gas to diffuse through the soil.

The net upward flux through the soil will be

$$F = D \frac{(C_{sa} - C_a)}{x} \qquad (1)$$

[Ref: Fick's Law of Diffusion, Handbook of Chemical Property Estimation Methods, W.J. Lyman, W.F. Reehl, D.H. Rosenblatt p. 15-9]

where $C_s a$ is the concentration in the air pores surrounding the soil and C_a is the air concentration at the surface of the soil.

The effective emission rate q (mass/sec) out of the sediment is $q = FA$ where A is area of contamination.

The concentration of the contaminant in the air above the contaminated soil is

$$C_a = q/f \tag{2}$$

where f is the cycling rate of the air (vol/sec).

A "box model" approach calculates the air cycling rate of an open area

$$f = (W)\ (V)\ (H) \tag{3}$$

where W is width dimension of contaminated area perpendicular to the wind direction, V is wind speed, and H is the height of the mixing zone.

Since q is also a function of C_a, equations 1 and 2 must be combined to yield:

$$C_a = \frac{rC_{sa}}{1 + r} \tag{4}$$

where $r = \frac{DA}{fx}$

Input Values:

$D = .04\ cm^2/sec = 4.3 \times 10^{-5}\ ft^2/sec$

[Ref: Lyman, W.J., W.F. Rechl, D.H. Rosenblatt Handbook of Chemical Properties Estimated Methods Table 16-6]

$A = (400\ ft.)\ (300\ ft.) = 120{,}000\ ft^2$

This represents the largest area of the site that could contribute air emissions to a receptor. This is approximated by an individual located at the center of combined Areas 1 and 1A.

$W = (A)^{1/2} = 350\ ft$

$V = 10\ ft/sec = 7\ mph$

The wind rose in the Risk Assessment shows an average velocity in the 7 to 10 knots range. 7 mph is chosen as a conservative value.

$H = 6\ ft$

6 ft is the height of the receptor.

$f = 21{,}000\ ft^3/sec$

For calculation purposes, the soil is conceptually divided into 3 layers: 1 to 2 ft, 2 to 3 ft and 3 to 4 ft. Therefore, x = 1, 2 or 3 ft depending on the soil layer of interest. The contaminants detected in the 0 to 2 ft. soil layer are assumed to be concentrated in the 1 to 2 ft. layer. Therefore detected concentration are doubled.

$$r = \frac{DA}{fx} = 0.00025 \qquad \text{(1 to 2 ft. layer)}$$

$r = 0.00013$ (2 to 3 ft. layer)

$r = 0.00008$ (3 to 4 ft. layer)

C_{sa} = Concentration of air in pores surrounding contaminated soil. Contaminant concentration in air surrounding soil is related to contaminant concentration in soil (C_{ss}) by

$$C_{sa} = \frac{41.6\ K_H\ C_{ss}}{K_{oc}\ f_{oc}}$$

[Ref: EPA-OHEA-E-187, 1986 Development of Advisory Levels for PCB Cleanup, Appendix A p. A-2]

where K_H is Henry's constant, K_{oc} is sediment (organic carbon) - water partition coefficient and f_{oc} is fraction organic carbon.

Table C-1 shows values of C_{ss}, C_{sa} and C_a computed for benzene, chlorobenzene, 1,2-dichlorobenzene and toluene at the UOP site.

TABLE C-1

SUMMARY OF SOIL AND AIR CONCENTRATIONS

Compound	C_{ss}, ppb	$K_{oc}f_{oc}$ (f_{oc}=1%)	$K_H x(41.6)$	C_{sa}, ppb	C_a, ppb
Benzene					
(1' to 2')	2,422*	.65	.23	857	.021
(2' to 3')	1,498	.65	.23	533	0.066
(3' to 4')	1,498	.65	.23	533	0.044
Chlorobenzene					
(1' to 2')	1,322*	3.3	0.15	60	0.015
(2' to 3')	5,191	3.3	0.15	236	0.029
(3' to 4')	5,191	3.3	0.15	236	0.020
1,2-dichlorobenzene					
(1' to 2')	6,374*	17	0.08	30	0.0075
(2' to 3')	19,813	17	0.08	93	0.012
(3' to 4')	19,813	17	0.08	93	0.0078
Toluene					
(1' to 2')	121,446*	1.18	0.016	1,647	0.412
(2' to 3')	39,333	1.18	0.016	533	0.067
(3' to 4')	39,333	1.18	0.016	533	0.044

*The listed concentration is twice the average concentration reported in Chapter 1 of the Risk Assessment. It is assumed that none of the compound is in the top foot of soil and that all the detected material is in the 1- to 2-foot layer.

PART 2: COMPUTATION OF TIME NEEDED TO COMPLETELY REMOVE A COMPOUND FROM THE SOIL BY VOLATILIZATION

The rate at which a compound is depleted from the soil (the flux rate) is estimated by the following equation:

$$F = \frac{D\ (C_{sa} - C_a)}{x}$$

where D = diffusivity: 4.3×10^{-5} ft^2/sec
x = depth to contaminated soil.

The concentrations C_{sa} amd C_a are listed in Table C-2.

An example computation for benzene is presented below. Results for the four compounds along with relevant input values are listed in Table C-3.

Computation for Benzene:

1 to 2 ft. soil layer:

$$C_{sa} = 0.857 \text{ ppm} \times \frac{3.24 \text{ mg/m}^3}{1 \text{ ppm}} = 2.78 \text{ mg/m}^3$$

$$C_a = 0.21 \times 10^{-3} \text{ppm} \times \frac{3.24 \text{ mg/m}^3}{\text{ppm}} = 0.68 \times 10^{-3} \text{ mg/m}^3$$

$$F = 4.3 \times 10^{-5} \frac{ft^2}{sec} \times \frac{2.78 \text{ mg/m}^3 - 0.68 \times 10^{-3} \text{mg/m}^3}{1 \text{ ft}}$$

$$\times \frac{1 \text{ m}^3}{35.31 \text{ ft}^3}$$

$$F = 0.34 \times 10^{-5} \frac{\text{mg}}{\text{sec - ft}^2}$$

TABLE C-2

AIR CONCENTRATIONS AND DISPERSION TIMES

Compound	Contaminated Layer, feet	x, ft.	Y*	Flux, mg./sec-ft^2	Air Concentration, Ca ppb	Air Concentration, Ca µg/m^3	Time to Disperse, Years
Benzene	1-2	1	3.24	0.34×10^{-5}	0.21	0.68	1.2
	2-3	2	3.24	0.104×10^{-5}	0.066	0.21	2.5
	3-4	3	3.24	0.070×10^{-5}	0.044	0.070	3.7
Chlorobenzene	1-2	1	4.70	0.034×10^{-5}	0.015	0.14	6.7
	2-3	2	4.70	0.067×10^{-5}	0.029	0.09	13.4
	3-4	3	4.70	0.044×10^{-5}	0.020	0.046	20.5
1,2-dichlorobenzene	1-2	1	6.11	0.022×10^{-5}	0.0075	0.073	50.2
	2-3	2	6.11	0.034×10^{-5}	0.012	0.048	101.0
	3-4	3	6.11	0.022×10^{-5}	-	-	-**
Toluene	1-2	1	3.82	0.767×10^{-5}	0.412	1.575	27
	2-3	2	3.82	0.128×10^{-5}	0.067	0.26	53
	3-4	3	3.82	0.083×10^{-5}	-	-	-**

*Factor for converting Air Concentrations (C_{sa} and C_a) from ppm to mg/m^3. 1 ppm = "y" mg/m^3. Reference: Verschueren (Verschueren, Karel, "Handbook of Environmental Data on Organic Chemicals," Second Edition, 1983.)

**Due to long time period required to disperse these compounds in the 1-2 ft. and 2-3 ft. layers, computation for 3-4 ft. layer is not need for risk calculations.

TABLE C-3

DAILY INHALATION INTAKE AND CORRESPONDING HEALTH RISK

Compound	Intake µg/kg/day	Cancer Potency Slope, µg/kg/day	Carcinogenic* Risk	AIC ug/kg/day	Non-carcinogenic** Hazard Index
Benzene	2.2×10^{-3}	2.6×10^{-5}	5.7×10^{-8}	-	-
Chlorobenzene	4.9×10^{-3}	-	-	5	9.8×10^{-4}
1,2-Dichlorobenzene	4.4×10^{-3}	-	-	40	1.1×10^{-4}
Toluene	6.2×10^{-2}	-	-	1000	6.2×10^{-5}
	Total		5.7×10^{-8}		1.2×10^{-3}

* Excess chance of getting cancer
** Hazard Index = Intake/AIC

The calculation of time for benzene to disperse from the 1- to 2-foot layer is:

$$T = \frac{1}{F} d C_s \rho_s$$

where $F = 0.34 \times 10^{-5} \frac{mg}{sec\text{-}ft^2}$

d = 1 ft thick of contaminated soil
C_s = concentration of contaminated soil - 2.422 ppm
ρ_s = average density of soil
= 1.9 g/cm^3 (fine sands)
T = time in years

$$T = \frac{sec\text{-}ft^2}{0.34 \times 10^{-5} mg} \times 1 \text{ ft} \times \frac{2.422}{1{,}000{,}000} \times \frac{1.93}{cm^3} \times \frac{1000mg}{g} \times \frac{1cm^3}{3.53 \times 10^{-5} ft^3}$$

$$\times \frac{1 \text{ yr.}}{31{,}536{,}000 \text{ sec.}} = 1.2 \text{ years}$$

Based on 2' to 3' and 3' to 4' layers:

Benzene @ 2' - C_{sa} = 0.53 ppm x 3.24 = 1.72 mg/m^3
C_a = 0

Benzene @ 3' - C_{sa} = 1.72 mg/m^3 (same soil concentration)

$$F_2 = 4.3 \times 10^{-5} \frac{ft^2}{sec} \times \frac{1.72 \text{ mg/m}^3}{2 \text{ ft.}} \frac{1m^3}{35.31 ft^3} = .104 \times 10^{-5} mg/sec - ft^2$$

$$F_{3'} = 4.3 \times 10^{-5} \times \frac{1.72 \text{ mg/m}^3}{3 \text{ ft.}} \frac{1 \text{m}^3}{35.31 \text{ ft}^3} = 0.070 \times 10^{-5} \text{mg/sec - ft}^2$$

$$T_{2'} = \frac{1'}{0.104 \times 10^{-5}} \times \frac{1.498}{1 \times 10^{6}} \times \frac{1.93 \text{g}}{3 \text{ cm}} \times \frac{1000 \text{ mg}}{\text{g}} \times \frac{1 \text{ cm}^3}{3.531 \times 10^{-5} \text{ ft}^3}$$

$$\times \frac{1 \text{ yr.}}{31{,}536{,}000 \text{ sec.}} = 2.5 \text{ years}$$

$$T_{3'} = \frac{1'}{0.070 \times 10^{-5}} \times \ldots\ldots\ldots$$

$$= 3.7 \text{ years}$$

PART 3: RISK COMPUTATIONS

The times and concentrations in the Table C-2 are used in the future site use scenario of the risk assessment to compute risk.

A sample computation for daily inhalation intake rates and resulting risk due to benzene is presented below.

$$\text{Intake} = (0.68 \text{ µg/m}^3 \times 1.2 \text{ yrs.} + 0.21 \times 2.5 + 0.14 \times 3.7) \times 1 \text{ M}^3\text{/hr} \times \frac{40 \text{ hrs.}}{\text{week}} \times \frac{1 \text{ week}}{7 \text{ days}} \times \frac{1}{70 \text{ kg}} \times \frac{1}{70 \text{ yrs.}}$$

$$= 2.2 \times 10^{-3} \text{ µg/kg/day}$$

Benzene Inhalation Cancer Potency Slope = 2.6×10^{-5} µg/kg/day

$$\text{Risk} = 2.2\times10^{-3}\ \mu g/kg/day \times 2.6\times10^{-5}/\mu g/kg/day$$

$$= 5.7\times10^{-8}$$

The intake rates and risks from all four compounds are shown in Table C-3. The computed risks are very low; 5.7×10^{-8} total carcinogenic risk and 1.2×10^{-3} total hazard index.

APPENDIX D
POTENTIAL OXIDATION OF Cr(III)

APPENDIX D
POTENTIAL OXIDATION OF Cr(III)

Background

Several factors control the oxidation and reduction (redox) reactions of chromium. Dissolved oxygen cannot oxidize Cr(III) under conditions found in soil; rather, the reaction occurs between Cr(III) and manganese oxides in soil. Cr(III) releases electrons to Mn(IV) in the oxide. The products of the reaction are Cr(VI) and Mn(II). Oxidation of Cr(III) is not shown in soils very low in manganese, or in acidic soil samples where the predominant manganese species is apparently in the reduced (Mn(II)) form. At common soil pH's, Cr(III) forms insoluble hydroxides and the insolubility of Cr(III) limits the oxidation reaction. As $Cr(OH)_3$ ages, it gradually transforms to the even less soluble oxide (Cr_2O_3). Thus, insolubility and the resulting limitation on the oxidation reaction increases with the 'age' of chromium in soil (Bartlett and James, 1979; Grove and Ellis, 1980a, 1980b; Eary and Ral, 1987).

Hexavalent chromium may be reduced to Cr(III) by reaction with organic matter, ferrous ions, or other electron acceptors. In aerobic soil, organic material or easily oxidised inorganic compounds will act as reducing agents. Under anaerobic conditions often found in marshy areas, ferrous ions and sulfide may act as reducing agents. Acidic pH encourages the reduction reaction. Researchers have found that, while some Cr(III) freshly applied to certain soils may initially oxidize to Cr(VI), it later reduces back to the trivalent form. (Grove and Ellis, 1980a; James and Bartlett, 1983; Bloomfield and Bruden, 1980)

Table D-1 summarizes a series of experiments on the oxidation of chromium that have been reported in the

TABLE D-1

CHROMIUM REACTION DATA

Soil Type	Initial Dose Cr(III)	Duration (days)	Variable	Percent of Dose Oxidized*	Comments	Reference
Raynham silt loam (Aeric Haplaquepts, 4.2% organic matter)	520 mg/kg	150	Moist soil, pH 6.0	1.5	Limed soils prepared with 3,000 mg/kg $CaCO_3$; Cr added as $CrCl_3$. Concentration of Cr(VI) still decreasing at end of experiment for moist soils (not steady state). "Air dried" soils dried and rewet, analogous to unsaturated soils.	Bartlett and James, 1979
			Moist, limed soil	0.5		
			Air dried soil, pH 5.4	0		
			Air dried, limed soil	0.001		
Field-moist Eldridge Ap soil (sandy over loamy, mixed, nonacid, mesic Aqnic Udorthent)	430 mg/kg	15	fresh $Cr(OH)_3$	12	Soil type chosen because of ability to rapidly oxidize Cr(III) and not adsorb Cr(VI) - represents worst case. Citrate used to model organic chelates. Concentration of Cr(VI) increased initially, then began to decrease; beginning to level off at 15 days.	James and Bartlett, 1983
			Cr-citrate addition	5		
			Aged $Cr(OH)_3$ w/citrate (pH 7.5)	1.2		
			Aged Cr $(OH)_3$	.05		
Aqueous Solution, pH 6.3	4 mg	25	Dosed with 5 g. MNO_2	4	Ratio MnO_2: Cr of 1252:1 (wt) or 749:1 moles Mn: moles Cr	Eary and Ral, 1987

*At close of experiment (i.e., at or near equilibrium)

literature. The results of these controlled laboratory experiments should be extrapolated cautiously to field conditions. Nonetheless, the results are encouraging: they indicate that very little oxidation of Cr(III) should occur.

Of chief concern is the extrapolation of short-term laboratory results to long-term field conditions. This appears to be a valid extrapolation, because the laboratory experiments reported in Table D-1 quickly reached or neared equilibrium.[a]

Bartlett and James (1979) reported that the amount of chromium oxidized increased rapidly during the first 24-hour period after dosing soil with $CrCl_3$, and then decreased slowly. They concluded that chromium was first oxidized to Cr(VI) by the soil, and then reduced back to Cr(III). Their data plots indicate dramatic decreases in Cr(VI) between one and one hundred days for moist soils, both limed and unlimed. After that time, the plot of concentration versus time leveled off, indicating that the systems were at or near equilibrium (in other words, that the oxidation and reduction reactions were nearly balanced). Their experiments with limed and unlimed dried/rewet samples (the latter analogous to field soils) indicated that only a small amount of Cr(III) oxidized at the beginning of the two-hundred day study. Oxidation decreased almost to zero, then increased slightly in about a month. However, the plot of Cr(VI) concentration versus time was essentially level over the study period, and the percentage of Cr(III) oxidized never reached even 1%.

The same team of researchers conducted a second series of experiments a few years later (James and Bartlett, 1983). In those experiments, as noted in Table D-1, they examined the

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a. Equilibrium: a state of dynamic balance, where rates of forward and reverse reactions (in this case, oxidation of Cr(III) and reduction of Cr(VI)) are equal, so that the system does not change with time.

oxidation of $Cr(OH)_3$ and Cr-citrate applied to soils, varying the "age" and thus the solubility of the $Cr(OH)_3$. They noted that three to six days after beginning the experiments the level of Cr(VI) declined slowly due to reduction back to Cr(III). Near the end of the fifteen-day experiments, the rates of change of the concentrations of Cr(VI) in the five treatments were nearly zero, indicating that oxidation of Cr(III) and reduction of Cr(VI) were nearly balanced.

The third study reported in Table D-1 (Eary and Ral, 1987) examined the potential for oxidation of Cr(III) under extreme pH conditions (generally pH 3.0-4.7, as necessary to keep Cr(III) dissolved) in aqueous solution. While these results cannot be as readily extrapolated to field soils as the other experiments, they do indicate a similar equilibrium effect. Data plots of oxidized chromium versus time leveled off fairly rapidly, generally between ten and twenty days.

Thus, the available experimental data support the notion that the concentrations of Cr(VI) and Cr(III) reach equilibrium over time. The short period required to reach equilibrium in laboratory experiments will not be duplicated under more complex field conditions. However, the percentages of Cr(III) oxidized at equilibrium in the laboratory studies may be cautiously extrapolated to long-term (i.e., 30 years) conditions in field soils.

Three of the experiments reported represent worst-case conditions with respect to oxidation of chromium. In the first of these, 12% of fresh $Cr(OH)_3$ applied to highly oxidizing soil later existed in the hexavalent state. Two variables made this experiment a worst case: (1) the soil was chosen for the experiment because of its dramatic ability to oxidize chromium, and (2) fresh chromic hydroxide leaches Cr(III) more easily (thus more is available to react) than the aged oxide, such as might be found in the soils at this facility. In the second worst-case experiment fresh $Cr(OH)_3$ was mixed with a chelating agent, which enhances chromium solubility, and the

same highly-oxidizing soil. 5% of the initial chromium dose existed as Cr(VI) after 15 days. The third experiment was part of a series designed to examine reaction kinetics. An aqueous solution of Cr(III) was overdosed with MnO_2, the compound which oxidizes Cr(III), at a weight ratio of 1252:1, yet only 4% of the Cr(III) oxidized after 25 days.

The remaining six experiments, conducted with a range of variables, showed oxidation of Cr(III) from unmeasureable levels to 1.5%. These experiments included conditions analogous to soils at the facility: "aged" Cr(III) in the soil; soil which had been successively dried and rewet (similar to unsaturated soils which are wet with rain water and then dry again); and soil which was kept moist (similar to saturated soil at this facility). Data produced by the preliminary soil sampling has enabled a better site-specific interpretation of these laboratory results.

Soil Characteristics

Previous site investigations have provided both quantitative and qualitative data relevent to this analysis. The data obtained during th Phase II investigation (Geraghty and Miller, 1985) provides essential quantitative information:

- The soil is basic, with pH of 7.8 (calculated interior average).
- The concentration of total manganese generally ranges between 44 and 1600 mg/kg, with an interior average concentration of 328 mg/kg.

Two qualitative observations provide furthur infomration:

- Hexavalent chromium was not detected in the sludge in the wastewater lagoons, despite a manganese concentration of 680 mg/kg. Although the soils and

sludge differ in context, the absence of hexavalent chromium in sludge is a general indication that the age of the chromium and the site environment preserved chromium in the trivalent state.

- Much of the soil is saturated. Marshy soil may well provide an anaerobic, reducing environment.

Interpretation of Results

As described above, the key variables in the oxidation of Cr(III) are the solubility of Cr(III) at the soil pH and the resulting availability for reaction, and the presence of oxidized manganese. The results of these soil analyses are discussed below.

Most of the UOP soil samples were basic. Neutral or basic pH implies that Cr(III) is essentially insoluble because it has formed the precipitated oxide or hydroxide.[b] The solubility limitation discourages both the rate and extent of oxidation of Cr(III).

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b. The theoretical concentration of Cr(III) in water in equilibrium with solid $Cr(OH)_3$ at pH 7.4 (the average soil pH for these samples) is 0.087 mg/l. The predominant species is $Cr(OH)_2^+$, at 0.075 mg/l. The mobility of Cr(III) is further limited by adsorbtion in soils.

Researchers have found that when experimenting with Cr(III), particularly in soil, they must maintain a pH below 5 to prevent precipitation (Griffin et. al. 1977; James and Bartlett, 1983; Eary and Ral, 1987). U.S. EPA has concluded, with respect to the application of sewage sludge to cropland, that Cr(III) is relatively innocuous because it is essentially insoluble in soils with pH > 5.5 (EPA 1976, 1977).

Most of the soil samples contained relatively low levels of manganese. The interior average concentration of manganese in site soils is 328 mg/kg. By comparison, manganese is found in concentrations of 100 to 4000 mg/kg in most soils (Adriano, 1986). Thus, the concentration of total manganese in the soil is relatively low.

Only a portion of the total manganese in the soil is oxidized, and of the oxidized fraction, probably only a smaller portion may react with Cr(III) (Bartlett and James, 1979). Thus, availability of manganese may limit the oxidation reaction.

The potential for oxidation was further evaluated by comparing the site soils with the experimental results reported in Table D-1. The site soils can generally be characterized as having neutral to basic pH; relatively low concentrations of manganese; "aged" $Cr(OH)_3$; and being successively dried and rewet (unsaturated soil) or continually wet (saturated soil). These conditions generally correspond to: (1) Bartlett and James' experiments with moist or air-dried limed soils, where only 0.001-1.5% of the applied chromium was oxidized at or near equilibrium; and (2) James and Bartletts' later experiments with aged $Cr(OH)_3$, with or without organic material added, where 1.2 and 0.5% respectively of the applied Cr(III) oxidized at or near equilibrium.

Conclusion

In general, the soil at the UOP site is basic and relatively low in total manganese content. These conditions limit the potential oxidation of Cr (III) to Cr (VI). Other site conditions may also limit oxidation reactions. For example, marshy soil typically provides a reducing environment which would limit oxidation reactions.

A comparison of site conditions to experimental results reported in the literature indicates that no more than a few percent of residual Cr (III) in the soil should oxidise to Cr (VI) at equilibrium. This risk assessment, therefore, assumes that five percent of the chromium in the soil would oxidise to Cr (VI) at equilibrium. Five percent conversion is a conservative assumption, given site conditions and the results of laboratory experiments (Table D-1).

REFERENCES

Adriano, D.C., 1986. Trace Elements in the Terrestrial Environment. Springer - Verlag, NY.

Bartlett, B.R. and B. James, 1979. Behavior of Chromium in Soils: III. Oxidation. J. Environ. Qual., 8:31-35.

Bloomfield, C. and G. Pruden, 1980. The Behavior of Cr(VI) in Soil under Aerobic and Anaerobic Conditions. Environmental Pollution (Series A), 23:103-114.

Eary, L.E. and D. Ral, 1987. Kinetics of Chromium (III) Oxidation to Chromium (VI) by Reaction with Manganese Dioxide. Environ. Sci. Technol., 21 (12): 1187-1193.

EPA, 1976. Application of Sewage Sludge to Cropland: Appraisal of Potential Hazards of the Heavy Metals to Plants and Animals. EPA-430/9-76-013.

EPA, 1977. Municipal Sludge Management: Environmental Factors. EPA-430/9-77-004.

Griffin, R.A., A.K. An, and R.R. Frost, 1977. Effect of pH on Adsorption of Chromium from Landfill - Leachate by Clay Minerals. J. Environ. Sci. Health, A12(8): 431-449.

Grove, J.H. and B.G. Ellis, 1980a. Extractable Chromium as Related to Soil pH and Applied Chromium. Soil Sci. Soc. Am. J., 44:238-242.

Grove, J.H. and B.G. Ellis, 1980b. Extractable Iron and Manganese as Related to Soil pH and Applied Chromium. Soil Sci. Soc. Am. J., 44:243-246.

James, B.R. and R.J. Bartlett, 1983. Behavior of Chromium in Soils: IV. Interactions Between Oxidation - Reduction and Organic Complexation. J. Environ. Qual., 12(2): 173-176.

Peters, D.G., J.M. Hayes, and G.M. Hieftje, 1974. Chemical Separations and Measurements: The Theory and Practice of Analytical Chemistry. W.B. Saunders Company, Philadephia, PA.

Peterson, M., District Conservation Officer, Soil Conservation Service, Gloucester County Field Office. Personal Communication, May 2, 1988.