Human Health Risk Assessment
Peerless Photo Products Site
Shoreham, New York
Site ID No.: 1-52-031

Prepared for

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LIST OF ACRONYMS AND ABBREVIATIONS

95UCLM	95th Percentile Upper Confidence Limit on the Mean
ADI	Average Daily Intake
APC	Area of Potential Concern
ASTM	American Society for Testing and Materials
AT	Averaging Time
CADI	Carcinogenic Average Daily Intake
COPC	Constituent of Potential Concern
ED	Exposure Duration
EPA	U.S. Environmental Protection Agency
EPC	Exposure Point Concentration
ft	Foot/Feet
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
HSA	Hollow Stem Auger
in.	Inch(es)
IRIS	Integrated Risk Information System
kg	Kilogram(s)
L	Liter(s)
LADI	Lifetime Average Daily Intake
mg	Milligram(s)
NCADI	Non-carcinogenic Average Daily Intake
NOAEL	No-Observed-Adverse-Effect-Level
NYS GQS	New York State Water Quality Standards for Class GA Groundwater
OSWER	Office of Solid Waste and Emergency Response
RAGS	Risk Assessment Guidance for Superfund
RfC	Reference Concentration
RfD	Reference Dose
RME	Reasonable Maximum Exposure

LIST OF ACRONYMS AND ABBREVIATIONS (continued)

RSCO	Recommended Soil Cleanup Objectives
SCGs	Standards, Criteria, and Guidances
SF	Slope Factor
SQL	Sample Quantitation Limit
TAGM	Technical and Administrative Guidance Memorandum
µg	Microgram(s)

1. INTRODUCTION

EA Engineering, Science, and Technology, Inc. was contracted by Agfa Corporation to provide risk assessment services for the Peerless Photo Products Site in Shoreham, New York. A human health risk assessment (HHRA) is presented in this document to address potential concerns with human use of the site.

The purpose of this HHRA is to determine whether, under hypothetical future exposure conditions and assuming no remediation at the site, chemicals detected in environmental media at the Peerless Photo Products Site are at concentrations that may cause unacceptable risk to humans using the area. This HHRA is also intended to further the work of a previous risk assessment (ENVIRON 1997) using more recent data. The site is currently industrial in nature, however, this risk assessment addresses all potential future uses of the site as a conservative measure, including residential and recreational.

The HHRA is conducted in accordance with U.S. Environmental Protection Agency (EPA) *Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual (Part A) (Interim Final)* (EPA 1989), EPA RAGS, *Volume 1 – Human Health Evaluation Manual (Part D), Standardized Planning, Reporting and Review of Superfund Risk Assessments Final* (EPA 2002) and, to an extent, follows methodologies agreed upon by regulators and parties interested in the site in the *Draft Risk Assessment-Phase I and 2 Remedial Investigation, Peerless Photo Products Site* (ENVIRON 1997). Where applicable, the latest EPA guidance is utilized instead of the ENVIRON 1997 document (e.g., exposure parameters). The original table formats have been retained in the document per request of regulators. RAGS D tables, as recommended by EPA, are included in the appendices as noted in the text.

The risk assessment methodology used in this HHRA involves a four-step process: hazard identification, exposure assessment, toxicity assessment, and risk characterization. A brief description of each step is provided below:

In the *hazard identification*, environmental monitoring data are evaluated, constituents of potential concern (COPCs) are selected for inclusion throughout the remainder of the risk assessment, and the rationale for their selection is documented.

In the *exposure assessment*, the human population, or groups of individuals potentially exposed to COPCs (i.e., potential human receptors) are characterized. From the many potential pathways of exposure, pathways applicable to potential receptors at the site are identified. The concentrations of COPCs in relevant media (e.g., soil, air) are converted into systemic doses, taking into account rates of contact (e.g., ingestion rates) and absorption rates of different COPCs. The magnitude, frequency, and duration of these exposures are then integrated to obtain estimates of daily doses over a specified period of time (e.g., lifetime, activity-specific duration).

In the *toxicity assessment*, the relationship between extent of exposure and extent of toxic injury or disease is estimated for each Constituent of Potential Concern (COPC). Chemical-specific toxicity values, such as cancer slope factors (SFs) and reference doses (RfDs) or reference concentrations (RfCs) for non-carcinogens are presented along with a discussion of their scientific basis and derivation. The toxicity assessment includes toxicological profiles for each COPC, which are provided in Appendix B.

Risk characterization integrates the results of the toxicity assessment and the exposure assessment to derive quantitative estimates of human health risk, including both the risks of cancer and of non-carcinogenic effects. The major uncertainties and limitations associated with the estimates of risk and their potential ramifications are presented in this section.

1.1 FACILITY HISTORY

The Peerless Photo Products site occupies 16 acres in a residential/commercial area with residences bordering the property on the north and east; to the south is Route 25A, and to the west is Randall Road. A Long Island Lighting Company[1] right-of-way runs along the northern border of the site. Figure 1 presents a site location map.

The site was first developed in 1903 as a residence and laboratory. In 1939, Peerless Photo Products, Inc. began manufacturing photographic paper at the site. Agfa purchased the facility in 1969 and continued to manufacture photographic paper. Manufacturing operations began to slow in 1984 and completely ceased in mid 1987. The primary operations throughout the site's industrial vitality were the production of photographic emulsions used in the manufacture of photographic film and the emulsion coating of photographic paper.

Currently, the site is completely encircled by a 6-ft high chain linked fence and is guarded 24-hours per day. The perimeter of the fence area is inspected daily for breaches.

There have been several environmental investigations and data gathering events at the site. The major investigations included: a Phase I Preliminary Investigation conducted by NYSDEC in 1983 (NYSDEC 1984); a Phase II Investigation conducted by agents of Agfa between 1986 and 1988 (ERM 1988); an underground storage tank removal program conducted by an agent of Agfa in 1990; and a Phase I and Phase 2 Remedial Investigation conducted by an agent of Agfa in 1994 and 1997 (ENVIRON 1997).

1.2 PEERLESS PHOTO PRODUCTS SITES

This HHRA focuses on the Peerless Photo Products Site, limited to the Area of Potential Concern (APC)-11 LILCO Right-of-Way and the APC-10 Tesla Tower Base. These two areas are described below. The two areas were combined for the purposes of the risk assessment and

[1] The Long Island Lighting Company (LILCO) historically owned the right-of-way (ROW). The ROW is currently owned by LILCO's successor, the Long Island Power Authority (LIPA), but is referred to as the LILCO ROW in this report for consistency with prior reports prepared for the site.

assessed together as onsite. The previous risk assessment addressed these areas as onsite but also included a limited assessment of offsite data (ENVIRON 1977). Based on the lack of additional offsite data, this HHRA focuses on the onsite samples and all potential receptors are evaluated for onsite exposure. Samples included in the risk assessment are listed in Table 1; data used in the risk assessment is provided in Appendix A.

1.2.1 APC-11 LILCO Right-of-Way

APC-11, the LILCO Right-of-Way, consists of an area to the northeast of the Peerless Photo Products facility in which silver has historically been detected at concentrations that exceed the surface soil Standards, Criteria, and Guidances (SCGs). The area is currently a right-of-way for electric utility transmission lines.

1.2.2 APC-10 Tesla Tower Base

APC-10, the Tesla Tower Base, consists of an octagonal concrete foundation, approximately 90 ft in diameter, which supported a radio tower during the early 1900s. The base was alleged to have contained an open shaft, approximately 120 ft deep, that was filled over time. The composition of the fill material was not documented, but current activities indicate that the material is a mixture of sandy soil and demolition debris such as concrete slabs and wood. Historic sampling indicated that cadmium, silver and mercury are present at concentrations that exceeded SCGs in three locations.

1.3 SAMPLING METHODOLOGIES

Twenty of the 22 soil borings installed during the 1994 Phase 1 Remedial Investigation were advanced using hollow stem auger (HSA) drilling equipment. Their associated soil samples were collected using 3-in. diameter split spoon samplers in accordance with the American Society for Testing and Materials (ASTM) Standard D 1586 for the Standard Penetration Resistance Test. Samples collected from the 2 remaining soil borings were obtained using a 4-in. inner diameter hand auger due to drill rig inaccessibility. The 26 surface soil samples were collected using a stainless steel trowel.

During the 1996 Phase 2 Remedial Investigation, 26 soil borings and 24 surface soil sample locations were investigated. Investigations conducted at APC-10 included a downhole geophysical survey in existing well MW-6, 7 test pits and 5 borings. The test pits were excavated to about 4 ft bgs using a backhoe. Two soil samples were collected from the base of 2 of the test pits. Five soil borings were advanced through the base of the pits using HSA drilling equipment. Samples were collected from various depths ranging from 4-22 ft bgs and submitted for laboratory analysis of Target Analyte List Metals.

During the 1996 Phase 2 Remedial Investigation, surface and subsurface samples were collected from 6 soil sample locations in APC-11. The 0-0.5 ft bgs interval was sampled with a stainless scoop. A stainless steel hand auger was used to collect the sample from the 3-4 ft bgs interval.

During the 2002 Supplemental Phase II Remedial Investigation, soil boring and sampling procedures were conducted using HSA drilling equipment and 2-in. diameter split spoon samplers in accordance with ASTM Method 1586. Sample locations are presented in Appendix E.

Prior to the 2002 sampling event, groundwater was sampled by the standard purge method A14. This method involved removing 3-5 well volumes from the wells at a fast purge rate and then reducing the purge rate to approximately 0.1-0.4 l/min. Field parameters were recorded at the end of each well volume and then at 5-minute intervals during the slow purge until stabilization. In the January 2002 sampling event, a comparison was done between two sampling techniques and groundwater was sampled using the standard purge method and the EPA's low stress purge method. Subsequent sampling events have followed the low stress purge method procedure. Water was purged from the wells using a 2-in. diameter stainless steel submersible pump and samples were collected using a disposable polyethylene bailer attached to a new monofilament line.

1.4 DATA QUALITY EVALUATION

Data for the HHRA were collected at the Peerless Photo Products site and are discussed in detail in the previous HHRA (ENVIRON 1997). Additional data was collected by EA. All data used in the HHRA is presented in Appendix A. The first step in the HHRA process is the evaluation of analytical data on the basis of qualifiers and the frequency of detection in each medium of concern. Inclusion or exclusion of data on the basis of analytical qualifiers was performed in accordance with EPA guidance (EPA 1989).

Analytical results bearing the U or UJ qualifier (indicating that the analyte is not detected at the given sample quantitation limit [SQL]) are retained in the data set and considered non-detects. Where warranted for statistical purposes, each COPC is assigned a numerical value of one-half its SQL.

Analytical results bearing the J qualifier (indicating that the reported value is estimated because the analyte is detected at a concentration below the SQL or for other reasons) are retained at the measured concentration.

Analytical results bearing the B or BJ qualifier were retained for further analysis. Analytical results for inorganic compounds were evaluated according to EPA guidance (EPA 1989) for inorganic COPCs bearing the B or BJ qualifiers (which indicate that the reported value is less than the contract-required detection limit, but greater than the instrument detection limit). Inorganic COPCs bearing the B or BJ qualifiers were retained in the data set at the measured concentration.

If a given analyte was not detected in any sample in any medium, the analyte was not considered further.

If duplicate samples were collected or duplicate analyses were conducted on a single sample, the following guidelines were employed to select the appropriate sample measurement:

- If both samples/analyses showed that the analyte was present, the average of the 2 detected concentrations was retained for analysis, based on conservative professional judgment.
- If only one sample/analysis indicated that the analyte was present, it was retained for analysis.
- If both samples/analyses were non detect, the lower SQL was retained for analysis, if appropriate.

2. HAZARD IDENTIFICATION

A hazard identification is conducted to determine which constituents are of potential concern at the site. In the hazard identification, a conceptual site model is developed and a screening analysis is conducted. The hazard identification serves to focus the HHRA on potential media, exposures and constituents of concern at the site.

A conceptual site model for the Peerless Photo Products Site is developed in the following sections and is depicted in Figure 3. The conceptual site model presents potential site media and human exposures to be considered in the HHRA. The same media and human exposures presented in the previous risk assessment are considered in this assessment. Hypothetical site uses include residential and recreational (park).

In the screening analysis, site-specific data are analyzed and compared to risk-based screening values to determine constituents of potential concern at the site.

2.1 MEDIA OF CONCERN

Site media of concern include onsite surface soil and groundwater. Surface soil is defined as 0 to 2.4 ft based on the available site data. The data assessed in this HHRA is a combination of the data collected during the Phase I and Phase II Remedial Investigations (RIs). The Phase I RI soil data was collected from 0 to 0.5 ft; the Phase II RI soil data was collected from 0 to 2.4 ft. To most accurately characterize the risk at the site, the data are combined into one database and assessed as surface soil in this assessment.

There are no surface water bodies on the site; therefore, sediment and surface water are not of concern. Groundwater at the site is not currently used as potable and depth to groundwater precludes contact via digging (much greater than 10 ft). As a conservative measure, groundwater is evaluated as a media of concern assuming that it is used as tap water in a hypothetical future scenario.

2.2 EXPOSURE PATHWAYS AND RECEPTORS OF CONCERN

An exposure pathway describes a mechanism by which a population or individual may be exposed to chemicals present at a site. A completed exposure pathway requires the following four components:

- A source and mechanism of chemical release to the environment
- An environmental transport medium for the released chemical
- A human receptor population
- A point of potential human contact with the contaminated medium
- A human exposure route at the point of exposure.

All four components must exist for an exposure pathway to be complete and for exposure to occur. Incomplete exposure pathways do not result in actual human exposure and are not included in the exposure assessment and resulting risk characterization. The following human exposure pathways were identified for evaluation for the Peerless Photo Products Site and are listed in Figure 3 (and in Table D-1).

As a conservative measure, a hypothetical residential scenario including both an adult and a child resident is considered. Another hypothetical future use included for the site is as a park. Park use would include recreational receptors including hypothetical park visitors, both an adult and a child, and youth trespassers. The park would require maintenance; therefore a park groundskeeper receptor is also addressed. The basis for the selection of these receptors follows the previous risk assessment (ENVIRON 1997).

2.3 EXPOSURES VIA SOIL

The following exposure pathways are considered to be complete for surface soil at the Peerless Photo Products Site:

- Incidental ingestion of surface soil during hypothetical residential activities
- Incidental ingestion of surface soil during hypothetical recreational activities
- Dermal contact with surface soil during hypothetical residential activities
- Dermal contact with surface soil during hypothetical recreational activities.

For several reasons, inhalation is considered a negligible pathway. Primarily, volatile compounds are not of concern at the site; therefore, inhalation of volatiles is not an exposure pathway. Also, inhalation of particulate (entrained from soil) is generally a negligible contributor to overall risk compared to the direct exposure pathways of ingestion and dermal contact. This is especially true for the chemicals of concern at this site. Further, inhalation was not considered a viable pathway in the original risk assessment (ENVIRON 1997).

Additionally, exposure to soil via ingestion of homegrown produce is not quantitatively evaluated in this assessment. While studies have been conducted to assess the potential for produce to uptake chemicals from soil, the percent of vegetables that are ingested from residential gardens and the ingestion rates of vegetables, there is a great uncertainty associated with estimating concentrations of chemicals in vegetables. Ingestion rates for home produced food items vary regionally and, in the United States, are lowest for populations in the Northeast (Exposure Factors Handbook, Section 13.3, EPA 1997a). Cadmium, one of the primary constituents of concern at this site, is taken up effectively by plants but varies greatly depending on the pH of the soil (ATSDR 1998). Cadmium content in garden soils is also likely to be driven by the type and amount of fertilizer utilized. Therefore, the ingestion of cadmium in garden vegetables by a hypothetical resident is not quantitatively evaluated in this assessment based on the uncertainty associated with quantifying the exposure concentrations; the low likelihood that a hypothetical resident will ingest large amounts of vegetables from a home garden at this site; and the relative impact of this pathway on total risks compared to ingestion and dermal contact with soils.

Exposure to soil at depth is also not quantitatively evaluated in this HHRA. The most likely human population to contact soils at depth would be a utility or construction worker. The groundskeeper scenario evaluated in this HHRA is adequately conservative to be protective for any construction worker or utility worker that may work on the site infrequently or for a shorter period of time than the groundskeeper (250 days per year for 25 years). Based on soil borings taken at the site, subsurface concentrations are consistent with surface concentrations (GT Engineering 1998). Therefore, risks associated with utility or construction workers are expected to be less (shorter exposure duration and frequency) than that of the groundskeeper scenario.

Exposure to offsite surface soil was not considered in this risk assessment. On 22 July 1996, NYSDEC and NYSDOH representatives collected soil samples from 3 residential properties on James Street, northeast of the Peerless Photo Products Site (GT Engineering 1998). The samples were analyzed for TAL metals, and the highest concentrations of each metal detected and considered a COC in the Phase 2 RI were as follows:

- Cadmium – 0.55 mg/kg
- Chromium – 9.2 mg/kg
- Lead – 26.4 mg/kg
- Mercury – 0.07 mg/kg
- Silver – 72.1 mg/kg.

NYSDOH concluded that none of the analytes were detected at concentrations above levels of regulatory or health-based concern, and reported the results of the sampling to the affected homeowners.

2.4 EXPOSURES VIA GROUNDWATER

The following exposure pathways are considered to be complete for groundwater at the Peerless Photo Products Site:

- Ingestion of groundwater during residential activities
- Ingestion of groundwater as tap water during maintenance activities
- Dermal contact with groundwater as residential tap water while showering/bathing.

For several reasons, inhalation is considered a negligible pathway. Primarily, volatile compounds are not of concern at the site; therefore, inhalation of volatiles is not an exposure pathway. Further, inhalation was not considered a viable pathway in the original risk assessment (ENVIRON 1997).

Additionally, use of groundwater for non-potable uses such as irrigation is not quantitatively evaluated. Groundwater at the site is located at much greater than 10 ft in depth; it is unlikely that residents or other site users will dig greater than 10 ft. Further, based on the availability of

public water, it is unlikely that wells will be sunk into groundwater at the site for irrigation purposes.

2.5 SELECTION OF CONSTITUENTS OF POTENTIAL CONCERN

The preliminary list of COPCs selected on the basis of risk-based screening (EPA 1989) was further evaluated, using additional considerations. The methodologies are the same as those presented in the previous risk assessment (ENVIRON 1997). The COPCs identified in the HHRA are listed in Table 2. The RAGS D risk screening tables are provided in Tables D-2.1 for surface soil and D-2.2 for groundwater.

An analyte is eliminated from further consideration as a COPC if it is not detected in onsite surface soil or ground water samples. Only inorganics were detected at concentrations of potential concern in soil or groundwater at the site in previous investigations (ENVIRON 1997). The most recent sampling at the site focused on inorganics as a result.

An analyte is eliminated from the list of COPCs if it is an essential nutrient of low toxicity, and its reported maximum concentration is unlikely to be associated with adverse health impacts. COPCs excluded from further consideration on this basis are calcium, iron, magnesium, potassium, and sodium.

Surface soils were compared to the New York State Technical and Administrative Guidance Memorandum (TAGM) Recommended Soil Cleanup Objectives (RSCO) (NYSDEC 1994, 2003). If the maximum detected concentration was below the TAGM RSCO, that analyte was eliminated from further consideration as a COPC. The analytes that exceeded the TAGM RSCO in onsite surface soil at the Peerless Photo Products Site are barium, beryllium, cadmium, copper, mercury and zinc (shown in Table D-2.1).

For groundwater, the New York State Water Quality Standards for Class GA Groundwater (NYS GQS) (NYSDEC 1998) are used for screening purposes. If the maximum detected concentration was below the NYS GQS, that analyte was eliminated from further consideration as a COPC. The analytes that exceeded the NYS GQS in groundwater at the Peerless Photo Products Site are antimony, cadmium, chromium, lead and manganese (as shown in Table D-2.2).

Table D-3.1 presents summary statistics and the 95th percentile upper confidence limit on the mean [95UCLM]) for the COPC in surface soil at the Peerless Photo Products site. The calculation of this statistic is discussed further in Section 3.1. The 95UCLM (presented as the medium exposure point concentration [EPC]) is utilized as the chemical-specific, medium-specific EPC in the exposure assessment for the reasonable maximum exposure (RME) assumptions. However, if the 95UCLM is greater than the maximum detected concentration, the maximum detected concentration value is used as the EPC and is listed in the table instead of the 95UCLM value, as per EPA guidance (EPA 1989). Similarly, a summary of COPCs in groundwater at the Peerless Photo Products site is presented in Table D-3.2.

2.6 COMPARISON TO BACKGROUND SCREENING

The selection of soil COPCs is also based on a comparison of inorganic surface soil data with background surface soil samples (EPA 1989). Soil sample SB-16 was designated as a background surface soil sample in the Phase 1 RI/FS Work Plan (FD GTI 1993). Following the methodology in the previous risk assessment (ENVIRON 1997), an analyte was eliminated from further consideration as a COPC in surface soil if the maximum background concentration plus 10 percent was found to be greater than the reasonable maximum exposure (RME) medium specific exposure point concentration (EPC).

Ground water data was compared to site-specific background well data (EPA 1989). In accordance with the Phase 1 RI/FS Work Plan (FD GTI 1993), the monitoring well MW-5 was designated as the background well. The methodology followed was also that of the ENVIRON risk assessment (ENVIRON 1997). If the arithmetic mean plus two standard deviations of the background concentration was found to be greater than the RME medium specific EPC value for that analyte in ground water, then that analyte was eliminated from further consideration as a COPC in groundwater.

2.7 IDENTIFICATION OF CONSTITUENTS OF POTENTIAL CONCERN

Based on the TAGM screening and background comparisons, the following were selected as COPCs: antimony, barium, cadmium, copper, mercury, silver and zinc in onsite surface soil; and, aluminum, cadmium, cobalt, manganese and vanadium in groundwater.

The COPCs for surface soil and groundwater for the Peerless Photo Products Site are shown in Table 2. Toxicological profiles for each of these chemicals are provided in Appendix B; Section 4 discusses the toxicological assessment.

3. EXPOSURE ASSESSMENT

An exposure assessment is conducted to estimate the magnitude of potential human exposures to COPCs in site media. In the exposure assessment, central tendency and reasonable maximum estimates of potential exposure are developed in accordance with EPA guidance. Conducting an exposure assessment involves analyzing releases of COPCs; identifying all potential pathways of exposure; estimating central tendency and reasonable maximum potential EPCs for specific pathways, based both on environmental monitoring data and predictive chemical modeling results; and estimating potential chronic daily intakes for specific pathways. The results of this assessment are pathway-specific estimates of potential intakes for exposures to individual COPCs.

3.1 QUANTIFICATION OF POTENTIAL EXPOSURES

The first step of the exposure assessment is to quantify potential exposure concentrations. This involves the evaluation of site data and the quantification of exposure concentrations for reasonable maximum exposure scenarios.

To assess human health risks, the statistical analyses of the COPC concentrations in each medium are performed. The methods used to analyze the data for each of these media are described below.

Reported concentrations are used to calculate the 95th percentile upper confidence limit on the mean (95UCLM) for COPCs in each medium (EPA 1992a). Exposure point concentrations (EPCs) in site media are estimated as the 95UCLM values for purposes of estimating reasonable maximum exposures. In cases where the 95UCLM values exceed the maximum detected concentration, the maximum detected concentration is used. The following steps are carried out to calculate 95UCLM. Because transformation is a necessary step in calculating the upper confidence limit of the mean (UCLM) for a log-normal distribution, the data are transformed by using the natural logarithm function (i.e., calculate ln(x), where x is the value from the data set). After transforming the data, 95UCLM for the data set is calculated by calculating the arithmetic mean of the transformed data; calculating standard deviation of the transformed data; determining H-statistic (Gilbert 1987); and calculating 95UCLM using the equation given below:

$$95\,UCLM = e^{(\bar{x} + 0.5\,s^2 - sH/\sqrt{n-1})} \qquad \text{(Equation 1)}$$

where:

95UCLM	=	95th percentile upper confidence limit on the mean
e	=	Constant (base of the natural logarithm; equal to 2.718)
x	=	Arithmetic Mean of the log-transformed data
s	=	Standard deviation of the log-transformed data
H	=	H-Statistic
n	=	Number of samples in the data set.

3.2 EXPOSURE POINT CONCENTRATIONS

Tables D-3.1 and D-3.2 present summary statistics (e.g., frequency of detection, range of detection, mean, and the 95UCLM) for each COPC per medium. These tables also present the selection of EPCs for use in the remainder of the risk assessment. The reasonable maximum exposure (RME) EPC value is utilized as the chemical-specific, medium-specific EPC in the exposure assessment for the RME assumptions. However, if the 95UCLM is greater than the maximum detected concentration, the maximum detected concentration value is used as the EPC and is listed in the table instead of the 95 UCLM value, as per EPA guidance (EPA 1989).

3.3 EXPOSURE EQUATIONS

The next step in this exposure assessment is to estimate COPC intakes for each of the pathways considered in the assessment. In this exposure assessment, we have provided 2 different measures of intake, depending on the nature of the effect being evaluated. When evaluating longer-term (i.e., subchronic and chronic) exposures to chemicals that produce adverse non-carcinogenic effects, intakes are averaged over the period of exposure (i.e., the averaging time [AT]) (EPA 1989). This measure of intake is referred to as the non-carcinogenic average daily intake (NCADI) and is a less than lifetime exposure. For chemicals that produce carcinogenic effects, intakes are averaged over an entire lifetime and are referred to as the lifetime carcinogenic average daily intake (CADI) (EPA 1989).

The generic equation to calculate intakes is given below:

$$(L)ADI = \frac{C \times IF \times EF \times ED \times RAF}{BW \times AT} \times CF \qquad \text{(Equation 2)}$$

where:

(L)ADI	=	(Lifetime) Average daily dose (mg/kg-day)
C	=	Concentration in a specific medium (mg/L or mg/kg)
IF	=	Intake factor[2] (mg/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
RAF	=	Relative absorption factor (unitless)
BW	=	Body weight (kg)
AT	=	Averaging time (days)
CF	=	Conversion Factor (10^{-6} kg/mg).

[2] The intake factor is the product of all intake variables that, when multiplied by the concentration of the chemical of potential concern in a specific medium, results in an estimate of the chemical intake in mg/kg-day for that population and exposure pathway. Intake factors may include ingestion rate, body surface area exposed to soil or water, dermal permeability constants, and soil adherence factors.

The exposure pathways considered to be complete are presented in Table 3. The specific equations used to estimate exposures for each of the exposure pathways assessed for the site are presented in Appendix C.

All of the COPCs at the site were assessed in the manner above with the exception of lead. Lead is evaluated separately per EPA protocol. Lead risks were evaluated using EPA's blood-lead level models for residents, as this is the most sensitive model. Lead risks are discussed in Section 5.4.

3.4 SELECTION OF EXPOSURE FACTOR VALUES

All exposure factor values used in estimating intakes are presented in Table 3 (and are described and referenced in Tables D-4.1 through D-4.9 per receptor and media). The following guidance documents are used in defining exposure factor values for estimating intakes for exposure pathways evaluated at the site:

- *Risk Assessment Guidance for Superfund (RAGS) Volume I Human Health Evaluation Manual Part A*, U.S. EPA December 1989 (EPA 1989).
- *Office of Solid Waste and Emergency Response (OSWER) Directive 9285.6-03; RAGS Volume 1-Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors,"* EPA, 1991 (EPA 1991).
- *Exposure Factors Handbook, Volume I, General Factors*, EPA, August 1997 (EPA 1997a).
- *Dermal Exposure Assessment: Principles and Applications*, EPA 1992 (EPA 1992b).
- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Dermal Risk Assessment, Interim Guidance.* EPA (EPA 2001).
- *Draft Risk Assessment-Phase 1 and 2 Remedial Investigation, Peerless Photo Products Site* (ENVIRON 1997).

For all exposure pathways that have exposure factor values specified in RAGS Part A and in OSWER Directive 9285.6-03, those values are used in this risk assessment. For exposure values not specified in RAGS Part A or the OSWER Directive, the Exposure Factors Handbook (EPA 1997a) and EPA dermal guidance documents are utilized (EPA 1992b, 2001).

3.4.1 Resident Adult

Exposure parameters for resident adult exposure are presented in Table 3 (Tables D-4.1 for surface soil and D-4.7 for groundwater) and in the adult residential risk calculation tables in Appendix C. Body weight for the adult resident is assumed to be 70 kg. Hypothetical future

adult residents are assumed to have an exposure duration of 30 years. An exposure frequency of 350 days/year for soil and groundwater is assumed (EPA 1997). Skin surface area available for contact with soil is assumed to be 5,700 cm^2, based on the lower legs, hands, forearms, and face with a soil adherence factor of 0.07 mg/cm^2 (U.S EPA 2001). Incidental ingestion of soil is assumed to be 100 mg/day (ENVIRON 1997). Ingestion of site groundwater is assumed to be 2 L/day (U. S. EPA 1991). The skin surface area available for dermal contact with groundwater is assumed to be 18,150 cm^2 (EPA 2001), with an event time of 0.2 hour/day (ENVIRON 1997).

3.4.2 Resident Child

Exposure parameters for hypothetical resident child exposure are presented in Table 3 (Tables D-4.2 for surface soil and D-4.8 for groundwater) and in the child residential risk calculation tables in Appendix C. Body weight for the child resident is assumed to be 15 kg. Hypothetical child residents are assumed to have an exposure duration of 6 years with an exposure frequency of 350 days/year for soil and groundwater (EPA 1997). Skin surface area available for contact with soil is assumed to be 2,800 cm^2, based on the lower legs, hands, forearms, and face with a soil adherence value of 0.2 mg/cm^2 (EPA 2001). Incidental ingestion of soil is assumed to be 200 mg/day (ENVIRON 1997). Ingestion of site groundwater is assumed at 2 L/day (ENVIRON 1997). The skin surface area available for contact with groundwater is assumed to be 7,280 cm^2, with an event time of 0.2 hour/day (ENVIRON 1997).

3.4.3 Park Visitor Adult

Exposure parameters for park visitor adult exposure are presented in Table 3 (Table D-4.3) and in the adult park visitor risk calculation tables in Appendix C. Body weight for the adult park visitor is assumed to be 70 kg. Hypothetical adult park visitors are assumed to have an exposure duration of 30 years. An exposure frequency of 78 days/year for soil is assumed based on 2 days per week (39 weeks) during the spring, summer and fall (ENVIRON 1997). Skin surface area available for contact with soil is assumed to be 5,700 cm^2, based on the lower legs, hands, and lower arms (EPA 2001). A soil adherence factor of 0.07 mg/cm^2 is assumed based on the 2001 EPA Draft Dermal Guidance (EPA 2001). Incidental ingestion of soil is assumed to be 100 mg/day (ENVIRON 1997).

3.4.4 Park Visitor Child

Exposure parameters for park visitor child exposure are presented in Table 3 (Table D-4.4) and in the child park visitor risk calculation tables in Appendix C. Body weight for the child park visitor is assumed to be 15 kg. Hypothetical child park visitors are assumed to have an exposure duration of 6 years with an exposure frequency of 78 days/year based on 2 days per week (39 weeks) during the spring, summer and fall (ENVIRON 1997). Skin surface area available for contact with soil is assumed to be 2,800 cm^2, based on the lower legs, hands, and lower arms (EPA 2001). A soil adherence value of 0.2 mg/cm^2 is assumed based on the 2001 EPA Draft Dermal Guidance (EPA 2001). Incidental ingestion of soil is assumed to be 200 mg/day.

3.4.5 Youth Trespasser

Exposure parameters for youth trespasser exposure are presented in Table 3 (Table D-4.5). Body weight for the youth trespasser is assumed to be 50 kg (ENVIRON 1997). Youth trespassers are assumed to have an exposure duration of 10 years with an exposure frequency of 117 days/year (ENVIRON 1997). A surface soil ingestion rate of 50 mg/day is assumed (ENVIRON 1997). Dermal exposure to soil is based on 4,690 cm^2 surface area, representing the lower arms, hands, and lower legs (ENVIRON 1997). A 0.07 mg/cm^2 adherence factor is assumed (EPA 2001).

3.4.6 Park Groundskeeper

Exposure parameters for groundskeeper exposure are presented in Table 3 (Tables D-4.6 for surface soil and D-4.9 for groundwater) for the park groundskeeper. Body weight for the groundskeeper is assumed to be 70 kg. Hypothetical groundskeepers are assumed to have an exposure duration of 25 years with an exposure frequency of 250 days/year (EPA 1997). A surface soil ingestion rate of 100 mg/day is assumed. Dermal exposure to soil is based on 5,700 cm^2 surface area, representing the lower arms, hands, and lower legs (EPA 2001). A 0.07 mg/cm^2 adherence factor is assumed (EPA 2001). Ingestion of groundwater is assumed at 1 L/day with an exposure frequency of 250 days/year (ENVIRON 1997).

4. TOXICITY ASSESSMENT

The toxicity assessment considers the types of potential adverse health affects associated with exposures to COPCs; the relationship between magnitude of exposure and potential adverse effects; and related uncertainties, such as the weight of evidence of a particular COPCs carcinogenicity in humans. The toxicity assessment for COPCs relies on existing toxicity information developed on specific organic compounds and inorganic constituents. EPA Guidance (EPA 1989) specifies that the assessment is accomplished in 2 steps: hazard identification and dose-response assessment.

Hazard identification is the process of determining whether studies claim that exposure to a COPC may cause the incidence of an adverse effect. EPA specifies that the dose-response assessment include: (1) EPA's quantitative evaluation of the existing toxicity information, and (2) EPA's characterization of the relationship between the dose of the COPC administered or received, and the incidence of potentially adverse health effects in the exposed population.

From this quantitative dose-response relationship, specific toxicity values are derived by EPA that can be used to estimate the incidence of potentially adverse effects occurring in humans at different exposure levels (EPA 1989). These EPA-derived toxicity values are called reference doses (RfDs) for non-carcinogens and slope factors (SFs) for potential carcinogens.

The toxicity values used for COPCs at Peerless Photo Products Site are presented in Table 4 (and in Table D-5 for non-carcinogens and in Table D-6 for carcinogens; related chemical-specific parameters are presented in Table D-7).

4.1 TOXICITY ASSESSMENT FOR NON-CARCINOGENS

For all COPCs, toxicity values for non-carcinogens are taken, when available, from the Integrated Risk Information Systems (IRIS) database (EPA 2003). IRIS chronic toxic potency concentrations are developed by EPA and undergo an extensive process of scientific peer review. Therefore, IRIS values are judged to be adequately verified.

If toxic potency concentrations for COPCs are not available from IRIS (EPA 2003), health effects assessment summary tables (HEAST) (EPA 1997b) are used as a secondary data source. As HEAST toxicity values are not scientifically peer-reviewed for quality or scientific acceptability, they may not be derived in strict accordance with EPA approved methodologies.

If IRIS or HEAST toxic potency concentrations are not available for one route of exposure but exist for another route, the existing value is examined for technical applicability to the alternate route and subsequently utilized, if appropriate.

The methodology used by EPA for deriving toxic potency concentrations for non-carcinogens, as well as site-specific considerations for modifying or using these concentrations, are discussed in detail in Barnes and Dourson (1988) and EPA guidance (EPA 1989). Non-carcinogens are

typically judged to have a threshold daily dose below which deleterious or harmful effects are unlikely to occur. This concentration is called the no-observed-adverse-effect-level (NOAEL) and may be derived from either animal laboratory experiments or human epidemiology investigations (usually workplace studies). In developing a toxicity value or human NOAEL for non-carcinogens (i.e., an RfD), the regulatory approach is first to (1) identify the critical toxic effect associated with chemical exposure (i.e., the most sensitive adverse effect); (2) identify the threshold dose in either an animal or human study; and (3) modify this dose to account for interspecies variability (where appropriate), differences in individual sensitivity (within-species variability), and other uncertainty and modifying factors. Uncertainty factors are intended to account for specific types of uncertainty inherent in extrapolation from the available data. Modifying factors account for the concentration of confidence in the scientific studies from which toxicity values are derived, according to such parameters as study quality and study reproducibility. The use of these factors is a conservative approach to protection of human health and is likely to overestimate the toxic potency associated with chemical exposure. The resulting RfD is expressed in units of milligrams of chemical per kilogram of body weight per day (mg/kg-bw/day).

Toxicity values used for exposures that involve dermal contact with chemicals typically require adjustment of the oral toxicity values (oral RfDs) to allow for the difference between the daily intake dose through dermal contact and ingestion. Most toxicity values are based on the actual administered dose, and must be corrected for the percent of chemical-specific absorption that occurs across the gastrointestinal tract prior to their use in dermal contact risk assessment (EPA 1989, 1992b, 2000). Recommended oral absorption efficiency factors are utilized in converting oral toxicity values to dermal toxicity values. These factors are shown in Table D-5.

4.2 TOXICITY ASSESSMENT FOR CARCINOGENICITY

Unlike non-carcinogens, carcinogens are generally assumed to have no threshold; that is, there is presumed to be no level of exposure below which carcinogenic effects will not manifest themselves. This "non-threshold" concept supports the idea that there are small, finite probabilities of inducing a carcinogenic response associated with every level of exposure to a potential carcinogen. EPA uses a two-part evaluation for carcinogenic effects, which includes the assignment of a weight-of-evidence classification to a chemical based on a thorough scientific examination of the body of available data, and the quantification of a cancer toxic potency concentration, i.e., the slope factor, which reflects the dose-response data for the carcinogenic endpoint(s) (EPA 1989).

The weight-of-evidence classification system assigns a letter or alphanumeric (A through E) to each potential carcinogen that reflects an assessment of its potential to be a human carcinogen.[3] Only compounds that have a weight-of-evidence classification of C or above are considered to have carcinogenic potential in this risk assessment.

[3]A = a known human carcinogen; B1 = a probable human carcinogen, based on sufficient animal data and limited human data; B2 = a probable human carcinogen based on sufficient animal data and inadequate or no human data; C = a possible human carcinogen; D = not classifiable as to human carcinogenicity; and E = evidence of non-carcinogenicity for humans.

Although currently a controversial approach, chemicals that are classified as human or rodent carcinogens are typically assumed to have no threshold, in that there is presumed to be no concentration of exposure below which carcinogenic effects will not be manifested. The EPA slope factor (SF) is the upper 95th percentile confidence limit of the probability of response per unit daily intake of a chemical over a lifetime. Typically, the slope factor is used to estimate the upper-bound lifetime probability of a person developing cancer from exposure to a given concentration of a carcinogen. Slope factors are generally based on experimental animal data, unless suitable epidemiological studies are available. Due to the difficulty in detecting and measuring carcinogenic endpoints at low exposure concentrations, slope factors are typically developed by using a model to fit the available high-dose, experimental animal data, and then extrapolating downward to the low-dose range to which humans are typically exposed. EPA usually employs the linear multistage model to derive a slope factor. The model is conservative, and provides an upper bound estimate of excess lifetime cancer risk. Thus, the actual risk may be lower and could be zero (EPA 1989). These methods and approaches are discussed in greater detail in the EPA *Risk Assessment Guidance for Superfund* (EPA 1989).

Carcinogenic slope factors used for exposures that involve dermal contact typically require adjustment of the oral slope factor to allow for the difference between the dermal dose and the ingested dose. Most toxicity values are based upon the actual administered dose and must be corrected for the percent of chemical-specific absorption that occurs across the gastrointestinal tract prior to their use in dermal contact risk assessments (EPA 1989).

4.3 TOXICITY OF CONSTITUENTS OF POTENTIAL CONCERN

Toxicity data available in the scientific literature are used to prepare a toxicity profile for each COPC. Each profile describes the potential for carcinogenicity and other health effects of each COPC, summarizes available data, presents a weight-of-evidence approach for identifying the hazards associated with chemical exposure to the COPC, and provides a scientific profile for selecting the most appropriate toxicity values (i.e., quantitative estimates of the strength of the dose-response) used later in the risk assessment.

A review of relevant toxicity data for each COPC is presented in Appendix B, along with a description of critical studies (i.e., studies from which the quantitative toxic potency values are derived). Toxicity values are obtained from the EPA IRIS (EPA 2003), a peer-reviewed toxicity database. If toxicity values are not available from IRIS, values from the EPA HEAST (EPA 1997b) or from the National Center for Environmental Assessment (NCEA) are used.

4.3.1 Summary of Toxicity Values for Non-Carcinogenic Effects

EPA-derived toxicity values for evaluating potential chronic non-carcinogenic effects for COPCs are summarized in Table 4 (and Table D-5 in Appendix D). Toxicity information presented in these tables includes the following EPA provided/derived information: chronic or subchronic RfD values for exposures via the oral pathway; reported health effects, uncertainty and modifying factors specific to the EPA-derived RfD; and the scientific source of the information.

4.3.2 Summary of Toxicity Values for Potential Carcinogenic Effects

EPA-derived toxicity values for evaluating potential carcinogenic effects for COPCs are summarized in Table 4 (and Table D-6 in Appendix D). Toxicity information presented in these tables includes the following EPA provided/derived information: a chemical-specific SF (cancer potency factor) for exposures via the oral pathway; EPA's weight-of-evidence cancer classification; and the scientific source of the information.

5. RISK CHARACTERIZATION

Risk characterization is the final step of the HHRA process. In this step, the toxicity values are combined with the estimated chemical intakes for the receptor populations to quantitatively estimate both carcinogenic and non-carcinogenic risks. Risks are estimated for the following receptor populations: residents (adult and child), construction workers, and commercial workers.

The methodologies used to estimate cancer risks and chronic and subchronic risks for non-carcinogens are described further in the sections below.

5.1 HAZARD QUOTIENT AND HAZARD INDEX FOR NON-CARCINOGENIC EFFECTS

The potential human health risks associated with exposures to non-carcinogenic COPCs at the Peerless Photo Products Site onsite and in the vicinity of the site are estimated by comparing the NCADI with the RfD, as per EPA Guidance (EPA 1989). A hazard quotient (HQ) is derived for each COPC, as shown in the equation below:

$$HQ = \frac{NCADI}{RfD} \qquad \text{(Equation 3)}$$

where:

HQ	=	Hazard Quotient; ratio of average daily intake level to acceptable daily intake level (unitless)
NCADI	=	Estimated non-carcinogenic average daily dose (mg/kg-day)
RfD	=	Reference dose (mg/kg-day).

If the average daily dose exceeds the RfD, then the HQ will exceed a ratio of 1.0 and there may be concern that potential adverse systemic health effects will be observed in the exposed populations. If the NCADI does not exceed the RfD, the HQ will not exceed 1.0 and there will be no concern that potential adverse systemic health effects will be observed in the exposed populations. However, if the sum of several HQs exceeds 1.0, and the COPCs affect the same target organ, there may be concern that potential adverse systemic health effects will be observed in the exposed populations. In general, the greater the value of the HQ above 1.0, the greater the level of concern. However, the HQ does not represent a statistical probability that an adverse health effect will occur.

For consideration of exposures to more than one chemical causing systemic toxicity via several different pathways, the individual HQs are summed to provide a total hazard index (HI) for each receptor across all pathways and all media. If the total HI is less than 1.0, then no adverse health effects are likely to be associated with exposures at the site for that receptor.

However, if the total HI is greater than 1.0, it is necessary to calculate separate endpoint-specific HIs based on toxic endpoint of concern or target organ (e.g., HQs for neurotoxins are summed separately from HQs for renal toxins) to determine the potential for adverse health effects. COPCs with common target organs are summed to consider potential additive effects for each target organ. Only if an endpoint-specific HI is greater than 1.0 is there reason for concern about potential health effects for that target organ. If all endpoint-specific HIs are lower than 1.0, there are no concerns for adverse health effects for that receptor.

5.2 CANCER RISKS

Carcinogenic risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen at the site. The numerical estimate of excess lifetime cancer risk is calculated by multiplying the CADI by the risk per unit dose (the slope factor), as shown in the following equation:

$$Risk = CADI \times SF \qquad \text{(Equation 4)}$$

where:

Risk	=	The unitless probability of an exposed individual developing cancer
CADI	=	Lifetime cancer average daily dose (mg/kg-day)
SF	=	Cancer slope factor $(mg/kg\text{-}day)^{-1}$.

Because the slope factor is the statistical 95th percent upper-bound confidence limit on the dose-response slope, this method provides a conservative, upper-bound estimate of risk.

It should be noted that the interpretation of the significance of the cancer risk estimate is based on the appropriate public policy. EPA in the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) [40 Code of Federal Regulations (CFR) Part 300] (EPA 1990) states that:

> *"...For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6}"*

5.3 RISK CALCULATION RESULTS

Chemical-specific, pathway-specific risk estimates are presented by receptor group in Appendix C and are discussed below. Risk calculation results are also summarized in Appendix D for each receptor.

For all COPCs, estimates of cumulative risks across pathways and media for non-carcinogenic and carcinogenic effects for each receptor population are presented in Tables D-9.1 through D-9.5 in Appendix D. These are discussed below.

Receptor-specific results are presented separately for soil and for groundwater (Appendix C). Also, as a conservative measure, results for groundwater are added to each of the soil results for a total receptor (scenario) result (Appendix D). Pathway-specific risk characterization equations and calculations are also presented in these tables. A summary of the risk results is presented in Table 5 for non-carcinogens and in Table 6 for carcinogens.

For non-carcinogenic risks, the exposure to more than one chemical causing systemic toxicity via several different pathways is considered. The COPC-specific HQs are summed to calculate a total HI for each receptor across all pathways and all media. If the total HI is less than 1.0, then no adverse health effects are likely to be associated with exposures at the site for that receptor.

However, if the total HI is greater than 1.0, it is necessary to calculate endpoint-specific HIs based on toxic endpoint of concern or target organ (e.g., HQs for neurotoxins are summed separately from HQs for renal toxins) to determine the potential for adverse health effects. COPCs with common endpoints are summed to consider potential additive effects for each target organ. Only if an endpoint-specific HI is greater than 1.0 is there reason for concern about potential health effects for that target organ. If all endpoint-specific HIs are lower than 1.0, there are no concerns for adverse health effects for that receptor.

Tables D-10.1 through D-10.5 show the calculation of endpoint-specific HIs. COPCs with HQs greater than 0.1 are considered potential risk drivers and are retained in the table to determine endpoint-specific HIs. Only if an endpoint-specific HI is greater than 1.0 is there reason for concern about potential health effects for that endpoint or target organ.

5.3.1 Residential Scenario Results

Residential results are presented for two media: onsite surface soil and groundwater (combined in Table D-9.1). The media specific results are presented below followed by the cumulative scenario-specific results. Under the residential scenario, adult and child residents are both evaluated. Non-carcinogenic risks for adult and child receptors are presented separately. In calculating cancer risks, the adult and child results are summed to account for lifetime exposure.

The non-carcinogenic HI for soil and groundwater combined for the hypothetical adult resident is 13. The non-carcinogenic HI for soil and groundwater combined for the hypothetical child resident is 76. These both exceed the target HI of 1.0. An analysis by target organ (Table D 10.1) indicates that the central nervous system, skin and kidneys for both the adult and the child receptors exceed the target benchmark. These are discussed further below by medium.

Cancer risks for the residential scenario are zero because there are no carcinogenic COPCs.

Residential Onsite Surface Soil Results

Residential results for onsite surface soil are presented in Table D-9.1. A summary of the potential risk drivers (those non-carcinogens greater than 0.1and carcinogens greater than 10^{-6}) is provided in Table D-10.1.

For the adult resident surface soil pathways, the total HI is 3.5 (Table D-9.1). Table 10.1, which presents a breakdown by target organ, reveals cadmium and silver as the risk drivers. The target organs exceeding the benchmark of 1.0 are the central nervous system and skin. These results are driven entirely by the ingestion of surface soil pathway. Dermal risks are negligible.

The onsite residential child surface soil HI is 33 (Table D-9.1). A breakdown by target organ is provided in Table 10.1, presenting the risk drivers cadmium and silver. The target organs exceeding the benchmark of 1.0 are kidneys, central nervous system and skin. These results are driven entirely by the ingestion of surface soil pathway. Dermal risks are negligible.

Carcinogenic risks for the residential scenario are combined for the adult and child resident receptors to assess potential lifetime risks. Cancer risks for surface soil (Tables D-9.1) are zero because there are no carcinogenic COPCs.

Residential Groundwater Results
Residential results for groundwater are presented in Table D-9.1. A summary of the risk drivers is provided in Table D-10.1.

The residential adult HI for groundwater is 9.7. The residential child cumulative HI for groundwater is 44. The analysis by target organ (Table D-10.1) indicates that cadmium and manganese are the risk drivers. Kidneys, central nervous system, and skin are the target organs of concern.

Cancer risks for the residential groundwater scenario are zero because there are no carcinogenic COPCs.

5.3.2 Park Visitor Scenario Results

Under the park visitor scenario, adult and child park visitor were both evaluated. The park visitor receptors were evaluated for exposure to onsite surface soil. The scenarios are presented for the adult in Table D-9.2 and for the child in D-9.3.

Adult Park Visitor Results
The adult park visitor results for onsite surface soil are presented in Table D-9.2. A summary of the adult surface soil risk drivers is provided in Table D-10.2.

For the adult park visitor surface soil pathways, the HI is 0.78 (Table D-9.2). There are no non-carcinogenic risk concerns for this receptor.

Child Park Visitor Results
The child park visitor results for onsite surface soil are presented in Table D-9.3. The onsite residential child surface soil HI is 7.3 (Table D-9.3). A breakdown by target organ is provided in table D-10.3. Silver is the primary risk driver. The central nervous system and skin are the target organs of concern.

Park Visitor Carcinogenic Results
Adult cancer risks (Table D-9.2) are zero as are the child cancer risks (Table D-9.3). There are no carcinogenic COPCs at the site.

5.3.3 Trespasser Scenario Results

The trespasser receptor is evaluated for onsite surface soil ingestion and dermal pathways. The results are presented D-9.4. A breakdown by target organ is provided in Table D-10.4.
For the youth trespasser onsite surface soil pathways, the total HI is 0.83 (Table D-9.4). There are no non-carcinogenic risk concerns for this receptor.

Youth trespasser cancer risks (Table D-9.4) are zero. There are no carcinogenic COPCs at the site.

5.3.4 Park Groundskeeper Scenario Results

The park groundskeeper receptor is evaluated for onsite surface soil ingestion and dermal pathways. The park groundskeeper is also evaluated for ingestion of groundwater. The results are presented D-9.5. A breakdown by target organ is provided in Table D-10.5.

The non-carcinogenic HI for soil and groundwater combined for the hypothetical park groundskeeper is 5.8. This exceeds the target HI of 1.0. An analysis by target organ (Table D-10.5) indicates that the central nervous system, skin and kidneys exceed the target benchmark. These are discussed further below by medium.

Cancer risks for the residential scenario are zero because there are no carcinogenic COPCs.

Surface Soil Park Groundskeeper Results
The park groundskeeper cumulative HI for onsite surface soil is 2.5. This is driven entirely by the ingestion of surface soil pathway. Target organs exceeding the benchmark of 1.0 are the central nervous system and skin. The risk driver is silver.

Cancer risks for park groundskeeper surface soil exposure are zero. There are no carcinogenic COPCs at the site.

Groundwater Park Groundskeeper Results
The park groundskeeper cumulative HI for groundwater is 3.3. Target organs exceeding the benchmark of 1.0 are kidneys. The risk driver is cadmium.

Cancer risks for park groundskeeper groundwater exposure are zero. There are no carcinogenic COPCs at the site.

6. UNCERTAINTY ASSESSMENT

There are numerous uncertainties involved in the human health risk assessment process. These are discussed briefly in the following sections.

6.1 SAMPLING AND ANALYSIS UNCERTAINTIES

The sampling plan can have a significant impact on the results obtained in calculating human health risks at a site. To the extent that samples are taken in areas that are expected to be contaminated (biased sampling), the exposure point concentration used in calculating risk exposures and risks is likely to overestimate the actual concentration encountered at the site from random exposure across the site. This sampling bias will generally result in an overestimate of exposures and risks at a site. The soil sampling at the study areas incorporated a combination of random and biased samples. As the majority of soil samples collected are biased toward potentially contaminated areas, the measured concentrations and calculated health risks would tend to be overestimated.

Analysis uncertainty may be introduced due to laboratory contamination or error. Common laboratory contaminants include phthalates and chloroform. Chloroform was included as COPCs in groundwater based on detection above tap water screening values. Although it is possible that this chemical was detected due to laboratory contamination, this chemical was included as a conservative measure. Chemicals that are common laboratory contaminants may not be site-related and their inclusion as COPCs tends to bias risk calculations high.

6.2 UNCERTAINTIES ANALYSIS OF EXPOSURE ASSESSMENT

An analysis of uncertainties is an important aspect of the exposure assessment. It provides the risk assessor and reviewer with information relevant to the individual uncertainties associated with exposure factor assumptions and their potential impact on the final assessment.

6.2.1 Exposure Point Concentrations

A significant uncertainty exists with the basic approach used in arriving at EPCs for the COPCs in surface and total soils.

Uncertainty results from the use of one-half detection limit for all non-detects. An objective of the guidance is to include some quantitative value for COPCs when analytical data indicate that those COPCs are not detected, so that an estimated potential intake and resultant potential risk can be calculated. This approach is referenced in *Risk Assessment Guidance for Superfund (Volume 1), Human Health Evaluation Manual* (EPA 1989). However, this approach generally overestimates the average value, and results in overestimates of intakes and subsequent risks, particularly for COPCs with low frequencies of detection.

Inorganic constituents occur naturally in the environment. It is likely that detected concentrations of some inorganic constituents are not associated with site use and are at naturally occurring levels. Therefore, it is probable that some of the calculated risks are contributed by naturally occurring background concentrations of inorganics in site soil and are not the result of activities at the site.

6.2.2 Soil Ingestion Rate

Soil ingestion rates for construction workers are based on studies performed by Hawley (1985). These are short-term studies, and as they are not based on average long-term exposures, they represent an overestimate of exposure.

6.2.3 Surface Soil as a Medium of Concern

Surface soil is conservatively assessed as an exposure media of concern for all receptors. Thus, this risk assessment assumes that the current surface soil will be present under future conditions. Under future development plans, should the surface soil with elevated concentrations be removed and covered with clean fill, covered with asphalt or buildings, or otherwise made inaccessible to future site users, this media and its associated elevated human health risks for hypothetical future residential adults and children, park visitor children, and park groundskeepers would be eliminated.

6.3 UNCERTAINTIES OF TOXICITY ASSESSMENT

There are numerous uncertainties associated with the toxicity assessment. These are generally due to the unavailability of data to thoroughly calculate the toxicity of COPCs. These are described in more detail in the following sections.

6.3.1 Uncertainties Associated With Non-Carcinogenic Effects

Interspecies Extrapolation – The majority of toxicological information comes from experiments with laboratory animals. Experimental animal data have been relied on by regulatory agencies to assess the hazards of human chemical exposures. Interspecies differences in chemical absorption, metabolism, excretion, and toxic response are not well understood; therefore, conservative assumptions are applied to animal data when extrapolating to humans. These probably result in an overestimation of toxicity.

Intraspecies Extrapolation – Differences in individual human susceptibilities to the effects of chemical exposures may be caused by such variables as genetic factors (e.g., glucose-6-phosphate dehydrogenase deficiency), lifestyle (e.g., cigarette smoking and alcohol consumption), age, hormonal status (e.g., pregnancy), and disease. To take into account the diversity of human populations and their differing susceptibilities to chemically induced injury or disease, a safety factor is used. EPA uses a factor between 1 and 10. This uncertainty may lead to overestimates of human health effects at given doses.

Exposure Routes – When experimental data available on one route of administration are different from the actual route of exposure that is of interest, route-to-route extrapolation must be performed before the risk can be assessed. Several criteria must be satisfied before route-to-route extrapolation can be undertaken. The most critical assumption is that a chemical injures the same organ(s) regardless of route, even though the injury can vary in degree. Another assumption is that the behavior of a substance in the body is similar by all routes of contact. This may not be the case when, for example, materials absorbed via the gastrointestinal tract pass through the liver prior to reaching the systemic circulation, whereas by inhalation the same chemical will reach other organs before the liver. However, when data are limited these extrapolations are made, and may result in overestimates of human toxicity.

6.3.2 Uncertainties Associated With Carcinogenic Effects

Interspecies Extrapolation – The majority of toxicological information for carcinogenic assessments comes from experiments with laboratory animals. There is uncertainty about whether animal carcinogens are also carcinogenic in humans. While many chemical substances are carcinogenic in one or more animal species, only a very small number of chemical substances are known to be human carcinogens. The fact that some chemicals are carcinogenic in some animal species but not in others raises the possibility that not all animal carcinogens are human carcinogens. Regulatory agencies assume that humans are as sensitive to carcinogens as the most sensitive animal species. This policy decision, designed to prevent underestimation of risk, introduces the potential to overestimate carcinogenic risk.

High-Dose to Low-Dose Extrapolation – Typical cancer bioassays provide limited low-dose data on responses in experimental animals for chemicals being assessed for carcinogenic or chronic effects. The usual dose regime involves 3 dose groups per assay. The first dose group is given the highest dose that can be tolerated, the second is exposed to one-half that dose, and the third group is unexposed (control group) [National Research Council (NRC) 1983]. Because this dosing method does not reflect how animals would react to much lower doses of a chemical, a dose-response assessment normally requires extrapolation from high to low doses using mathematical modeling that incorporates to varying degrees information about physiologic processes in the body (NRC 1983).

A central problem with the low-dose extrapolation models is that they all too often fit the data from animal bioassays equally well, and it is not possible to determine their validity based on goodness of fit. Several models may fit experimental data equally well, but they may not all be equally plausible biologically. The dose-response curves derived from different models diverge substantially in the dose range of interest (NRC 1983). Therefore, low-dose extrapolation is more than a curve-fitting process and considerations of biological plausibility of the models must be taken into account before choosing the best model for a particular set of data.

6.4 UNCERTAINTIES IN RISK CHARACTERIZATION

Uncertainties in the risk characterization can stem from the inherent uncertainties in the data evaluation, the exposure assessment process, including any modeling of exposure point

concentrations in secondary media from primary media, and the toxicity assessment process. The individual uncertainties in these respective processes are addressed in previous sections of the HHRA.

Uncertainty may also stem from the inclusion or exclusion of chemicals that are detected at low frequencies. Chemicals that are infrequently detected may be artifacts in the data due to sampling, analytical, or other problems. Therefore, these chemicals may not be related to site operations or disposal practices. When considering the site data in the context of detection frequency, it is evident that several COPCs in groundwater may not be site related. Inclusion of these chemicals in the quantitative risk estimates is likely to bias the results high because these chemicals may not truly be site-related. Therefore these chemicals were excluded from further analysis.

6.5 UNCERTAINTIES IN DERMAL RISK CALCULATION

Dermal risks are calculated utilizing a dermal absorption fraction from soil. These factors are shown in Table D-5. Cadmium is the only site-related metal that has a dermal absorption factor identified in the EPA guidance (EPA 2001). As a result, risk from dermal absorption of other site-related metals was not evaluated per the latest EPA guidance (EPA 2001).

7. SUMMARY AND CONCLUSIONS

The risk assessment is conducted to assess potential non-carcinogenic effects and cancer risks from hypothetical residential and recreational (park visitor, trespasser, groundskeeper) scenarios for environmental media of concern at the Peerless Photo Products Site under current site conditions (i.e. no remediation occurs). Onsite surface soil and groundwater were assessed for potential human exposure as depicted in Figure 3 and Table 1. Results are summarized in Table 5 for non-carcinogens and in Table 6 for carcinogens.

7.1 POTENTIAL RISKS

7.1.1 Non-Carcinogenic Risks

Non-carcinogenic risks exceed the threshold of 1.0 for hypothetical future residential adults and children, park visitor children, and park groundskeepers; non-carcinogenic risks do not exceed 1.0 for the youth trespasser and the park visitor adult.

Onsite groundwater use as tap water is a primary risk driver for the resident adult and child and for the park groundskeeper. Groundwater is not currently used as a source of drinking water, and is not expected to be used as a potable source. Based on these risks, it is not recommended that site groundwater be used as a potable source.

Cumulative HIs are calculated as greater than the 1.0 threshold for onsite surface soil for the adult resident, child resident, child park visitor and park groundskeeper. The site is currently not used for any of these scenarios. Cadmium and silver in surface soil are of potential concern for these receptors.

7.1.2 Carcinogenic Risks

None of the chemicals of potential concern at the site are carcinogenic in nature for the pathways considered. Cadmium is considered a probable human carcinogen via inhalation (weight of evidence B1). However, the inhalation of soil or fugitive dust pathway was not considered in this risk assessment. The maximum concentration of cadmium detected in soil at Areas of Concern 10 and 11 is lower than the EPA soil screening level of 1,800 part per million. The soil screening level was developed to be protective of cancer risks from fugitive dusts. Therefore, there are no carcinogenic risks at the Peerless Photo Products Site.

7.2 CONCLUSIONS

Potential risks were determined for several of the hypothetical use scenarios evaluated in this HHRA. Ingestion of onsite surface soil was of potential concern for cadmium and silver. Use of onsite groundwater as tap water was of potential concern for cadmium and manganese.

The youth trespasser is the only evaluated receptor that may currently contact soil at the site. The risks for this receptor were negligible. Therefore there are no current risk concerns at the site.

It is important to note that under current use conditions there are no exposures to groundwater for any potential human receptor. Public water supply is used at the site. Therefore there are currently no human health risks associated with groundwater. It is also expected that future use of the site groundwater will remain unchanged, with no use of groundwater. Should site groundwater be used as potable, potential health risks are expected.

Furthermore, the potential risks associated with offsite receptor exposure to groundwater were not considered in this risk assessment. The potential for offsite receptors to contact groundwater migrating offsite is discussed in the *Final Groundwater Modeling Summary Report* (EA 2003). Although there are no public supply wells operating in the vicinity of the site, groundwater modeling was conducted using a hypothetical supply well. A series of predictive scenarios were modeled. Maximum modeled concentrations at the hypothetical supply well were significantly below the Federal Maximum Contaminant Level of 5 μg/L for cadmium, reaching a maximum concentration of 0.8 μg/L. The model predicted concentrations are likely to be overestimated and it is possible that no detectable cadmium would actually reach the hypothetical supply well.

The evaluation of risk under future conditions indicated potential risk from surface soil under hypothetical future exposure scenarios. However, with future development, should the surface soil with elevated concentrations be removed, covered with clean fill, covered with asphalt or buildings, or otherwise made inaccessible to future site users, this media and its associated potential human health risks would be eliminated.

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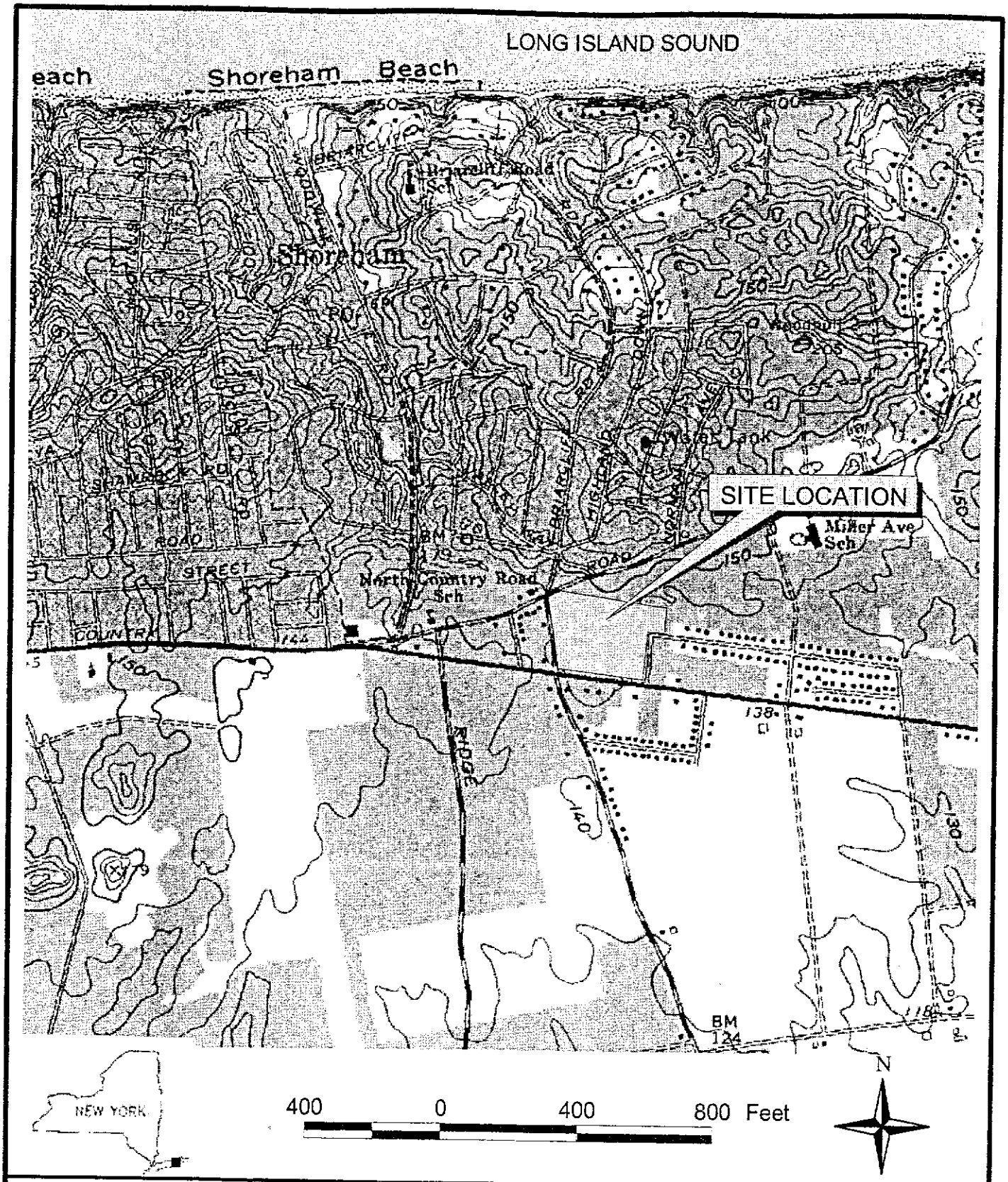
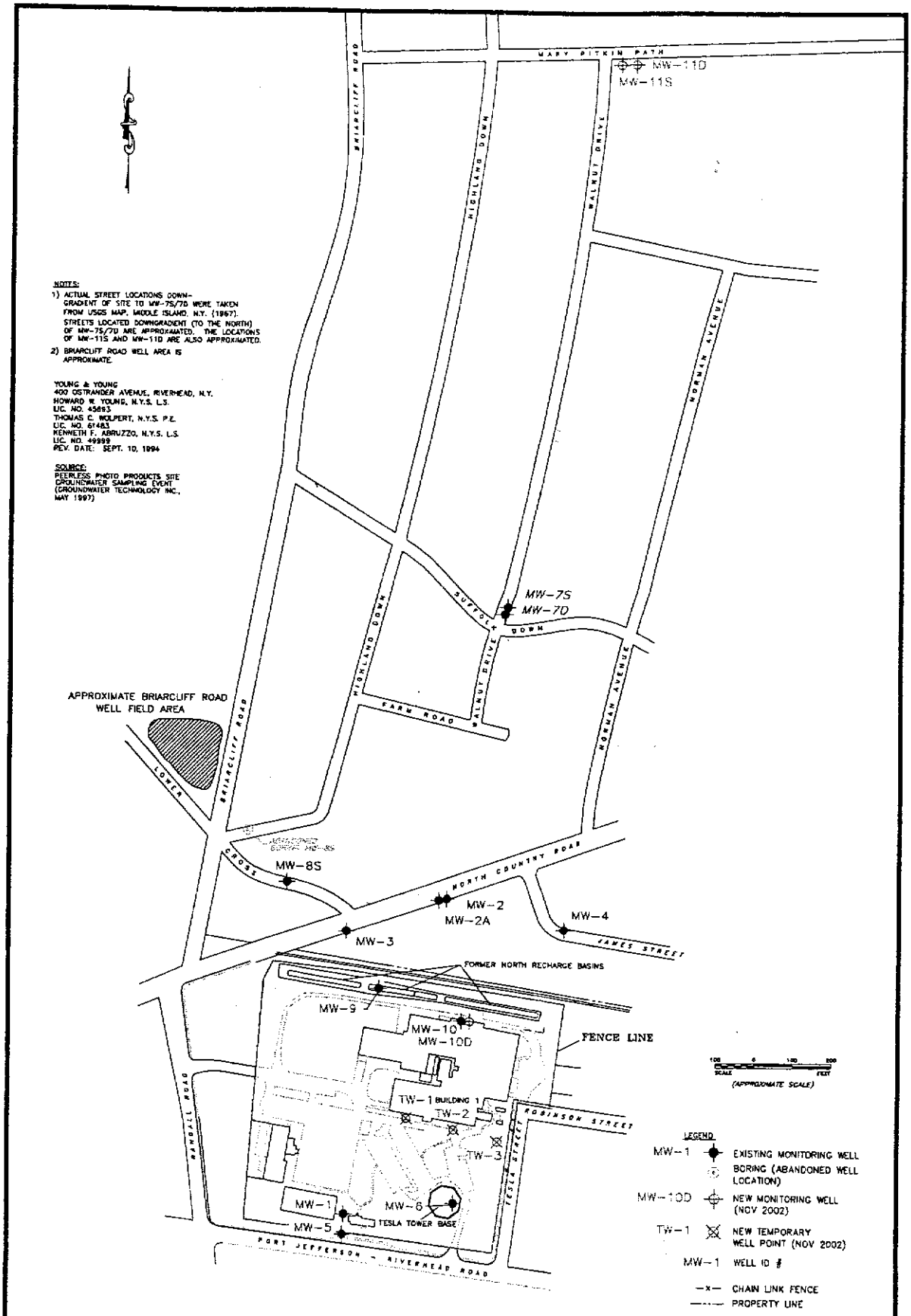
Figures



EA ENGINEERING, SCIENCE, AND TECHNOLOGY	PEERLESS PHOTO SITE SHOREHAM, NEW YORK				FIGURE 1-1 SITE LOCATION MAP		
PROJECT MGR: PN	DESIGNED BY: DC	CREATED BY: DC	CHECKED BY: PN	SCALE: AS SHOWN	DATE: 20 NOV 2002	PROJECT NO: 1371211	FILE NO: I:\AGFA\ AGFA.APR



NOTES:
1) ACTUAL STREET LOCATIONS DOWN-GRADIENT OF SITE TO MW-7S/7D WERE TAKEN FROM USGS MAP, MIDDLE ISLAND, N.Y. (1967). STREETS LOCATED DOWNGRADIENT (TO THE NORTH) OF MW-7S/7D ARE APPROXIMATED. THE LOCATIONS OF MW-11S AND MW-11D ARE ALSO APPROXIMATED.
2) BRIARCLIFF ROAD WELL AREA IS APPROXIMATE.

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LIC. NO. 49999
REV. DATE: SEPT. 10, 1994

SOURCE:
PEERLESS PHOTO PRODUCTS SITE GROUNDWATER SAMPLING EVENT (GROUNDWATER TECHNOLOGY INC., MAY 1997)

EA ENGINEERING, SCIENCE, AND TECHNOLOGY	PEERLESS PHOTO SITE SHOREHAM, NEW YORK	SITE LAYOUT	DATE 24 FEB 2003	DRAWN BY TB	PROJECT NO. 13712.11
			SCALE AS SHOWN	PROJECT MGR. CK	FILE NAME SITE_LAYOUT.DWG
					FIGURE 2

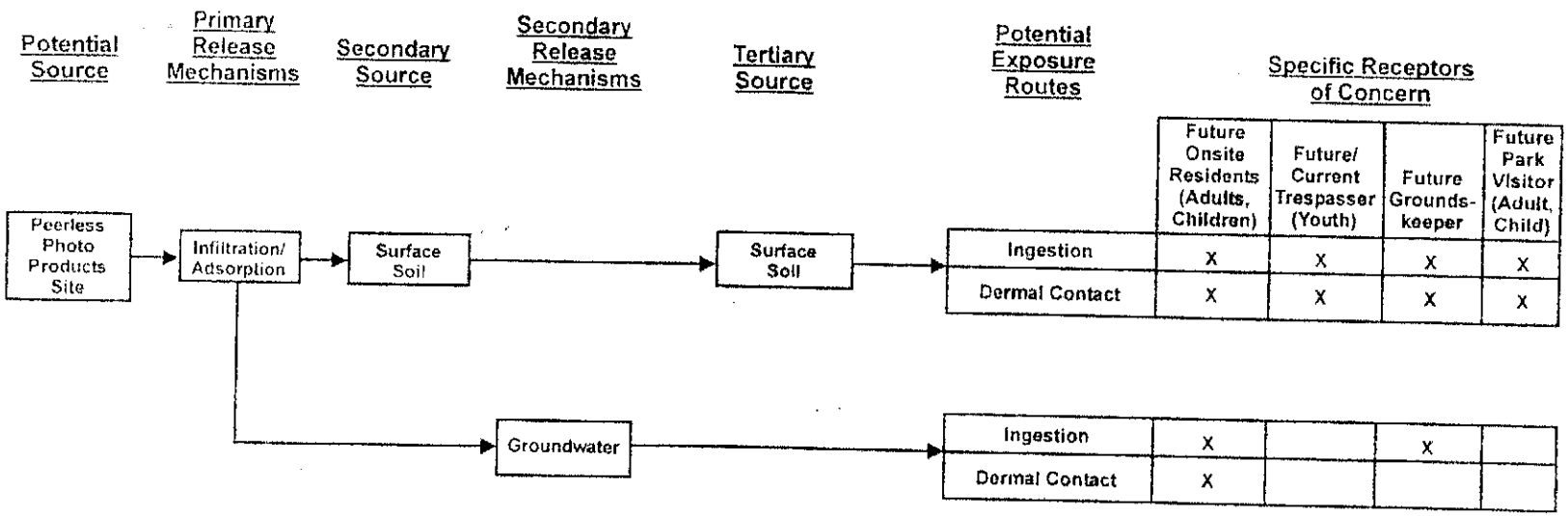

Figure 3. Human Health Conceptual Site Model: Peerless Photo Products Site, Shoreham, New York.

P:\Projects\Industrial&Other\Misc\AGFA\Fig3.cdr

Tables

TABLE 1
SAMPLES USED IN RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Sample Identificaation	Date Collected	Notes
Monitoring Well Background Samples		
MW-5	11/29/1994	MW-5
MW-5	3/28/1996	MW-5
MW-5	6/2/1997	MW-5
MW-5	7/16/1996	MW-5
MW-5	8/15/1994	MW-5
MW-5R	8/15/1994	Duplicate of MW-5
Monitoring Well Samples		
DR. PARDOS WELL	1/18/2002	DR. PARDOS WELL
DUP-1	12/4/2002	Duplicate of MW-7S
MW-1	1/21/2002	MW-1
MW-1	11/26/2002	MW-1
MW-1	11/29/1994	MW-1
MW-1	2/13/2001	MW-1
MW-1	3/28/1996	MW-1
MW-1	6/2/1997	MW-1
MW-1	7/17/1996	MW-1
MW-1	8/15/1994	MW-1
MW-2	1/21/2002	MW-2
MW-2	11/21/2002	MW-2
MW-2	11/30/1994	MW-2
MW-2	2/13/2001	MW-2
MW-2	3/29/1996	MW-2
MW-2	6/2/1997	MW-2
MW-2	7/17/1996	MW-2
MW-2	8/16/1994	MW-2
MW-2A	1/18/2002	Deep well couplet of MW-2A
MW-2A	11/22/2002	Deep well couplet of MW-2A
MW-2A	12/1/1994	Deep well couplet of MW-2A
MW-2A	2/14/2001	Deep well couplet of MW-2A
MW-2A	3/29/1996	Deep well couplet of MW-2A
MW-2A	6/2/1997	Deep well couplet of MW-2A
MW-2A	7/18/1996	Deep well couplet of MW-2A
MW-2A	8/17/1994	Deep well couplet of MW-2A
MW-3	1/21/2002	MW-3
MW-3	11/21/2002	MW-3
MW-3	11/30/1994	MW-3
MW-3	2/13/2001	MW-3
MW-3	4/3/1996	MW-3
MW-3	6/2/1997	MW-3
MW-3	7/18/1996	MW-3
MW-3	8/16/1994	MW-3
MW-4	1/18/2002	MW-4
MW-4	11/21/2002	MW-4
MW-4	11/30/1994	MW-4
MW-4	2/14/2001	MW-4
MW-4	3/29/1996	MW-4

TABLE 1
SAMPLES USED IN RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Sample Identificaation	Date Collected	Notes
MW-4	6/2/1997	MW-4
MW-4	7/17/1996	MW-4
MW-4	8/17/1994	MW-4
MW-6	1/29/2002	MW-6
MW-6	11/25/2002	MW-6
MW-6	12/1/1994	MW-6
MW-6	2/14/2001	MW-6
MW-6	3/28/1996	MW-6
MW-6	6/2/1997	MW-6
MW-6	7/17/1996	MW-6
MW-6	8/18/1994	MW-6
MW-7D	1/29/2002	MW-7D
MW-7D	12/4/2002	MW-7D
MW-7D	2/15/2001	MW-7D
MW-7D	6/2/1997	MW-7D
MW-7D	7/16/1996	MW-7D
MW-7S	1/21/2002	MW-7S
MW-7S	12/4/2002	MW-7S
MW-7S	2/5/2001	MW-7S
MW-7S	6/2/1997	MW-7S
MW-7S	7/16/1996	MW-7S
MW-8S	1/18/2002	MW-8S
MW-8S	11/25/2002	MW-8S
MW-8S	2/14/2001	MW-8S
MW-8S	6/2/1997	MW-8S
MW-8S	9/12/1996	MW-8S
MW-9	1/22/2002	MW-9
MW-9	11/25/2002	MW-9
MW-9	12/1/1994	MW-9
MW-9	2/13/2001	MW-9
MW-9	3/28/1996	MW-9
MW-9	6/2/1997	MW-9
MW-9	8/18/1994	MW-9
MW-9R	12/1/1994	Duplicate of MW-9
MW-10	1/23/2002	MW-10
MW-10	11/29/1994	MW-10
MW-10	12/5/2002	MW-10
MW-10	2/15/2001	MW-10
MW-10	3/29/1996	MW-10
MW-10	6/2/1997	MW-10
MW-10	7/18/1996	MW-10
MW-10	8/17/1994	MW-10
MW-10D	12/5/2002	MW-10D
MW-11D	12/5/2002	MW-11D
MW-11S	12/5/2002	MW-11S
REP-1 (7S)	6/2/1997	Duplicate of MW-7S
TW-1	12/3/2002	TW-1

TABLE 1
SAMPLES USED IN RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Sample Identificaation	Date Collected	Notes
TW-2	12/3/2002	TW-2
TW-3	12/3/2002	TW-3
Background Surface Soil Samples		
H1	7/22/1996	H1
H2	7/22/1996	H2
H3	7/22/1996	H3
K1	7/22/1996	K1
K2	7/22/1996	K2
M1	7/22/1996	M1
M2	7/22/1996	M2
M3	7/22/1996	M3
SB-16	6/21/1994	SB-16
On-site Surface Soil Samples		
B-2	10/3/1994	B-2
B-2-1D	9/5/1996	B-2-1D
B-2-1S	9/5/1996	B-2-1S
B-2-2D	9/5/1996	B-2-2D
B-2-2S	9/5/1996	B-2-2S
B-2-3D	9/5/1996	B-2-3D
B-2-3S	9/5/1996	B-2-3S
B-2-4D	9/5/1996	B-2-4D
B-2-4S	9/5/1996	B-2-4S
B-2-5D	9/5/1996	B-2-5D
B-2-5S	9/5/1996	B-2-5S
B-2-6D	9/5/1996	B-2-6D
B-2-6S	9/5/1996	B-2-6S
B-2-7D	9/5/1996	B-2-7D
B-2-7S	9/5/1996	B-2-7S
B-2-8D	9/5/1996	B-2-8D
B-2-8S	9/5/1996	B-2-8S
B-2-9D	9/5/1996	B-2-9D
B-2-9S	9/5/1996	B-2-9S
B-2-10D	9/5/1996	B-2-10D
B-2-10S	9/5/1996	B-2-10S
B-2-11D	9/5/1996	B-2-11D
B-2-11S	9/5/1996	B-2-11S
B-2-12D	9/5/1996	B-2-12D
B-2-12S	9/5/1996	B-2-12S
B-2-13D	9/5/1996	B-2-13D
B-2-13S	9/5/1996	B-2-13S
B-2-14D	9/5/1996	B-2-14D
B-2-14S	9/5/1996	B-2-14S
B-2-15D	9/5/1996	B-2-15D
B-2-15S	9/5/1996	B-2-15S
B-2-16D	9/5/1996	B-2-16D
B-2-16S	9/5/1996	B-2-16S
B-2-17D	9/5/1996	B-2-17D

TABLE 1
SAMPLES USED IN RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Sample Identificaation	Date Collected	Notes
B-2-17S	9/5/1996	B-2-17S
B-2-18S	9/5/1996	B-2-18S
B-7	10/3/1994	B-7
SB-1	5/19/1994	SB-1
SB-2	5/17/1994	SB-2
SB-3	5/18/1994	SB-3
SB-4	5/18/1994	SB-4
SB-6A-1	3/29/1999	SB-6A-1
SB-6A-2	3/29/1999	SB-6A-2
SB-6A-22	3/30/1999	SB-6A-22
SB-6A-25	3/29/1999	SB-6A-1
SB-6A-3	3/29/1999	SB-6A-3
SB-6B-1	3/30/1999	SB-6B-1
SB-6B-2	3/30/1999	SB-6B-2
SB-6B-22	3/31/1999	SB-6B-22
SB-6B-25	3/30/1999	SB-6B-1
SB-6B-3	3/30/1999	SB-6B-3
SB-6F-1	4/1/1999	SB-6F-1
SB-6F-10	4/1/1999	SB-6F-10
SB-6F-11	4/1/1999	SB-6F-11
SB-6F-12	4/1/1999	SB-6F-12
SB-6F-13	4/1/1999	SB-6F-13
SB-6F-14	4/1/1999	SB-6F-14
SB-6F-15	4/1/1999	SB-6F-15
SB-6F-16	4/1/1999	SB-6F-16
SB-6F-2	4/1/1999	SB-6F-2
SB-6F-3	4/1/1999	SB-6F-3
SB-6F-5	4/1/1999	SB-6F-5
SB-6F-6	4/1/1999	SB-6F-6
SB-6F-7	4/1/1999	SB-6F-7
SB-6F-8	4/1/1999	SB-6F-8
SB-6F-9	4/1/1999	SB-6F-9
SB-7	5/26/1994	SB-7
SB-8	5/26/1994	SB-8
SB-9	5/26/1994	SB-9
SB-9R	5/26/1994	Duplicate of SB-9
SB-10	5/26/1994	SB-10
SB-11	5/26/1994	SB-11
SB-12	5/26/1994	SB-12
SB-13	5/26/1994	SB-13
SB-20	8/4/1994	SB-20
SB-20R	8/4/1994	Duplicate of SB-20

TABLE 2
CHEMICALS OF CONCERN EVALUATED IN THE RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

On-site Surface Soils	Ground Water
--	Aluminum
Antimony	--
Barium	--
--	--
Cadmium	Cadmium
--	--
--	--
--	Cobalt
Copper	--
--	--
--	Lead*
--	Manganese
Mercury	--
Silver	--
--	Vanadium
Zinc	--

* = Lead was evaluated as a COPC as a conservative measure although it was eliminated as a COPC based on background.

TABLE 3
EXPOSURE ROUTES AND FACTORS FOR THE PEERLESS PHOTO PRODUCTS SITE

	Future On-site Adult Resident		Future On-site Child Resident		Future/Current Off-site (Area 11) Youth (age 9-18) Trespasser		Future Park Groundskeeper		Future Adult Park Visitor		Future Child (age 1-6) Park Visitor	
	Value	Reference	Value	Reference	Value	Reference	Value	Reference	Value	Reference	Value	Reference
GROUND WATER												
General Assumptions												
Exposure Frequency (days/year)	350	U.S. EPA 1997	350	U.S. EPA 1997			250	U.S. EPA 1997				
Exposure Duration (years)	24	U.S. EPA 1989	6	U.S. EPA 1991			25	U.S. EPA 1991				
Body Weight (kg)	70	U.S. EPA 1989	15	U.S. EPA 1991			70	U.S. EPA 1991				
Ingestion of Groundwater												
Ingestion Rate (L/day)	2	U.S. EPA 1991	2	U.S. EPA 1991			1	U.S. EPA 1991				
Dermal Contact with Groundwater While Showering												
Exposure Time (hours/day)	0.20	U.S. EPA 1992	0.2	U.S. EPA 1992								
Skin Surface Area Contacted (cm^2)	18,000	U.S. EPA 1992	6,600	U.S. EPA 1992								
Conversion Factor (L/cm^3)	1.00E-03	U.S. EPA 1989	0.001	U.S. EPA 1989								
SURFACE SOILS												
General Assumptions												
Exposure Frequency (days/year)	350	U.S. EPA 1997	350	U.S. EPA 1997	117	BPJ	250	U.S. EPA 1997	78	BPJ	78	BPJ
Exposure Duration (years)	24	U.S. EPA 1989	6	U.S. EPA 1991	10	U.S. EPA 1991	25	U.S. EPA 1991	30	U.S. EPA 1989	6	U.S. EPA 1991
Body Weight (kg)	70	U.S. EPA 1991	15	U.S. EPA 1991	50	U.S. EPA 1990	70	U.S. EPA 1991	70	U.S. EPA 1991	15	U.S. EPA 1991
Ingestion of Surface Soils												
Ingestion Rate (mg/day)	100	U.S. EPA 1989	200	U.S. EPA 1989	50	U.S. EPA 1991	100	U.S. EPA 1991	100	U.S. EPA 1989	200	U.S. EPA 1989a
Conversion Factor (kg/mg)	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Fraction Ingested (unitless)	1		1		1		1		1		1	
Dermal Contact with Surface Soils												
Skin Surface Area (cm^2)	5,700	U.S. EPA 2001	2,800	U.S. EPA 2001	4,690	ENVIRON 1997	5,700	U.S. EPA 2001	5.70E+03	U.S. EPA 2001	2,800	U.S. EPA 2001
Adherence Factor (mg/cm^2)	0.07	U.S. EPA 2001	0.2	U.S. EPA 2001	0.07	ENVIRON 1997	0.07	U.S. EPA 2001	0.07	U.S. EPA 2001	0.2	U.S. EPA 2001
Absorption Factor (unitless)	CS	see Table D-7	CS	see Table D-7	CS	see Table D-7	CS	see Table D-7	CS	see Table D-7	CS	see Table D-7

CS = Chemical Specific
BPJ = Best Professional Judgement

TABLE 4
TOXICITY VALUES FOR CHEMICALS OF CONCERN AT THE PEERLESS PHOTO PRODUCTS SITE

Chemical of Concern	Weight-of-Evidence Classification	Oral Cancer Slope Factor (per mg/kg-day) CSFo	CSFo Reference	CSFo Reference Date	Oral RfD (mg/kg-day) RfDo	Derived Dermal RfD (mg/kg-day) RfDd	RfDo/RfDd Reference	RfDo/RfDd Reference Date
Inorganics								
ALUMINUM	D	NA	EPA-NCEA	5/30/1997	1.00E+00	1.00E+00	EPA-NCEA	5/30/1997
ANTIMONY	NA	NA	IRIS	3/21/2003	4.00E-04	6.00E-05	IRIS	3/21/2003
BARIUM	D	NA	IRIS	3/21/2003	7.00E-02	4.90E-03	IRIS	3/21/2003
CADMIUM	B1	NA	IRIS	3/21/2003	5.00E-04	1.25E-05	IRIS	3/21/2003
COBALT	NA	NA	EPA-NCEA	8/7/2001	2.00E-02	2.00E-02	EPA-NCEA	8/7/2001
COPPER	D	NA	IRIS	3/21/2003	4.00E-02	4.00E-02	HEAST	7/25/1997
MANGANESE	D	NA	IRIS	3/21/2003	2.00E-02	8.00E-04	IRIS	3/21/2003
MERCURY	D	NA	IRIS	3/21/2003	1.00E-04	1.00E-04	IRIS	3/21/2003
SILVER	D	NA	IRIS	3/21/2003	5.00E-03	2.00E-04	IRIS	3/21/2003
VANADIUM	D	NA	HEAST	7/25/1997	7.00E-03	1.82E-04	HEAST	7/25/1997
ZINC	D	NA	IRIS	3/21/2003	3.00E-01	3.00E-01	IRIS	3/21/2003

N/A= Not Applicable

(1) Dermal Toxicological values adjusted from oral values using USEPA 2000 recommended chemical-specific gastrointestinal absorption factors(GI

(2) For IRIS values, the date IRIS was searched is provided.
For HEAST values, the date of HEAST is provided.
For EPA-NCEA values, the date of the article provided by NCEA is provided.

Weight of Evidence: A - Human carcinogen
B1 - Probable human carcinogen - indicate that llimited human data are available
B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
C - Posible human carcinogen
D - Not classifiable as a human carcinogen
E - Evidence of noncarcinogenicity

TABLE 5
SUMMARY OF HAZARD INDICES AT THE PEERLESS PHOTO PRODUCTS SITE

	Hazard Index	
	Excluding Ground Water Exposure	Including Ground Water Exposure
Future On-site Adult Resident		
Surface Soil Ingestion	3.46E+00	3.46E+00
Surface Soil Dermal Contact	6.39E-02	6.39E-02
Ground Water Ingestion		9.10E+00
Ground Water Dermal Contact		6.09E-01
Total	3.52E+00	1.32E+01
Future On-site Child Resident		
Surface Soil Ingestion	3.23E+01	3.23E+01
Surface Soil Dermal Contact	4.19E-01	4.19E-01
Ground Water Ingestion		4.25E+01
Ground Water Dermal Contact		1.04E+00
Total	3.27E+01	7.62E+01
Future/Current Youth Trespasser		
Surface Soil Ingestion	8.09E-01	8.09E-01
Surface Soil Dermal Contact	2.46E-02	2.46E-02
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	8.34E-01	8.34E-01
Future On-site Park Groundskeeper		
Surface Soil Ingestion	2.47E+00	2.47E+00
Surface Soil Dermal Contact	4.57E-02	4.57E-02
Ground Water Ingestion		3.25E+00
Ground Water Dermal Contact		
Total	2.52E+00	5.77E+00
Future Adult Park Visitor		
Surface Soil Ingestion	7.71E-01	7.71E-01
Surface Soil Dermal Contact	1.42E-02	1.42E-02
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	7.85E-01	7.85E-01
Future Child (age 1-6) Park Visitor		
Surface Soil Ingestion	7.19E+00	7.19E+00
Surface Soil Dermal Contact	9.33E-02	9.33E-02
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	7.29E+00	7.29E+00

TABLE 6
SUMMARY CANCER OF RISKS AT THE PEERLESS PHOTO PRODUCTS SITE

	Cancer Risk	
	Excluding Ground Water Exposure	Including Ground Water Exposure
Future On-site Adult Resident		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		0.00E+00
Ground Water Dermal Contact		0.00E+00
Total	None	None
Future On-site Child Resident		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		0.00E+00
Ground Water Dermal Contact		0.00E+00
Total	None	None
Future On-site Resident		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		0.00E+00
Ground Water Dermal Contact		0.00E+00
Total	None	None
Future/Current Youth Trespasser		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	None	None
Future On-site Park Groundskeeper		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		0.00E+00
Ground Water Dermal Contact		
Total	None	None
Future Adult Park Visitor		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	None	None
Future Child (age 1-6) Park Visitor		
Surface Soil Ingestion	0.00E+00	0.00E+00
Surface Soil Dermal Contact	0.00E+00	0.00E+00
Ground Water Ingestion		
Ground Water Dermal Contact		
Total	None	None

Appendix A

Analytical Data

APPENDIX A
ANALYTICAL DATA USED IN RISK ASSESSMENT
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Chemical	Sample Identification	Date Collected	Result
Groundwater Background Samples (ppb)			
Aluminum	MW-5	11/29/1994	388
Aluminum	MW-5	3/28/1996	233 U
Aluminum	MW-5	7/16/1996	262
Aluminum	MW-5	8/15/1994	474
Antimony	MW-5	11/29/1994	19.6 B
Antimony	MW-5	3/28/1996	3 U
Antimony	MW-5	7/16/1996	6 U
Antimony	MW-5	8/15/1994	12 U
Arsenic	MW-5	11/29/1994	4 U
Arsenic	MW-5	3/28/1996	5 U
Arsenic	MW-5	7/16/1996	5 U
Arsenic	MW-5	8/15/1994	3.6 J
Barium	MW-5	11/29/1994	66.4 B
Barium	MW-5	3/28/1996	50 B
Barium	MW-5	7/16/1996	39.8 B
Barium	MW-5	8/15/1994	59.8 B
Beryllium	MW-5	11/29/1994	1 U
Beryllium	MW-5	3/28/1996	1 U
Beryllium	MW-5	7/16/1996	1 U
Beryllium	MW-5	8/15/1994	1 U
Cadmium	MW-5	11/29/1994	3 U
Cadmium	MW-5	3/28/1996	1 U
Cadmium	MW-5	6/2/1997	2.9 B
Cadmium	MW-5	7/16/1996	1 U
Cadmium	MW-5	8/15/1994	3.9 J
Calcium	MW-5	11/29/1994	12300
Calcium	MW-5	3/28/1996	7920
Calcium	MW-5	7/16/1996	7550
Calcium	MW-5	8/15/1994	11400
Chromium	MW-5	11/29/1994	4.7 B
Chromium	MW-5	3/28/1996	1.2 U
Chromium	MW-5	6/2/1997	14.1
Chromium	MW-5	7/16/1996	4 U
Chromium	MW-5	8/15/1994	2 U
Cobalt	MW-5	11/29/1994	3.7 B
Cobalt	MW-5	3/28/1996	1 U
Cobalt	MW-5	7/16/1996	1 U
Cobalt	MW-5	8/15/1994	2 U
Copper	MW-5	11/29/1994	4.9 B
Copper	MW-5	3/28/1996	9 U
Copper	MW-5	7/16/1996	1 U
Copper	MW-5	8/15/1994	6.6 U

Chemical	Sample Identification	Date Collected	Result
Cyanide	MW-5	11/29/1994	10 U
Cyanide	MW-5	3/28/1996	2 U
Cyanide	MW-5	8/15/1994	10 U
Iron	MW-5	11/29/1994	1100
Iron	MW-5	3/28/1996	101
Iron	MW-5	7/16/1996	308
Iron	MW-5	8/15/1994	1280
Lead	MW-5	11/29/1994	22.2
Lead	MW-5	3/28/1996	2 U
Lead	MW-5	6/2/1997	3.8
Lead	MW-5	7/16/1996	2.1 J
Lead	MW-5	8/15/1994	5.1 U
Magnesium	MW-5	11/29/1994	5090
Magnesium	MW-5	3/28/1996	4090 B
Magnesium	MW-5	7/16/1996	3690 B
Magnesium	MW-5	8/15/1994	4040 B
Manganese	MW-5	11/29/1994	188
Manganese	MW-5	3/28/1996	50.2
Manganese	MW-5	7/16/1996	47
Manganese	MW-5	8/15/1994	358
Mercury	MW-5	11/29/1994	0.24 U
Mercury	MW-5	3/28/1996	0.15 U
Mercury	MW-5	6/2/1997	0.06 U
Mercury	MW-5	7/16/1996	0.09 B
Mercury	MW-5	8/15/1994	0.24 U
Nickel	MW-5	11/29/1994	12 U
Nickel	MW-5	3/28/1996	5.8 B
Nickel	MW-5	7/16/1996	3 U
Nickel	MW-5	8/15/1994	12 U
Potassium	MW-5	11/29/1994	5390
Potassium	MW-5	3/28/1996	1980 B
Potassium	MW-5	7/16/1996	2370 B
Potassium	MW-5	8/15/1994	3420 B
Selenium	MW-5	11/29/1994	1 U
Selenium	MW-5	3/28/1996	3 U
Selenium	MW-5	7/16/1996	3 U
Selenium	MW-5	8/15/1994	2 U
Silver	MW-5	11/29/1994	2.4 B
Silver	MW-5	3/28/1996	1 U
Silver	MW-5	6/2/1997	2 U
Silver	MW-5	7/16/1996	1 U
Silver	MW-5	8/15/1994	2 UJ
Sodium	MW-5	11/29/1994	23300
Sodium	MW-5	3/28/1996	18700
Sodium	MW-5	7/16/1996	17700
Sodium	MW-5	8/15/1994	25700
Thallium	MW-5	11/29/1994	3 U
Thallium	MW-5	3/28/1996	5 U
Thallium	MW-5	7/16/1996	6 U

Chemical	Sample Identification	Date Collected	Result
Thallium	MW-5	8/15/1994	3 U
Vanadium	MW-5	11/29/1994	3.7 B
Vanadium	MW-5	3/28/1996	1 U
Vanadium	MW-5	7/16/1996	1 U
Vanadium	MW-5	8/15/1994	3 U
Zinc	MW-5	11/29/1994	20.2
Zinc	MW-5	3/28/1996	17.7 U
Zinc	MW-5	7/16/1996	13.8 U
Zinc	MW-5	8/15/1994	39 U
Aluminum	MW-5R	8/15/1994	326
Antimony	MW-5R	8/15/1994	12 U
Arsenic	MW-5R	8/15/1994	3 U
Barium	MW-5R	8/15/1994	57 B
Beryllium	MW-5R	8/15/1994	1 U
Cadmium	MW-5R	8/15/1994	3 U
Calcium	MW-5R	8/15/1994	11100
Chromium	MW-5R	8/15/1994	2 U
Cobalt	MW-5R	8/15/1994	2 U
Copper	MW-5R	8/15/1994	3.5 U
Cyanide	MW-5R	8/15/1994	10 U
Iron	MW-5R	8/15/1994	426
Lead	MW-5R	8/15/1994	6 U
Magnesium	MW-5R	8/15/1994	3900 B
Manganese	MW-5R	8/15/1994	258
Mercury	MW-5R	8/15/1994	0.24 U
Nickel	MW-5R	8/15/1994	12 U
Potassium	MW-5R	8/15/1994	3570 B
Selenium	MW-5R	8/15/1994	2 U
Silver	MW-5R	8/15/1994	2 UJ
Sodium	MW-5R	8/15/1994	25300
Thallium	MW-5R	8/15/1994	1 U
Vanadium	MW-5R	8/15/1994	3 U
Zinc	MW-5R	8/15/1994	35.3 U
Monitoring Well Samples (ppb)			
Cadmium	DR. PARDOS WELL	1/18/2002	1 U
Chromium	DR. PARDOS WELL	1/18/2002	8 U
Lead	DR. PARDOS WELL	1/18/2002	2 U
Mercury	DR. PARDOS WELL	1/18/2002	0.5 U
Silver	DR. PARDOS WELL	1/18/2002	2 U
Cadmium	DUP-1	12/4/2002	2.56
Chromium	DUP-1	12/4/2002	19.2
Lead	DUP-1	12/4/2002	2 U
Mercury	DUP-1	12/4/2002	0.5 U
Silver	DUP-1	12/4/2002	2 U
Aluminum	MW-1	11/29/1994	3830
Aluminum	MW-1	3/28/1996	4710
Aluminum	MW-1	7/17/1996	1060
Aluminum	MW-1	8/15/1994	4880
Antimony	MW-1	11/29/1994	14.4 B

Chemical	Sample Identification	Date Collected	Result
Antimony	MW-1	3/28/1996	3 U
Antimony	MW-1	7/17/1996	6 U
Antimony	MW-1	8/15/1994	12 U
Arsenic	MW-1	11/29/1994	4 U
Arsenic	MW-1	3/28/1996	5.9 B
Arsenic	MW-1	7/17/1996	5 U
Arsenic	MW-1	8/15/1994	7 J
Barium	MW-1	11/29/1994	125 B
Barium	MW-1	3/28/1996	329
Barium	MW-1	7/17/1996	59.5 B
Barium	MW-1	8/15/1994	178 B
Beryllium	MW-1	11/29/1994	1 U
Beryllium	MW-1	3/28/1996	1 U
Beryllium	MW-1	7/17/1996	1 U
Beryllium	MW-1	8/15/1994	1 U
Cadmium	MW-1	1/21/2002	1 U
Cadmium	MW-1	11/26/2002	1 U
Cadmium	MW-1	11/29/1994	3 U
Cadmium	MW-1	2/13/2001	0.5 U
Cadmium	MW-1	3/28/1996	1 U
Cadmium	MW-1	6/2/1997	0.5 U
Cadmium	MW-1	7/17/1996	1 U
Cadmium	MW-1	8/15/1994	4.8 J
Calcium	MW-1	11/29/1994	17900
Calcium	MW-1	3/28/1996	51300
Calcium	MW-1	7/17/1996	9800
Calcium	MW-1	8/15/1994	23000
Chromium	MW-1	1/21/2002	8 U
Chromium	MW-1	11/26/2002	8.83
Chromium	MW-1	11/29/1994	21
Chromium	MW-1	2/13/2001	10.5
Chromium	MW-1	3/28/1996	64
Chromium	MW-1	6/2/1997	16.5
Chromium	MW-1	7/17/1996	6.7 B
Chromium	MW-1	8/15/1994	18.6
Cobalt	MW-1	11/29/1994	9.9 B
Cobalt	MW-1	3/28/1996	12 B
Cobalt	MW-1	7/17/1996	2.6 B
Cobalt	MW-1	8/15/1994	13.4 B
Copper	MW-1	11/29/1994	21.8 B
Copper	MW-1	3/28/1996	30.6
Copper	MW-1	7/17/1996	4.4 B
Copper	MW-1	8/15/1994	35.2
Cyanide	MW-1	11/29/1994	10 U
Cyanide	MW-1	3/28/1996	2 U
Cyanide	MW-1	8/15/1994	10 U
Iron	MW-1	11/29/1994	10700
Iron	MW-1	3/28/1996	14000
Iron	MW-1	7/17/1996	2510

Chemical	Sample Identification	Date Collected	Result
Iron	MW-1	8/15/1994	14800
Lead	MW-1	1/21/2002	2 U
Lead	MW-1	11/26/2002	2 U
Lead	MW-1	11/29/1994	34
Lead	MW-1	2/13/2001	3.4
Lead	MW-1	3/28/1996	14.4
Lead	MW-1	6/2/1997	6.2
Lead	MW-1	7/17/1996	4.6 J
Lead	MW-1	8/15/1994	29.8
Magnesium	MW-1	11/29/1994	5680
Magnesium	MW-1	3/28/1996	4860 B
Magnesium	MW-1	7/17/1996	4200 B
Magnesium	MW-1	8/15/1994	5080
Manganese	MW-1	11/29/1994	1280
Manganese	MW-1	3/28/1996	1460
Manganese	MW-1	7/17/1996	276
Manganese	MW-1	8/15/1994	1680
Mercury	MW-1	1/21/2002	0.5 U
Mercury	MW-1	11/26/2002	0.5 U
Mercury	MW-1	11/29/1994	0.24 U
Mercury	MW-1	2/13/2001	0.1 U
Mercury	MW-1	3/28/1996	0.19 J
Mercury	MW-1	6/2/1997	0.06 U
Mercury	MW-1	7/17/1996	0.08 U
Mercury	MW-1	8/15/1994	0.24 U
Nickel	MW-1	11/29/1994	15.9 B
Nickel	MW-1	3/28/1996	50.1
Nickel	MW-1	7/17/1996	5.7 B
Nickel	MW-1	8/15/1994	22.1 B
Potassium	MW-1	11/29/1994	3440 B
Potassium	MW-1	3/28/1996	2720 B
Potassium	MW-1	7/17/1996	2460 B
Potassium	MW-1	8/15/1994	3380 B
Selenium	MW-1	11/29/1994	1 U
Selenium	MW-1	3/28/1996	3 U
Selenium	MW-1	7/17/1996	3 U
Selenium	MW-1	8/15/1994	2 U
Silver	MW-1	1/21/2002	2 U
Silver	MW-1	11/26/2002	2 U
Silver	MW-1	11/29/1994	2 U
Silver	MW-1	2/13/2001	2.4 U
Silver	MW-1	3/28/1996	1.5 B
Silver	MW-1	6/2/1997	2 U
Silver	MW-1	7/17/1996	1.2 B
Silver	MW-1	8/15/1994	2 UJ
Sodium	MW-1	11/29/1994	15900
Sodium	MW-1	3/28/1996	16000
Sodium	MW-1	7/17/1996	12900
Sodium	MW-1	8/15/1994	17400

Chemical	Sample Identification	Date Collected	Result
Thallium	MW-1	11/29/1994	1 U
Thallium	MW-1	3/28/1996	5 U
Thallium	MW-1	7/17/1996	6 U
Thallium	MW-1	8/15/1994	3 U
Vanadium	MW-1	11/29/1994	13.5 B
Vanadium	MW-1	3/28/1996	13.4 B
Vanadium	MW-1	7/17/1996	2.8 B
Vanadium	MW-1	8/15/1994	18 B
Zinc	MW-1	11/29/1994	56.8
Zinc	MW-1	3/28/1996	65.6 U
Zinc	MW-1	7/17/1996	26.3 U
Zinc	MW-1	8/15/1994	158
Aluminum	MW-10	11/29/1994	107 B
Aluminum	MW-10	3/29/1996	97.1 B
Aluminum	MW-10	7/18/1996	249
Aluminum	MW-10	8/17/1994	171 B
Antimony	MW-10	11/29/1994	12 U
Antimony	MW-10	3/29/1996	3 U
Antimony	MW-10	7/18/1996	6 U
Antimony	MW-10	8/17/1994	12 U
Arsenic	MW-10	11/29/1994	4 U
Arsenic	MW-10	3/29/1996	2 U
Arsenic	MW-10	7/18/1996	5 U
Arsenic	MW-10	8/17/1994	3 U
Barium	MW-10	11/29/1994	53.2 B
Barium	MW-10	3/29/1996	23 B
Barium	MW-10	7/18/1996	20.6 B
Barium	MW-10	8/17/1994	33 B
Beryllium	MW-10	11/29/1994	1 U
Beryllium	MW-10	3/29/1996	1 U
Beryllium	MW-10	7/18/1996	1 U
Beryllium	MW-10	8/17/1994	1 U
Cadmium	MW-10	1/23/2002	57
Cadmium	MW-10	11/29/1994	73.4
Cadmium	MW-10	12/5/2002	51.9
Cadmium	MW-10	2/15/2001	50.2
Cadmium	MW-10	3/29/1996	44.1
Cadmium	MW-10	6/2/1997	68.7
Cadmium	MW-10	7/18/1996	14.9
Cadmium	MW-10	8/17/1994	46.4
Calcium	MW-10	11/29/1994	11600
Calcium	MW-10	3/29/1996	7120
Calcium	MW-10	7/18/1996	7580
Calcium	MW-10	8/17/1994	11200
Chromium	MW-10	1/23/2002	8 U
Chromium	MW-10	11/29/1994	2 U
Chromium	MW-10	12/5/2002	16
Chromium	MW-10	2/15/2001	2.1 U
Chromium	MW-10	3/29/1996	2.2 U

Chemical	Sample Identification	Date Collected	Result
Chromium	MW-10	6/2/1997	2.4 B
Chromium	MW-10	7/18/1996	4 U
Chromium	MW-10	8/17/1994	2 U
Cobalt	MW-10	11/29/1994	2 U
Cobalt	MW-10	3/29/1996	4 U
Cobalt	MW-10	7/18/1996	1 U
Cobalt	MW-10	8/17/1994	2 U
Copper	MW-10	11/29/1994	2 U
Copper	MW-10	3/29/1996	2.7 U
Copper	MW-10	7/18/1996	1 U
Copper	MW-10	8/17/1994	2 U
Cyanide	MW-10	11/29/1994	10 U
Cyanide	MW-10	3/29/1996	2 U
Cyanide	MW-10	8/17/1994	10 U
Iron	MW-10	11/29/1994	174
Iron	MW-10	3/29/1996	70.9 U
Iron	MW-10	7/18/1996	150 U
Iron	MW-10	8/17/1994	104
Lead	MW-10	1/23/2002	2 U
Lead	MW-10	11/29/1994	20.3
Lead	MW-10	12/5/2002	2 U
Lead	MW-10	2/15/2001	2.6 U
Lead	MW-10	3/29/1996	2 U
Lead	MW-10	6/2/1997	2.5 B
Lead	MW-10	7/18/1996	2 U
Lead	MW-10	8/17/1994	2.7 U
Magnesium	MW-10	11/29/1994	4230 B
Magnesium	MW-10	3/29/1996	2910 B
Magnesium	MW-10	7/18/1996	3700 B
Magnesium	MW-10	8/17/1994	3520 B
Manganese	MW-10	11/29/1994	104
Manganese	MW-10	3/29/1996	58.2
Manganese	MW-10	7/18/1996	29.4
Manganese	MW-10	8/17/1994	94.6
Mercury	MW-10	1/23/2002	0.5 U
Mercury	MW-10	11/29/1994	0.24 U
Mercury	MW-10	12/5/2002	0.5 U
Mercury	MW-10	2/15/2001	0.1 U
Mercury	MW-10	3/29/1996	0.15 U
Mercury	MW-10	6/2/1997	0.06 U
Mercury	MW-10	7/18/1996	0.08 U
Mercury	MW-10	8/17/1994	0.24 U
Nickel	MW-10	11/29/1994	12 U
Nickel	MW-10	3/29/1996	2.8 U
Nickel	MW-10	7/18/1996	3 U
Nickel	MW-10	8/17/1994	12 U
Potassium	MW-10	11/29/1994	1710 U
Potassium	MW-10	3/29/1996	1230 B
Potassium	MW-10	7/18/1996	1610 B

Chemical	Sample Identification	Date Collected	Result
Potassium	MW-10	8/17/1994	2110 B
Selenium	MW-10	11/29/1994	1 U
Selenium	MW-10	3/29/1996	2 U
Selenium	MW-10	7/18/1996	3 U
Selenium	MW-10	8/17/1994	2 U
Silver	MW-10	1/23/2002	2 U
Silver	MW-10	11/29/1994	2 UJ
Silver	MW-10	12/5/2002	2 U
Silver	MW-10	2/15/2001	2.3 U
Silver	MW-10	3/29/1996	1 U
Silver	MW-10	6/2/1997	2 U
Silver	MW-10	7/18/1996	1 U
Silver	MW-10	8/17/1994	2 UJ
Sodium	MW-10	11/29/1994	19100
Sodium	MW-10	3/29/1996	8990
Sodium	MW-10	7/18/1996	8510
Sodium	MW-10	8/17/1994	16100
Thallium	MW-10	11/29/1994	1 U
Thallium	MW-10	3/29/1996	3 U
Thallium	MW-10	7/18/1996	6 U
Thallium	MW-10	8/17/1994	3 U
Vanadium	MW-10	11/29/1994	3 U
Vanadium	MW-10	3/29/1996	1 U
Vanadium	MW-10	7/18/1996	1 U
Vanadium	MW-10	8/17/1994	3 U
Zinc	MW-10	11/29/1994	20.8
Zinc	MW-10	3/29/1996	30 U
Zinc	MW-10	7/18/1996	23.8
Zinc	MW-10	8/17/1994	27.4
Cadmium	MW-10D	12/5/2002	1 U
Chromium	MW-10D	12/5/2002	8.14
Lead	MW-10D	12/5/2002	2 U
Mercury	MW-10D	12/5/2002	0.5 U
Silver	MW-10D	12/5/2002	2 U
Cadmium	MW-11D	12/5/2002	1 U
Chromium	MW-11D	12/5/2002	10.7
Lead	MW-11D	12/5/2002	2 U
Mercury	MW-11D	12/5/2002	0.5 U
Silver	MW-11D	12/5/2002	2 U
Cadmium	MW-11S	12/5/2002	1 U
Chromium	MW-11S	12/5/2002	13.4
Lead	MW-11S	12/5/2002	2 U
Mercury	MW-11S	12/5/2002	0.5 U
Silver	MW-11S	12/5/2002	2 U
Aluminum	MW-2	11/30/1994	1430
Aluminum	MW-2	3/29/1996	2490
Aluminum	MW-2	7/17/1996	829
Aluminum	MW-2	8/16/1994	3310
Antimony	MW-2	11/30/1994	12 U

Chemical	Sample Identification	Date Collected	Result
Antimony	MW-2	3/29/1996	3 U
Antimony	MW-2	7/17/1996	6 U
Antimony	MW-2	8/16/1994	15.1 B
Arsenic	MW-2	11/30/1994	4 U
Arsenic	MW-2	3/29/1996	5 U
Arsenic	MW-2	7/17/1996	5 U
Arsenic	MW-2	8/16/1994	4.3 J
Barium	MW-2	11/30/1994	96.5 B
Barium	MW-2	3/29/1996	91.4 B
Barium	MW-2	7/17/1996	51.6 B
Barium	MW-2	8/16/1994	118 B
Beryllium	MW-2	11/30/1994	1 U
Beryllium	MW-2	3/29/1996	1 U
Beryllium	MW-2	7/17/1996	1 U
Beryllium	MW-2	8/16/1994	1 U
Cadmium	MW-2	1/21/2002	80
Cadmium	MW-2	11/21/2002	79.8
Cadmium	MW-2	11/30/1994	107
Cadmium	MW-2	2/13/2001	77.1
Cadmium	MW-2	3/29/1996	115
Cadmium	MW-2	6/2/1997	150
Cadmium	MW-2	7/17/1996	84.7
Cadmium	MW-2	8/16/1994	135
Calcium	MW-2	11/30/1994	12800
Calcium	MW-2	3/29/1996	10900
Calcium	MW-2	7/17/1996	8950
Calcium	MW-2	8/16/1994	12300
Chromium	MW-2	1/21/2002	8 U
Chromium	MW-2	11/21/2002	72.3
Chromium	MW-2	11/30/1994	8.9 B
Chromium	MW-2	2/13/2001	27.8
Chromium	MW-2	3/29/1996	17.8
Chromium	MW-2	6/2/1997	18.9
Chromium	MW-2	7/17/1996	7.7 B
Chromium	MW-2	8/16/1994	11.1 U
Cobalt	MW-2	11/30/1994	5 B
Cobalt	MW-2	3/29/1996	12 B
Cobalt	MW-2	7/17/1996	2.4 B
Cobalt	MW-2	8/16/1994	11.5 B
Copper	MW-2	11/30/1994	12 B
Copper	MW-2	3/29/1996	27.3
Copper	MW-2	7/17/1996	4.5 B
Copper	MW-2	8/16/1994	31.5
Cyanide	MW-2	11/30/1994	10 U
Cyanide	MW-2	3/29/1996	2 U
Cyanide	MW-2	8/16/1994	10 U
Iron	MW-2	11/30/1994	5300
Iron	MW-2	3/29/1996	6920
Iron	MW-2	7/17/1996	2110

Chemical	Sample Identification	Date Collected	Result
Iron	MW-2	8/16/1994	10700
Lead	MW-2	1/21/2002	2 U
Lead	MW-2	11/21/2002	2 U
Lead	MW-2	11/30/1994	10.2
Lead	MW-2	2/13/2001	4.4
Lead	MW-2	3/29/1996	7.4
Lead	MW-2	6/2/1997	15.8
Lead	MW-2	7/17/1996	3 J
Lead	MW-2	8/16/1994	20.3 U
Magnesium	MW-2	11/30/1994	6380
Magnesium	MW-2	3/29/1996	5710
Magnesium	MW-2	7/17/1996	4570 B
Magnesium	MW-2	8/16/1994	6210
Manganese	MW-2	11/30/1994	533
Manganese	MW-2	3/29/1996	917
Manganese	MW-2	7/17/1996	256
Manganese	MW-2	8/16/1994	1390
Mercury	MW-2	1/21/2002	0.5 U
Mercury	MW-2	11/21/2002	0.5 U
Mercury	MW-2	11/30/1994	0.24 U
Mercury	MW-2	2/13/2001	0.1 U
Mercury	MW-2	3/29/1996	0.15 U
Mercury	MW-2	6/2/1997	0.06 U
Mercury	MW-2	7/17/1996	0.08 U
Mercury	MW-2	8/16/1994	0.24 U
Nickel	MW-2	11/30/1994	12 U
Nickel	MW-2	3/29/1996	29.2 B
Nickel	MW-2	7/17/1996	11.7 B
Nickel	MW-2	8/16/1994	21.4 B
Potassium	MW-2	11/30/1994	3630 B
Potassium	MW-2	3/29/1996	2500 B
Potassium	MW-2	7/17/1996	2460 B
Potassium	MW-2	8/16/1994	5430
Selenium	MW-2	11/30/1994	1 U
Selenium	MW-2	3/29/1996	3 U
Selenium	MW-2	7/17/1996	3 U
Selenium	MW-2	8/16/1994	2 U
Silver	MW-2	1/21/2002	2 U
Silver	MW-2	11/21/2002	2 U
Silver	MW-2	11/30/1994	2 U
Silver	MW-2	2/13/2001	2.3 U
Silver	MW-2	3/29/1996	1.2 B
Silver	MW-2	6/2/1997	2 U
Silver	MW-2	7/17/1996	1 U
Silver	MW-2	8/16/1994	3 J
Sodium	MW-2	11/30/1994	23600
Sodium	MW-2	3/29/1996	13000
Sodium	MW-2	7/17/1996	10300
Sodium	MW-2	8/16/1994	22400

Chemical	Sample Identification	Date Collected	Result
Thallium	MW-2	11/30/1994	1 U
Thallium	MW-2	3/29/1996	5 U
Thallium	MW-2	7/17/1996	6 U
Thallium	MW-2	8/16/1994	3 U
Vanadium	MW-2	11/30/1994	7.4 B
Vanadium	MW-2	3/29/1996	8.1 B
Vanadium	MW-2	7/17/1996	2.4 B
Vanadium	MW-2	8/16/1994	13 B
Zinc	MW-2	11/30/1994	52.3
Zinc	MW-2	3/29/1996	61.8 U
Zinc	MW-2	7/17/1996	41.2
Zinc	MW-2	8/16/1994	76.1
Aluminum	MW-2A	12/1/1994	159 B
Aluminum	MW-2A	3/29/1996	363 U
Aluminum	MW-2A	7/18/1996	257 U
Aluminum	MW-2A	8/17/1994	167 B
Antimony	MW-2A	12/1/1994	12 U
Antimony	MW-2A	3/29/1996	3 U
Antimony	MW-2A	7/18/1996	6 U
Antimony	MW-2A	8/17/1994	12 U
Arsenic	MW-2A	12/1/1994	4 U
Arsenic	MW-2A	3/29/1996	5 U
Arsenic	MW-2A	7/18/1996	5 U
Arsenic	MW-2A	8/17/1994	3 U
Barium	MW-2A	12/1/1994	13.6 B
Barium	MW-2A	3/29/1996	13.7 B
Barium	MW-2A	7/18/1996	7.9 U
Barium	MW-2A	8/17/1994	36.2 B
Beryllium	MW-2A	12/1/1994	1 U
Beryllium	MW-2A	3/29/1996	1 U
Beryllium	MW-2A	7/18/1996	1 U
Beryllium	MW-2A	8/17/1994	1 U
Cadmium	MW-2A	1/18/2002	7.9
Cadmium	MW-2A	11/22/2002	1.6
Cadmium	MW-2A	12/1/1994	6.2
Cadmium	MW-2A	2/14/2001	0.68 B
Cadmium	MW-2A	3/29/1996	5.1
Cadmium	MW-2A	6/2/1997	10
Cadmium	MW-2A	7/18/1996	8.8
Cadmium	MW-2A	8/17/1994	3.4 J
Calcium	MW-2A	12/1/1994	10800
Calcium	MW-2A	3/29/1996	5780
Calcium	MW-2A	7/18/1996	5020
Calcium	MW-2A	8/17/1994	19600
Chromium	MW-2A	1/18/2002	8 U
Chromium	MW-2A	11/22/2002	32.7
Chromium	MW-2A	12/1/1994	7.7 B
Chromium	MW-2A	2/14/2001	2.1 U
Chromium	MW-2A	3/29/1996	3.6 U

Chemical	Sample Identification	Date Collected	Result
Chromium	MW-2A	6/2/1997	5.1 B
Chromium	MW-2A	7/18/1996	4 U
Chromium	MW-2A	8/17/1994	8.6 B
Cobalt	MW-2A	12/1/1994	2.1 B
Cobalt	MW-2A	3/29/1996	1 B
Cobalt	MW-2A	7/18/1996	1 U
Cobalt	MW-2A	8/17/1994	2 U
Copper	MW-2A	12/1/1994	5.4 B
Copper	MW-2A	3/29/1996	11.7 B
Copper	MW-2A	7/18/1996	3.3 B
Copper	MW-2A	8/17/1994	14.4 B
Cyanide	MW-2A	12/1/1994	10 U
Cyanide	MW-2A	3/29/1996	2 U
Cyanide	MW-2A	8/17/1994	10 U
Iron	MW-2A	12/1/1994	552
Iron	MW-2A	3/29/1996	757
Iron	MW-2A	7/18/1996	397
Iron	MW-2A	8/17/1994	553
Lead	MW-2A	1/18/2002	6.7
Lead	MW-2A	11/22/2002	5.56
Lead	MW-2A	12/1/1994	7 U
Lead	MW-2A	2/14/2001	2.6 U
Lead	MW-2A	3/29/1996	2 U
Lead	MW-2A	6/2/1997	4.7
Lead	MW-2A	7/18/1996	2.1 J
Lead	MW-2A	8/17/1994	11.2 U
Magnesium	MW-2A	12/1/1994	1550 B
Magnesium	MW-2A	3/29/1996	1520 B
Magnesium	MW-2A	7/18/1996	1900 B
Magnesium	MW-2A	8/17/1994	3650 B
Manganese	MW-2A	12/1/1994	31.3
Manganese	MW-2A	3/29/1996	31.8
Manganese	MW-2A	7/18/1996	14.2 U
Manganese	MW-2A	8/17/1994	129
Mercury	MW-2A	1/18/2002	0.5 U
Mercury	MW-2A	11/22/2002	0.5 U
Mercury	MW-2A	12/1/1994	0.24 U
Mercury	MW-2A	2/14/2001	0.1 U
Mercury	MW-2A	3/29/1996	0.15 U
Mercury	MW-2A	6/2/1997	0.06 U
Mercury	MW-2A	7/18/1996	0.08 U
Mercury	MW-2A	8/17/1994	0.24 U
Nickel	MW-2A	12/1/1994	12 U
Nickel	MW-2A	3/29/1996	5.1 B
Nickel	MW-2A	7/18/1996	6 B
Nickel	MW-2A	8/17/1994	14.6 B
Potassium	MW-2A	12/1/1994	2650 B
Potassium	MW-2A	3/29/1996	2420 B
Potassium	MW-2A	7/18/1996	823 B

Chemical	Sample Identification	Date Collected	Result
Potassium	MW-2A	8/17/1994	5200
Selenium	MW-2A	12/1/1994	1 U
Selenium	MW-2A	3/29/1996	3 U
Selenium	MW-2A	7/18/1996	3 U
Selenium	MW-2A	8/17/1994	2 U
Silver	MW-2A	1/18/2002	2 U
Silver	MW-2A	11/22/2002	2 U
Silver	MW-2A	12/1/1994	2 U
Silver	MW-2A	2/14/2001	2.3 U
Silver	MW-2A	3/29/1996	1 U
Silver	MW-2A	6/2/1997	2 U
Silver	MW-2A	7/18/1996	1.2 B
Silver	MW-2A	8/17/1994	2.5 J
Sodium	MW-2A	12/1/1994	5510
Sodium	MW-2A	3/29/1996	20600
Sodium	MW-2A	7/18/1996	4780 B
Sodium	MW-2A	8/17/1994	8010
Thallium	MW-2A	12/1/1994	1 U
Thallium	MW-2A	3/29/1996	5 U
Thallium	MW-2A	7/18/1996	6 U
Thallium	MW-2A	8/17/1994	3 U
Vanadium	MW-2A	12/1/1994	4.9 B
Vanadium	MW-2A	3/29/1996	1.3 B
Vanadium	MW-2A	7/18/1996	1 U
Vanadium	MW-2A	8/17/1994	3 U
Zinc	MW-2A	12/1/1994	251
Zinc	MW-2A	3/29/1996	149
Zinc	MW-2A	7/18/1996	144
Zinc	MW-2A	8/17/1994	56.6
Aluminum	MW-3	11/30/1994	298
Aluminum	MW-3	4/3/1996	95.5 B
Aluminum	MW-3	7/18/1996	242 U
Aluminum	MW-3	8/16/1994	734
Antimony	MW-3	11/30/1994	12 U
Antimony	MW-3	4/3/1996	3 U
Antimony	MW-3	7/18/1996	6 U
Antimony	MW-3	8/16/1994	12 U
Arsenic	MW-3	11/30/1994	4 U
Arsenic	MW-3	4/3/1996	2 U
Arsenic	MW-3	7/18/1996	5 U
Arsenic	MW-3	8/16/1994	3 U
Barium	MW-3	11/30/1994	27.2 B
Barium	MW-3	4/3/1996	26.1 B
Barium	MW-3	7/18/1996	21.3 B
Barium	MW-3	8/16/1994	23.6 B
Beryllium	MW-3	11/30/1994	1 U
Beryllium	MW-3	4/3/1996	1 U
Beryllium	MW-3	7/18/1996	1 U
Beryllium	MW-3	8/16/1994	1 U

Chemical	Sample Identification	Date Collected	Result
Cadmium	MW-3	1/21/2002	11
Cadmium	MW-3	11/21/2002	13.5
Cadmium	MW-3	11/30/1994	17.3
Cadmium	MW-3	2/13/2001	13.8
Cadmium	MW-3	4/3/1996	15.9
Cadmium	MW-3	6/2/1997	18.4
Cadmium	MW-3	7/18/1996	13.4
Cadmium	MW-3	8/16/1994	11.2 J
Calcium	MW-3	11/30/1994	10500
Calcium	MW-3	4/3/1996	9520
Calcium	MW-3	7/18/1996	6750
Calcium	MW-3	8/16/1994	8720
Chromium	MW-3	1/21/2002	8 U
Chromium	MW-3	11/21/2002	19
Chromium	MW-3	11/30/1994	2.1 B
Chromium	MW-3	2/13/2001	7.2 B
Chromium	MW-3	4/3/1996	3.5 U
Chromium	MW-3	6/2/1997	3.5 B
Chromium	MW-3	7/18/1996	4 U
Chromium	MW-3	8/16/1994	6.4 U
Cobalt	MW-3	11/30/1994	2 U
Cobalt	MW-3	4/3/1996	4 U
Cobalt	MW-3	7/18/1996	1 U
Cobalt	MW-3	8/16/1994	2 U
Copper	MW-3	11/30/1994	8.8 B
Copper	MW-3	4/3/1996	8.4 U
Copper	MW-3	7/18/1996	2.9 B
Copper	MW-3	8/16/1994	19.4 B
Cyanide	MW-3	11/30/1994	10 U
Cyanide	MW-3	4/3/1996	2 U
Cyanide	MW-3	8/16/1994	10 U
Iron	MW-3	11/30/1994	1900
Iron	MW-3	4/3/1996	285
Iron	MW-3	7/18/1996	199 U
Iron	MW-3	8/16/1994	5070
Lead	MW-3	1/21/2002	8.1
Lead	MW-3	11/21/2002	2.19
Lead	MW-3	11/30/1994	20.4
Lead	MW-3	2/13/2001	16.1
Lead	MW-3	4/3/1996	5.3
Lead	MW-3	6/2/1997	9
Lead	MW-3	7/18/1996	3.8 J
Lead	MW-3	8/16/1994	26.3 U
Magnesium	MW-3	11/30/1994	4080 B
Magnesium	MW-3	4/3/1996	4610 B
Magnesium	MW-3	7/18/1996	3560 B
Magnesium	MW-3	8/16/1994	3240 B
Manganese	MW-3	11/30/1994	172 U
Manganese	MW-3	4/3/1996	16.3

Chemical	Sample Identification	Date Collected	Result
Manganese	MW-3	7/18/1996	5.7 B
Manganese	MW-3	8/16/1994	63.7
Mercury	MW-3	1/21/2002	0.5 U
Mercury	MW-3	11/21/2002	0.5 U
Mercury	MW-3	11/30/1994	0.24 U
Mercury	MW-3	2/13/2001	0.1 U
Mercury	MW-3	4/3/1996	0.15 U
Mercury	MW-3	6/2/1997	0.06 U
Mercury	MW-3	7/18/1996	0.08 U
Mercury	MW-3	8/16/1994	0.24 U
Nickel	MW-3	11/30/1994	12 U
Nickel	MW-3	4/3/1996	2.9 U
Nickel	MW-3	7/18/1996	3 U
Nickel	MW-3	8/16/1994	12 U
Potassium	MW-3	11/30/1994	1710 U
Potassium	MW-3	4/3/1996	1100 B
Potassium	MW-3	7/18/1996	1610 B
Potassium	MW-3	8/16/1994	2500 B
Selenium	MW-3	11/30/1994	1 U
Selenium	MW-3	4/3/1996	2 U
Selenium	MW-3	7/18/1996	3 U
Selenium	MW-3	8/16/1994	2 U
Silver	MW-3	1/21/2002	2 U
Silver	MW-3	11/21/2002	2 U
Silver	MW-3	11/30/1994	2 U
Silver	MW-3	2/13/2001	2.5 U
Silver	MW-3	4/3/1996	1 U
Silver	MW-3	6/2/1997	2 U
Silver	MW-3	7/18/1996	1 U
Silver	MW-3	8/16/1994	2 J
Sodium	MW-3	11/30/1994	14400
Sodium	MW-3	4/3/1996	8990
Sodium	MW-3	7/18/1996	9120
Sodium	MW-3	8/16/1994	12800
Thallium	MW-3	11/30/1994	1 U
Thallium	MW-3	4/3/1996	3.4 U
Thallium	MW-3	7/18/1996	6 U
Thallium	MW-3	8/16/1994	3 U
Vanadium	MW-3	11/30/1994	3 U
Vanadium	MW-3	4/3/1996	1 U
Vanadium	MW-3	7/18/1996	1 U
Vanadium	MW-3	8/16/1994	6.4 B
Zinc	MW-3	11/30/1994	34.9
Zinc	MW-3	4/3/1996	19.9 U
Zinc	MW-3	7/18/1996	19.6 U
Zinc	MW-3	8/16/1994	42.8
Aluminum	MW-4	11/30/1994	109 B
Aluminum	MW-4	3/29/1996	409 U
Aluminum	MW-4	7/17/1996	220 U

Chemical	Sample Identification	Date Collected	Result
Aluminum	MW-4	8/17/1994	72.3 U
Antimony	MW-4	11/30/1994	15.5 B
Antimony	MW-4	3/29/1996	3 U
Antimony	MW-4	7/17/1996	6 U
Antimony	MW-4	8/17/1994	12 U
Arsenic	MW-4	11/30/1994	4 U
Arsenic	MW-4	3/29/1996	5 U
Arsenic	MW-4	7/17/1996	5 U
Arsenic	MW-4	8/17/1994	3 U
Barium	MW-4	11/30/1994	87.7 B
Barium	MW-4	3/29/1996	81 B
Barium	MW-4	7/17/1996	79.9 B
Barium	MW-4	8/17/1994	67.7 B
Beryllium	MW-4	11/30/1994	1 U
Beryllium	MW-4	3/29/1996	1 U
Beryllium	MW-4	7/17/1996	1 U
Beryllium	MW-4	8/17/1994	1 U
Cadmium	MW-4	1/18/2002	2.6
Cadmium	MW-4	11/21/2002	12.3
Cadmium	MW-4	11/30/1994	12.8
Cadmium	MW-4	2/14/2001	16.4
Cadmium	MW-4	3/29/1996	2.6 B
Cadmium	MW-4	6/2/1997	36.8
Cadmium	MW-4	7/17/1996	10.9
Cadmium	MW-4	8/17/1994	30.8
Calcium	MW-4	11/30/1994	13000
Calcium	MW-4	3/29/1996	29000
Calcium	MW-4	7/17/1996	11500
Calcium	MW-4	8/17/1994	16300
Chromium	MW-4	1/18/2002	20
Chromium	MW-4	11/21/2002	20.9
Chromium	MW-4	11/30/1994	2.4 B
Chromium	MW-4	2/14/2001	3.2 B
Chromium	MW-4	3/29/1996	2.4 U
Chromium	MW-4	6/2/1997	1.8 U
Chromium	MW-4	7/17/1996	4 U
Chromium	MW-4	8/17/1994	2 U
Cobalt	MW-4	11/30/1994	2 U
Cobalt	MW-4	3/29/1996	1 U
Cobalt	MW-4	7/17/1996	1 U
Cobalt	MW-4	8/17/1994	2 U
Copper	MW-4	11/30/1994	2 U
Copper	MW-4	3/29/1996	9.7 B
Copper	MW-4	7/17/1996	1 U
Copper	MW-4	8/17/1994	2 U
Cyanide	MW-4	11/30/1994	10 U
Cyanide	MW-4	3/29/1996	2 U
Cyanide	MW-4	8/17/1994	10 U
Iron	MW-4	11/30/1994	258

Chemical	Sample Identification	Date Collected	Result
Iron	MW-4	3/29/1996	610
Iron	MW-4	7/17/1996	161
Iron	MW-4	8/17/1994	79 B
Lead	MW-4	1/18/2002	4.3
Lead	MW-4	11/21/2002	2 U
Lead	MW-4	11/30/1994	4.3
Lead	MW-4	2/14/2001	2.6 U
Lead	MW-4	3/29/1996	2 U
Lead	MW-4	6/2/1997	2.5 B
Lead	MW-4	7/17/1996	2 U
Lead	MW-4	8/17/1994	1.6 U
Magnesium	MW-4	11/30/1994	6460
Magnesium	MW-4	3/29/1996	6170
Magnesium	MW-4	7/17/1996	6220
Magnesium	MW-4	8/17/1994	4930 B
Manganese	MW-4	11/30/1994	355
Manganese	MW-4	3/29/1996	264
Manganese	MW-4	7/17/1996	258
Manganese	MW-4	8/17/1994	333
Mercury	MW-4	1/18/2002	0.5 U
Mercury	MW-4	11/21/2002	0.5 U
Mercury	MW-4	11/30/1994	0.24 U
Mercury	MW-4	2/14/2001	0.1 U
Mercury	MW-4	3/29/1996	0.15 U
Mercury	MW-4	6/2/1997	0.06 U
Mercury	MW-4	7/17/1996	0.08 U
Mercury	MW-4	8/17/1994	0.24 U
Nickel	MW-4	11/30/1994	12 U
Nickel	MW-4	3/29/1996	9.2 B
Nickel	MW-4	7/17/1996	3 U
Nickel	MW-4	8/17/1994	14.1 B
Potassium	MW-4	11/30/1994	5010
Potassium	MW-4	3/29/1996	12700
Potassium	MW-4	7/17/1996	4370 B
Potassium	MW-4	8/17/1994	4210 B
Selenium	MW-4	11/30/1994	1 U
Selenium	MW-4	3/29/1996	3 U
Selenium	MW-4	7/17/1996	3 U
Selenium	MW-4	8/17/1994	2 U
Silver	MW-4	1/18/2002	2 U
Silver	MW-4	11/21/2002	2 U
Silver	MW-4	11/30/1994	2 U
Silver	MW-4	2/14/2001	2.3 U
Silver	MW-4	3/29/1996	1 B
Silver	MW-4	6/2/1997	2 U
Silver	MW-4	7/17/1996	1 U
Silver	MW-4	8/17/1994	2 UJ
Sodium	MW-4	11/30/1994	20900
Sodium	MW-4	3/29/1996	31100

Chemical	Sample Identification	Date Collected	Result
Sodium	MW-4	7/17/1996	17400
Sodium	MW-4	8/17/1994	16700
Thallium	MW-4	11/30/1994	1 U
Thallium	MW-4	3/29/1996	5 U
Thallium	MW-4	7/17/1996	6 U
Thallium	MW-4	8/17/1994	3 U
Vanadium	MW-4	11/30/1994	3 U
Vanadium	MW-4	3/29/1996	1.5 B
Vanadium	MW-4	7/17/1996	1 U
Vanadium	MW-4	8/17/1994	3 U
Zinc	MW-4	11/30/1994	31.9
Zinc	MW-4	3/29/1996	423
Zinc	MW-4	7/17/1996	44.4
Zinc	MW-4	8/17/1994	39.6
Aluminum	MW-6	12/1/1994	972
Aluminum	MW-6	3/28/1996	246 U
Aluminum	MW-6	7/17/1996	218
Aluminum	MW-6	8/18/1994	602
Antimony	MW-6	12/1/1994	16.1 B
Antimony	MW-6	3/28/1996	3 U
Antimony	MW-6	7/17/1996	6 U
Antimony	MW-6	8/18/1994	12 U
Arsenic	MW-6	12/1/1994	4 U
Arsenic	MW-6	3/28/1996	5 U
Arsenic	MW-6	7/17/1996	5 U
Arsenic	MW-6	8/18/1994	3 U
Barium	MW-6	12/1/1994	44.9 B
Barium	MW-6	3/28/1996	38.1 B
Barium	MW-6	7/17/1996	24.6 B
Barium	MW-6	8/18/1994	60.6 B
Beryllium	MW-6	12/1/1994	1.1 B
Beryllium	MW-6	3/28/1996	1 U
Beryllium	MW-6	7/17/1996	1 U
Beryllium	MW-6	8/18/1994	1 U
Cadmium	MW-6	1/29/2002	36
Cadmium	MW-6	11/25/2002	7.67
Cadmium	MW-6	12/1/1994	165
Cadmium	MW-6	2/14/2001	80
Cadmium	MW-6	3/28/1996	33.9
Cadmium	MW-6	6/2/1997	163
Cadmium	MW-6	7/17/1996	192
Cadmium	MW-6	8/18/1994	269
Calcium	MW-6	12/1/1994	14200
Calcium	MW-6	3/28/1996	12500
Calcium	MW-6	7/17/1996	14700
Calcium	MW-6	8/18/1994	18100
Chromium	MW-6	1/29/2002	8 U
Chromium	MW-6	11/25/2002	10.4
Chromium	MW-6	12/1/1994	5 B

Chemical	Sample Identification	Date Collected	Result
Chromium	MW-6	2/14/2001	2.1 U
Chromium	MW-6	3/28/1996	1 U
Chromium	MW-6	6/2/1997	2.4 B
Chromium	MW-6	7/17/1996	4 U
Chromium	MW-6	8/18/1994	2.3 B
Cobalt	MW-6	12/1/1994	2.9 B
Cobalt	MW-6	3/28/1996	1 U
Cobalt	MW-6	7/17/1996	1.3 B
Cobalt	MW-6	8/18/1994	6.5 B
Copper	MW-6	12/1/1994	13 B
Copper	MW-6	3/28/1996	9 U
Copper	MW-6	7/17/1996	1.2 B
Copper	MW-6	8/18/1994	3.9 U
Cyanide	MW-6	12/1/1994	10 U
Cyanide	MW-6	3/28/1996	2 U
Cyanide	MW-6	8/18/1994	10 U
Iron	MW-6	12/1/1994	4190
Iron	MW-6	3/28/1996	295
Iron	MW-6	7/17/1996	141
Iron	MW-6	8/18/1994	2000
Lead	MW-6	1/29/2002	2 U
Lead	MW-6	11/25/2002	2 U
Lead	MW-6	12/1/1994	11.3
Lead	MW-6	2/14/2001	2.6 U
Lead	MW-6	3/28/1996	2 U
Lead	MW-6	6/2/1997	2.2 B
Lead	MW-6	7/17/1996	2 U
Lead	MW-6	8/18/1994	3.2 U
Magnesium	MW-6	12/1/1994	5970
Magnesium	MW-6	3/28/1996	7240
Magnesium	MW-6	7/17/1996	5990
Magnesium	MW-6	8/18/1994	6060
Manganese	MW-6	12/1/1994	73.7
Manganese	MW-6	3/28/1996	27.1
Manganese	MW-6	7/17/1996	28.2
Manganese	MW-6	8/18/1994	103
Mercury	MW-6	1/29/2002	0.5 U
Mercury	MW-6	11/25/2002	0.5 U
Mercury	MW-6	12/1/1994	0.24 U
Mercury	MW-6	2/14/2001	0.1 U
Mercury	MW-6	3/28/1996	0.15 U
Mercury	MW-6	6/2/1997	0.06 U
Mercury	MW-6	7/17/1996	0.08 U
Mercury	MW-6	8/18/1994	0.24 U
Nickel	MW-6	12/1/1994	12 U
Nickel	MW-6	3/28/1996	5.4 B
Nickel	MW-6	7/17/1996	6.7 B
Nickel	MW-6	8/18/1994	12 U
Potassium	MW-6	12/1/1994	4110 B

Chemical	Sample Identification	Date Collected	Result
Potassium	MW-6	3/28/1996	2120 B
Potassium	MW-6	7/17/1996	2590 B
Potassium	MW-6	8/18/1994	1710 U
Selenium	MW-6	12/1/1994	1 U
Selenium	MW-6	3/28/1996	3 U
Selenium	MW-6	7/17/1996	3 U
Selenium	MW-6	8/18/1994	2 U
Silver	MW-6	1/29/2002	2 U
Silver	MW-6	11/25/2002	2 U
Silver	MW-6	12/1/1994	2.6 B
Silver	MW-6	2/14/2001	2.4 B
Silver	MW-6	3/28/1996	1 U
Silver	MW-6	6/2/1997	2 U
Silver	MW-6	7/17/1996	1 U
Silver	MW-6	8/18/1994	2 UJ
Sodium	MW-6	12/1/1994	1100
Sodium	MW-6	3/28/1996	20500
Sodium	MW-6	7/17/1996	16300
Sodium	MW-6	8/18/1994	23400
Thallium	MW-6	12/1/1994	1 U
Thallium	MW-6	3/28/1996	5 U
Thallium	MW-6	7/17/1996	6 U
Thallium	MW-6	8/18/1994	3 U
Vanadium	MW-6	12/1/1994	6.9 B
Vanadium	MW-6	3/28/1996	1 U
Vanadium	MW-6	7/17/1996	1 U
Vanadium	MW-6	8/18/1994	3.1 B
Zinc	MW-6	12/1/1994	81.2
Zinc	MW-6	3/28/1996	35.5 U
Zinc	MW-6	7/17/1996	35.4
Zinc	MW-6	8/18/1994	55.8
Aluminum	MW-7D	7/16/1996	806
Antimony	MW-7D	7/16/1996	6 U
Arsenic	MW-7D	7/16/1996	5 U
Barium	MW-7D	7/16/1996	41.9 B
Beryllium	MW-7D	7/16/1996	1 U
Cadmium	MW-7D	1/29/2002	1.2
Cadmium	MW-7D	12/4/2002	1 U
Cadmium	MW-7D	2/15/2001	2.4 B
Cadmium	MW-7D	6/2/1997	0.5 U
Cadmium	MW-7D	7/16/1996	1 U
Calcium	MW-7D	7/16/1996	16900
Chromium	MW-7D	1/29/2002	8 U
Chromium	MW-7D	12/4/2002	20.3
Chromium	MW-7D	2/15/2001	2.2 B
Chromium	MW-7D	6/2/1997	2.1 B
Chromium	MW-7D	7/16/1996	4 U
Cobalt	MW-7D	7/16/1996	2.2 B
Copper	MW-7D	7/16/1996	2.3 B

Chemical	Sample Identification	Date Collected	Result
Iron	MW-7D	7/16/1996	1050
Lead	MW-7D	1/29/2002	2 U
Lead	MW-7D	12/4/2002	2 U
Lead	MW-7D	2/15/2001	2.6 U
Lead	MW-7D	6/2/1997	1.6 U
Lead	MW-7D	7/16/1996	3.4 J
Magnesium	MW-7D	7/16/1996	6590
Manganese	MW-7D	7/16/1996	462
Mercury	MW-7D	1/29/2002	0.5 U
Mercury	MW-7D	12/4/2002	0.5 U
Mercury	MW-7D	2/15/2001	0.1 U
Mercury	MW-7D	6/2/1997	0.1 B
Mercury	MW-7D	7/16/1996	0.08 U
Nickel	MW-7D	7/16/1996	21.6 B
Potassium	MW-7D	7/16/1996	3670 B
Selenium	MW-7D	7/16/1996	3 U
Silver	MW-7D	1/29/2002	2 U
Silver	MW-7D	12/4/2002	2 U
Silver	MW-7D	2/15/2001	2.3 U
Silver	MW-7D	6/2/1997	2 U
Silver	MW-7D	7/16/1996	1 U
Sodium	MW-7D	7/16/1996	11300
Thallium	MW-7D	7/16/1996	6 U
Vanadium	MW-7D	7/16/1996	1.4 B
Zinc	MW-7D	7/16/1996	25 U
Aluminum	MW-7S	7/16/1996	321 U
Antimony	MW-7S	7/16/1996	6 U
Arsenic	MW-7S	7/16/1996	5 U
Barium	MW-7S	7/16/1996	39.2 B
Beryllium	MW-7S	7/16/1996	1 U
Cadmium	MW-7S	1/21/2002	30
Cadmium	MW-7S	12/4/2002	2.92
Cadmium	MW-7S	2/5/2001	36.2
Cadmium	MW-7S	6/2/1997	2.9 B
Cadmium	MW-7S	7/16/1996	1 U
Calcium	MW-7S	7/16/1996	13700
Chromium	MW-7S	1/21/2002	15
Chromium	MW-7S	12/4/2002	18.4
Chromium	MW-7S	2/5/2001	5.2 B
Chromium	MW-7S	6/2/1997	8.3 B
Chromium	MW-7S	7/16/1996	10.1
Cobalt	MW-7S	7/16/1996	2.4 B
Copper	MW-7S	7/16/1996	1 U
Iron	MW-7S	7/16/1996	428
Lead	MW-7S	1/21/2002	2.1
Lead	MW-7S	12/4/2002	2 U
Lead	MW-7S	2/5/2001	2.6 U
Lead	MW-7S	6/2/1997	3 B
Lead	MW-7S	7/16/1996	2 U

Chemical	Sample Identification	Date Collected	Result
Magnesium	MW-7S	7/16/1996	7540
Manganese	MW-7S	7/16/1996	253
Mercury	MW-7S	1/21/2002	0.5 U
Mercury	MW-7S	12/4/2002	0.5 U
Mercury	MW-7S	2/5/2001	0.1 U
Mercury	MW-7S	6/2/1997	0.06 U
Mercury	MW-7S	7/16/1996	0.08 U
Nickel	MW-7S	7/16/1996	14.7 B
Potassium	MW-7S	7/16/1996	1990 B
Selenium	MW-7S	7/16/1996	3 U
Silver	MW-7S	1/21/2002	2 U
Silver	MW-7S	12/4/2002	2 U
Silver	MW-7S	2/5/2001	2.3 U
Silver	MW-7S	6/2/1997	2 U
Silver	MW-7S	7/16/1996	1 U
Sodium	MW-7S	7/16/1996	12800
Thallium	MW-7S	7/16/1996	6 U
Vanadium	MW-7S	7/16/1996	1 U
Zinc	MW-7S	7/16/1996	36.5 U
Aluminum	MW-8S	9/12/1996	121 U
Antimony	MW-8S	9/12/1996	6 U
Arsenic	MW-8S	9/12/1996	5 U
Barium	MW-8S	9/12/1996	30.5 B
Beryllium	MW-8S	9/12/1996	1 U
Cadmium	MW-8S	1/18/2002	1 U
Cadmium	MW-8S	11/25/2002	1 U
Cadmium	MW-8S	2/14/2001	0.5 U
Cadmium	MW-8S	6/2/1997	0.5 U
Cadmium	MW-8S	9/12/1996	1 U
Calcium	MW-8S	9/12/1996	13200
Chromium	MW-8S	1/18/2002	8.6
Chromium	MW-8S	11/25/2002	11.1
Chromium	MW-8S	2/14/2001	2.9 B
Chromium	MW-8S	6/2/1997	5.2 B
Chromium	MW-8S	9/12/1996	4 U
Cobalt	MW-8S	9/12/1996	1.6 B
Copper	MW-8S	9/12/1996	5.6 U
Cyanide	MW-8S	9/12/1996	4 U
Iron	MW-8S	9/12/1996	321
Lead	MW-8S	1/18/2002	3.7
Lead	MW-8S	11/25/2002	2 U
Lead	MW-8S	2/14/2001	2.6 U
Lead	MW-8S	6/2/1997	4
Lead	MW-8S	9/12/1996	2 U
Magnesium	MW-8S	9/12/1996	3790
Manganese	MW-8S	9/12/1996	500
Mercury	MW-8S	1/18/2002	0.5 U
Mercury	MW-8S	11/25/2002	0.5 U
Mercury	MW-8S	2/14/2001	0.1 U

Chemical	Sample Identification	Date Collected	Result
Mercury	MW-8S	6/2/1997	0.06 U
Mercury	MW-8S	9/12/1996	0.05 U
Nickel	MW-8S	9/12/1996	8.9 B
Potassium	MW-8S	9/12/1996	1150 B
Selenium	MW-8S	9/12/1996	3 UJ
Silver	MW-8S	1/18/2002	2 U
Silver	MW-8S	11/25/2002	2 U
Silver	MW-8S	2/14/2001	2.3 U
Silver	MW-8S	6/2/1997	2 U
Silver	MW-8S	9/12/1996	1 U
Sodium	MW-8S	9/12/1996	12900 J
Thallium	MW-8S	9/12/1996	6 U
Vanadium	MW-8S	9/12/1996	1 U
Zinc	MW-8S	9/12/1996	46.5
Aluminum	MW-9	12/1/1994	3120
Aluminum	MW-9	3/28/1996	1170
Aluminum	MW-9	8/18/1994	140 B
Antimony	MW-9	12/1/1994	12.9 B
Antimony	MW-9	3/28/1996	3 U
Antimony	MW-9	8/18/1994	12 U
Arsenic	MW-9	12/1/1994	4 U
Arsenic	MW-9	3/28/1996	5 U
Arsenic	MW-9	8/18/1994	3 U
Barium	MW-9	12/1/1994	83.4 B
Barium	MW-9	3/28/1996	65.9 B
Barium	MW-9	8/18/1994	58.9 B
Beryllium	MW-9	12/1/1994	1.4 B
Beryllium	MW-9	3/28/1996	1 U
Beryllium	MW-9	8/18/1994	1 U
Cadmium	MW-9	1/22/2002	11
Cadmium	MW-9	11/25/2002	10
Cadmium	MW-9	12/1/1994	36.4
Cadmium	MW-9	2/13/2001	14.5
Cadmium	MW-9	3/28/1996	17.3
Cadmium	MW-9	6/2/1997	16.5
Cadmium	MW-9	8/18/1994	57.8
Calcium	MW-9	12/1/1994	10300
Calcium	MW-9	3/28/1996	8030
Calcium	MW-9	8/18/1994	11800
Chromium	MW-9	1/22/2002	8 U
Chromium	MW-9	11/25/2002	17.5
Chromium	MW-9	12/1/1994	14.7
Chromium	MW-9	2/13/2001	13.7
Chromium	MW-9	3/28/1996	9.3 B
Chromium	MW-9	6/2/1997	7.5 B
Chromium	MW-9	8/18/1994	2 U
Cobalt	MW-9	12/1/1994	7.4 B
Cobalt	MW-9	3/28/1996	25.5 B
Cobalt	MW-9	8/18/1994	2 U

Chemical	Sample Identification	Date Collected	Result
Copper	MW-9	12/1/1994	17.9 B
Copper	MW-9	3/28/1996	10.1 B
Copper	MW-9	8/18/1994	3.8 U
Cyanide	MW-9	12/1/1994	10 U
Cyanide	MW-9	3/28/1996	2 U
Cyanide	MW-9	8/18/1994	10 U
Iron	MW-9	12/1/1994	9370
Iron	MW-9	3/28/1996	3070
Iron	MW-9	8/18/1994	291
Lead	MW-9	1/22/2002	2 U
Lead	MW-9	11/25/2002	2 U
Lead	MW-9	12/1/1994	17
Lead	MW-9	2/13/2001	2.6 U
Lead	MW-9	3/28/1996	10
Lead	MW-9	6/2/1997	1.8 B
Lead	MW-9	8/18/1994	5 U
Magnesium	MW-9	12/1/1994	4790 B
Magnesium	MW-9	3/28/1996	3980 B
Magnesium	MW-9	8/18/1994	4580 B
Manganese	MW-9	12/1/1994	1040
Manganese	MW-9	3/28/1996	447
Manganese	MW-9	8/18/1994	203
Mercury	MW-9	1/22/2002	0.5 U
Mercury	MW-9	11/25/2002	0.5 U
Mercury	MW-9	12/1/1994	0.24 U
Mercury	MW-9	2/13/2001	0.1 U
Mercury	MW-9	3/28/1996	0.15 U
Mercury	MW-9	6/2/1997	0.06 U
Mercury	MW-9	8/18/1994	0.24 U
Nickel	MW-9	12/1/1994	12 U
Nickel	MW-9	3/28/1996	7.3 B
Nickel	MW-9	8/18/1994	12 U
Potassium	MW-9	12/1/1994	1710 U
Potassium	MW-9	3/28/1996	1600 B
Potassium	MW-9	8/18/1994	1710 U
Selenium	MW-9	12/1/1994	1 U
Selenium	MW-9	3/28/1996	3 U
Selenium	MW-9	8/18/1994	2 U
Silver	MW-9	1/22/2002	2 U
Silver	MW-9	11/25/2002	2 U
Silver	MW-9	12/1/1994	2 UJ
Silver	MW-9	2/13/2001	2.5 B
Silver	MW-9	3/28/1996	1.3 B
Silver	MW-9	6/2/1997	2.7 B
Silver	MW-9	8/18/1994	2 UJ
Sodium	MW-9	12/1/1994	12400
Sodium	MW-9	3/28/1996	10700
Sodium	MW-9	8/18/1994	14200
Thallium	MW-9	12/1/1994	1 U

Chemical	Sample Identification	Date Collected	Result
Thallium	MW-9	3/28/1996	5 U
Thallium	MW-9	8/18/1994	3 U
Vanadium	MW-9	12/1/1994	11.3 B
Vanadium	MW-9	3/28/1996	3.5 B
Vanadium	MW-9	8/18/1994	3 U
Zinc	MW-9	12/1/1994	69.9
Zinc	MW-9	3/28/1996	36.6 U
Zinc	MW-9	8/18/1994	24.4
Aluminum	MW-9R	12/1/1994	3120
Antimony	MW-9R	12/1/1994	20.3 B
Arsenic	MW-9R	12/1/1994	4 U
Barium	MW-9R	12/1/1994	89.1 B
Beryllium	MW-9R	12/1/1994	1.3 B
Cadmium	MW-9R	12/1/1994	39.4
Calcium	MW-9R	12/1/1994	11400
Chromium	MW-9R	12/1/1994	17.6
Cobalt	MW-9R	12/1/1994	9.3 B
Copper	MW-9R	12/1/1994	20.1 B
Cyanide	MW-9R	12/1/1994	10 U
Iron	MW-9R	12/1/1994	9400
Lead	MW-9R	12/1/1994	12.6
Magnesium	MW-9R	12/1/1994	5270
Manganese	MW-9R	12/1/1994	1060
Mercury	MW-9R	12/1/1994	0.24 U
Nickel	MW-9R	12/1/1994	12 U
Potassium	MW-9R	12/1/1994	3940 B
Selenium	MW-9R	12/1/1994	1 U
Silver	MW-9R	12/1/1994	6.3 B
Sodium	MW-9R	12/1/1994	13800
Thallium	MW-9R	12/1/1994	1 U
Vanadium	MW-9R	12/1/1994	14.9 B
Zinc	MW-9R	12/1/1994	64.6
Cadmium	REP-1 (7S)	6/2/1997	0.5 U
Chromium	REP-1 (7S)	6/2/1997	3.8 B
Lead	REP-1 (7S)	6/2/1997	3.2
Mercury	REP-1 (7S)	6/2/1997	0.07 B
Silver	REP-1 (7S)	6/2/1997	2 U
Cadmium	TW-1	12/3/2002	3.65
Chromium	TW-1	12/3/2002	13.9
Lead	TW-1	12/3/2002	2 U
Mercury	TW-1	12/3/2002	0.5 U
Silver	TW-1	12/3/2002	2 U
Cadmium	TW-2	12/3/2002	24.1
Chromium	TW-2	12/3/2002	12
Lead	TW-2	12/3/2002	2 U
Mercury	TW-2	12/3/2002	0.5 U
Silver	TW-2	12/3/2002	2 U
Cadmium	TW-3	12/3/2002	1 U
Chromium	TW-3	12/3/2002	11.7

Chemical	Sample Identification	Date Collected	Result
Lead	TW-3	12/3/2002	2 U
Mercury	TW-3	12/3/2002	0.5 U
Silver	TW-3	12/3/2002	2 U
Background Soil Samples (ppm)			
Aluminum	H1	7/22/1996	791
Antimony	H1	7/22/1996	0.28 U
Arsenic	H1	7/22/1996	0.7 B
Barium	H1	7/22/1996	3.6 B
Beryllium	H1	7/22/1996	0.06 B
Cadmium	H1	7/22/1996	0.55
Calcium	H1	7/22/1996	465 B
Chromium	H1	7/22/1996	2.2
Cobalt	H1	7/22/1996	0.87 B
Copper	H1	7/22/1996	5.3
Iron	H1	7/22/1996	1710
Lead	H1	7/22/1996	3.8
Magnesium	H1	7/22/1996	253 B
Manganese	H1	7/22/1996	53.4
Mercury	H1	7/22/1996	0.05 U
Nickel	H1	7/22/1996	1.8 B
Potassium	H1	7/22/1996	59.6 B
Selenium	H1	7/22/1996	0.14 U
Silver	H1	7/22/1996	72.1
Sodium	H1	7/22/1996	20.6 B
Thallium	H1	7/22/1996	0.2 U
Vanadium	H1	7/22/1996	3.5 B
Zinc	H1	7/22/1996	15 E
Aluminum	H2	7/22/1996	5040
Antimony	H2	7/22/1996	0.31 U
Arsenic	H2	7/22/1996	2
Barium	H2	7/22/1996	7.7 B
Beryllium	H2	7/22/1996	0.17 B
Cadmium	H2	7/22/1996	0.04 B
Calcium	H2	7/22/1996	113 B
Chromium	H2	7/22/1996	6
Cobalt	H2	7/22/1996	1 B
Copper	H2	7/22/1996	5.4
Iron	H2	7/22/1996	5790
Lead	H2	7/22/1996	26.4
Magnesium	H2	7/22/1996	435 B
Manganese	H2	7/22/1996	23.5
Mercury	H2	7/22/1996	0.06 B
Nickel	H2	7/22/1996	3.2 B
Potassium	H2	7/22/1996	211 B
Selenium	H2	7/22/1996	0.16 U
Silver	H2	7/22/1996	15.8
Sodium	H2	7/22/1996	26.4 B
Thallium	H2	7/22/1996	0.22 U
Vanadium	H2	7/22/1996	21.9

Chemical	Sample Identification	Date Collected	Result
Zinc	H2	7/22/1996	11.5 E
Aluminum	H3	7/22/1996	2140
Antimony	H3	7/22/1996	0.31 U
Arsenic	H3	7/22/1996	1.5
Barium	H3	7/22/1996	5.5 B
Beryllium	H3	7/22/1996	0.06 B
Cadmium	H3	7/22/1996	0.03 B
Calcium	H3	7/22/1996	70.7 B
Chromium	H3	7/22/1996	2.5
Cobalt	H3	7/22/1996	0.39 B
Copper	H3	7/22/1996	2.3 B
Iron	H3	7/22/1996	2950
Lead	H3	7/22/1996	11.7
Magnesium	H3	7/22/1996	147 B
Manganese	H3	7/22/1996	8.7
Mercury	H3	7/22/1996	0.05 U
Nickel	H3	7/22/1996	1.1 B
Potassium	H3	7/22/1996	89.7 B
Selenium	H3	7/22/1996	0.18 B
Silver	H3	7/22/1996	0.54 B
Sodium	H3	7/22/1996	20.9 B
Thallium	H3	7/22/1996	0.22 U
Vanadium	H3	7/22/1996	10.2
Zinc	H3	7/22/1996	6.1 E
Aluminum	K1	7/22/1996	7990
Antimony	K1	7/22/1996	0.35 B
Arsenic	K1	7/22/1996	3.3
Barium	K1	7/22/1996	16.1 B
Beryllium	K1	7/22/1996	0.23 B
Cadmium	K1	7/22/1996	0.38 B
Calcium	K1	7/22/1996	428 B
Chromium	K1	7/22/1996	9.1
Cobalt	K1	7/22/1996	1.7 B
Copper	K1	7/22/1996	15.6
Iron	K1	7/22/1996	9510
Lead	K1	7/22/1996	21.6
Magnesium	K1	7/22/1996	789
Manganese	K1	7/22/1996	36.4
Mercury	K1	7/22/1996	0.06 B
Nickel	K1	7/22/1996	5.6
Potassium	K1	7/22/1996	183 B
Selenium	K1	7/22/1996	0.45 B
Silver	K1	7/22/1996	0.7 B
Sodium	K1	7/22/1996	39 B
Thallium	K1	7/22/1996	0.24 U
Vanadium	K1	7/22/1996	20.5
Zinc	K1	7/22/1996	22.8 E
Aluminum	K2	7/22/1996	7070
Antimony	K2	7/22/1996	0.32 U

Chemical	Sample Identification	Date Collected	Result
Arsenic	K2	7/22/1996	3.7
Barium	K2	7/22/1996	19.4 B
Beryllium	K2	7/22/1996	0.25 B
Cadmium	K2	7/22/1996	0.08 B
Calcium	K2	7/22/1996	1220
Chromium	K2	7/22/1996	9.2
Cobalt	K2	7/22/1996	2.3 B
Copper	K2	7/22/1996	8.7
Iron	K2	7/22/1996	7560
Lead	K2	7/22/1996	17.6
Magnesium	K2	7/22/1996	936
Manganese	K2	7/22/1996	89.5
Mercury	K2	7/22/1996	0.07 B
Nickel	K2	7/22/1996	5.3
Potassium	K2	7/22/1996	349 B
Selenium	K2	7/22/1996	0.26 B
Silver	K2	7/22/1996	16.2
Sodium	K2	7/22/1996	32.4 B
Thallium	K2	7/22/1996	0.23 U
Vanadium	K2	7/22/1996	17.1
Zinc	K2	7/22/1996	25.6 E
Aluminum	M1	7/22/1996	5490
Antimony	M1	7/22/1996	0.31 U
Arsenic	M1	7/22/1996	3.3
Barium	M1	7/22/1996	15.2 B
Beryllium	M1	7/22/1996	0.24 B
Cadmium	M1	7/22/1996	0.12 B
Calcium	M1	7/22/1996	1000
Chromium	M1	7/22/1996	7.4
Cobalt	M1	7/22/1996	1.7 B
Copper	M1	7/22/1996	4.6
Iron	M1	7/22/1996	5540
Lead	M1	7/22/1996	11.6
Magnesium	M1	7/22/1996	814
Manganese	M1	7/22/1996	64.7
Mercury	M1	7/22/1996	0.05 U
Nickel	M1	7/22/1996	3.9 B
Potassium	M1	7/22/1996	205 B
Selenium	M1	7/22/1996	0.16 U
Silver	M1	7/22/1996	0.09 B
Sodium	M1	7/22/1996	32.3 B
Thallium	M1	7/22/1996	0.22 U
Vanadium	M1	7/22/1996	12.1
Zinc	M1	7/22/1996	18.1 E
Aluminum	M2	7/22/1996	4880
Antimony	M2	7/22/1996	0.34 B
Arsenic	M2	7/22/1996	1.6
Barium	M2	7/22/1996	11.9 B
Beryllium	M2	7/22/1996	0.19 B

Chemical	Sample Identification	Date Collected	Result
Cadmium	M2	7/22/1996	0.12 B
Calcium	M2	7/22/1996	1650
Chromium	M2	7/22/1996	9.2
Cobalt	M2	7/22/1996	1.1 B
Copper	M2	7/22/1996	4.2
Iron	M2	7/22/1996	5040
Lead	M2	7/22/1996	11
Magnesium	M2	7/22/1996	769
Manganese	M2	7/22/1996	48.7
Mercury	M2	7/22/1996	0.05 U
Nickel	M2	7/22/1996	3.3 B
Potassium	M2	7/22/1996	177 B
Selenium	M2	7/22/1996	0.26 B
Silver	M2	7/22/1996	0.32 B
Sodium	M2	7/22/1996	30.8 B
Thallium	M2	7/22/1996	0.21 U
Vanadium	M2	7/22/1996	12.4
Zinc	M2	7/22/1996	15.2 E
Aluminum	M3	7/22/1996	5870
Antimony	M3	7/22/1996	0.31 U
Arsenic	M3	7/22/1996	2
Barium	M3	7/22/1996	12.2 B
Beryllium	M3	7/22/1996	0.24 B
Cadmium	M3	7/22/1996	0.11 B
Calcium	M3	7/22/1996	631
Chromium	M3	7/22/1996	9.1
Cobalt	M3	7/22/1996	1.5 B
Copper	M3	7/22/1996	4.3
Iron	M3	7/22/1996	7120
Lead	M3	7/22/1996	11.2
Magnesium	M3	7/22/1996	652
Manganese	M3	7/22/1996	62
Mercury	M3	7/22/1996	0.05 U
Nickel	M3	7/22/1996	3.8 B
Potassium	M3	7/22/1996	269 B
Selenium	M3	7/22/1996	0.21 B
Silver	M3	7/22/1996	0.33 B
Sodium	M3	7/22/1996	59.1 B
Thallium	M3	7/22/1996	0.22 U
Vanadium	M3	7/22/1996	14.4
Zinc	M3	7/22/1996	15.4 E
Aluminum	SB-16	6/21/1994	8990
Antimony	SB-16	6/21/1994	3.1 U
Arsenic	SB-16	6/21/1994	1.6 B
Barium	SB-16	6/21/1994	26.1 B
Beryllium	SB-16	6/21/1994	0.32 B
Cadmium	SB-16	6/21/1994	0.7 U
Calcium	SB-16	6/21/1994	280 U
Chromium	SB-16	6/21/1994	8.5

Chemical	Sample Identification	Date Collected	Result
Cobalt	SB-16	6/21/1994	2.1 B
Copper	SB-16	6/21/1994	2.5 B
Cyanide	SB-16	6/21/1994	1.1 U
Iron	SB-16	6/21/1994	8410
Lead	SB-16	6/21/1994	3
Magnesium	SB-16	6/21/1994	960 B
Manganese	SB-16	6/21/1994	77
Mercury	SB-16	6/21/1994	0.1 U
Nickel	SB-16	6/21/1994	5.2 B
Potassium	SB-16	6/21/1994	509 B
Selenium	SB-16	6/21/1994	0.4 UJ
Silver	SB-16	6/21/1994	0.4 U
Sodium	SB-16	6/21/1994	22.8 U
Thallium	SB-16	6/21/1994	0.7 U
Vanadium	SB-16	6/21/1994	15
Zinc	SB-16	6/21/1994	13.2 U
On-Site Surface Soils (ppm)			
Aluminum	B-2	10/3/1994	1360
Antimony	B-2	10/3/1994	2.5 U
Arsenic	B-2	10/3/1994	0.42 U
Barium	B-2	10/3/1994	1240
Beryllium	B-2	10/3/1994	0.2 U
Cadmium	B-2	10/3/1994	1.6 U
Calcium	B-2	10/3/1994	507 B
Chromium	B-2	10/3/1994	3
Cobalt	B-2	10/3/1994	1.8 B
Copper	B-2	10/3/1994	18.4
Cyanide	B-2	10/3/1994	0.1 U
Iron	B-2	10/3/1994	1670
Lead	B-2	10/3/1994	5.3 J
Magnesium	B-2	10/3/1994	236 B
Manganese	B-2	10/3/1994	19.4
Mercury	B-2	10/3/1994	0.13 UJ
Nickel	B-2	10/3/1994	2.5 U
Potassium	B-2	10/3/1994	359 U
Selenium	B-2	10/3/1994	0.21 U
Silver	B-2	10/3/1994	158
Sodium	B-2	10/3/1994	31 U
Thallium	B-2	10/3/1994	0.21 U
Vanadium	B-2	10/3/1994	4.2 B
Zinc	B-2	10/3/1994	25.3 J
Cadmium	B-2-10D	9/5/1996	0.22 U
Silver	B-2-10D	9/5/1996	0.22 U
Cadmium	B-2-10S	9/5/1996	2.6
Silver	B-2-10S	9/5/1996	222
Cadmium	B-2-11D	9/5/1996	0.22 U
Silver	B-2-11D	9/5/1996	0.22 U
Cadmium	B-2-11S	9/5/1996	2.1
Silver	B-2-11S	9/5/1996	129

Chemical	Sample Identification	Date Collected	Result
Cadmium	B-2-12D	9/5/1996	0.22 U
Silver	B-2-12D	9/5/1996	0.22 U
Cadmium	B-2-12S	9/5/1996	1.8
Silver	B-2-12S	9/5/1996	93.3
Cadmium	B-2-13D	9/5/1996	0.22 U
Silver	B-2-13D	9/5/1996	0.22 U
Cadmium	B-2-13S	9/5/1996	2.2
Silver	B-2-13S	9/5/1996	448
Cadmium	B-2-14D	9/5/1996	1.2
Silver	B-2-14D	9/5/1996	375
Cadmium	B-2-14S	9/5/1996	1.5
Silver	B-2-14S	9/5/1996	90.8
Cadmium	B-2-15D	9/5/1996	0.22 U
Silver	B-2-15D	9/5/1996	0.22 U
Cadmium	B-2-15S	9/5/1996	0.52 B
Silver	B-2-15S	9/5/1996	398
Cadmium	B-2-16D	9/5/1996	0.22 U
Silver	B-2-16D	9/5/1996	9.5
Cadmium	B-2-16S	9/5/1996	3.5
Silver	B-2-16S	9/5/1996	223
Cadmium	B-2-17D	9/5/1996	0.22 U
Silver	B-2-17D	9/5/1996	0.22 U
Cadmium	B-2-17S	9/5/1996	4.3
Silver	B-2-17S	9/5/1996	239
Cadmium	B-2-18S	9/5/1996	2.4
Silver	B-2-18S	9/5/1996	29.9
Cadmium	B-2-1D	9/5/1996	0.39 B
Silver	B-2-1D	9/5/1996	2 B
Cadmium	B-2-1S	9/5/1996	3.1
Silver	B-2-1S	9/5/1996	43
Cadmium	B-2-2D	9/5/1996	0.23 U
Silver	B-2-2D	9/5/1996	0.23 B
Cadmium	B-2-2S	9/5/1996	6.9
Silver	B-2-2S	9/5/1996	236
Cadmium	B-2-3D	9/5/1996	0.23 U
Silver	B-2-3D	9/5/1996	0.23 U
Cadmium	B-2-3S	9/5/1996	2
Silver	B-2-3S	9/5/1996	27.3
Cadmium	B-2-4D	9/5/1996	0.23 U
Silver	B-2-4D	9/5/1996	0.23 U
Cadmium	B-2-4S	9/5/1996	5.5
Silver	B-2-4S	9/5/1996	154
Cadmium	B-2-5D	9/5/1996	0.22 U
Silver	B-2-5D	9/5/1996	1.9 B
Cadmium	B-2-5S	9/5/1996	105
Silver	B-2-5S	9/5/1996	288
Cadmium	B-2-6D	9/5/1996	24.2
Silver	B-2-6D	9/5/1996	247
Cadmium	B-2-6S	9/5/1996	3.3

Chemical	Sample Identification	Date Collected	Result
Silver	B-2-6S	9/5/1996	99
Cadmium	B-2-7D	9/5/1996	1.4
Silver	B-2-7D	9/5/1996	0.29 B
Cadmium	B-2-7S	9/5/1996	2.7
Silver	B-2-7S	9/5/1996	107
Cadmium	B-2-8D	9/5/1996	0.97 B
Silver	B-2-8D	9/5/1996	290
Cadmium	B-2-8S	9/5/1996	2.8
Silver	B-2-8S	9/5/1996	88.8
Cadmium	B-2-9D	9/5/1996	0.22 U
Silver	B-2-9D	9/5/1996	0.22 U
Cadmium	B-2-9S	9/5/1996	6.1
Silver	B-2-9S	9/5/1996	157
Aluminum	B-7	10/3/1994	5750
Antimony	B-7	10/3/1994	2.7 UJ
Arsenic	B-7	10/3/1994	1.6 B
Barium	B-7	10/3/1994	9.3 B
Beryllium	B-7	10/3/1994	0.2 U
Cadmium	B-7	10/3/1994	0.66 U
Calcium	B-7	10/3/1994	266 B
Chromium	B-7	10/3/1994	5.7
Cobalt	B-7	10/3/1994	0.94 B
Copper	B-7	10/3/1994	3.5 B
Cyanide	B-7	10/3/1994	0.11 U
Iron	B-7	10/3/1994	7660
Lead	B-7	10/3/1994	45.8
Magnesium	B-7	10/3/1994	451 B
Manganese	B-7	10/3/1994	26.2
Mercury	B-7	10/3/1994	0.13 UJ
Nickel	B-7	10/3/1994	2.7 U
Potassium	B-7	10/3/1994	378 U
Selenium	B-7	10/3/1994	0.4 B
Silver	B-7	10/3/1994	2.5
Sodium	B-7	10/3/1994	32.5 U
Thallium	B-7	10/3/1994	0.22 U
Vanadium	B-7	10/3/1994	13.4
Zinc	B-7	10/3/1994	14.4
Aluminum	SB-1	5/19/1994	10200
Antimony	SB-1	5/19/1994	5 J
Arsenic	SB-1	5/19/1994	2.4 J
Barium	SB-1	5/19/1994	20.8 B
Beryllium	SB-1	5/19/1994	0.35 B
Cadmium	SB-1	5/19/1994	0.85 B
Calcium	SB-1	5/19/1994	175 U
Chromium	SB-1	5/19/1994	9.8
Cobalt	SB-1	5/19/1994	2 B
Copper	SB-1	5/19/1994	6.4 U
Cyanide	SB-1	5/19/1994	0.11 U
Iron	SB-1	5/19/1994	10800

Chemical	Sample Identification	Date Collected	Result
Lead	SB-1	5/19/1994	17.6 J
Magnesium	SB-1	5/19/1994	760 B
Manganese	SB-1	5/19/1994	44.2 J
Mercury	SB-1	5/19/1994	0.16
Nickel	SB-1	5/19/1994	6.6 B
Potassium	SB-1	5/19/1994	434 U
Selenium	SB-1	5/19/1994	0.3 B
Silver	SB-1	5/19/1994	2.6
Sodium	SB-1	5/19/1994	37.1 U
Thallium	SB-1	5/19/1994	3 U
Vanadium	SB-1	5/19/1994	19.7
Zinc	SB-1	5/19/1994	19 U
Aluminum	SB-10	5/26/1994	779
Antimony	SB-10	5/26/1994	2.4 U
Arsenic	SB-10	5/26/1994	0.4 UJ
Barium	SB-10	5/26/1994	66.5
Beryllium	SB-10	5/26/1994	0.2 U
Cadmium	SB-10	5/26/1994	0.77 B
Calcium	SB-10	5/26/1994	120 U
Chromium	SB-10	5/26/1994	3.7
Cobalt	SB-10	5/26/1994	0.72 B
Copper	SB-10	5/26/1994	54.5 J
Cyanide	SB-10	5/26/1994	0.11 U
Iron	SB-10	5/26/1994	1430
Lead	SB-10	5/26/1994	4.6 U
Magnesium	SB-10	5/26/1994	130 B
Manganese	SB-10	5/26/1994	18.4
Mercury	SB-10	5/26/1994	0.16
Nickel	SB-10	5/26/1994	2.4 U
Potassium	SB-10	5/26/1994	350 B
Selenium	SB-10	5/26/1994	0.2 U
Silver	SB-10	5/26/1994	244
Sodium	SB-10	5/26/1994	20.5 U
Thallium	SB-10	5/26/1994	0.2 U
Vanadium	SB-10	5/26/1994	3.4 B
Zinc	SB-10	5/26/1994	5.7 U
Aluminum	SB-11	5/26/1994	2120
Antimony	SB-11	5/26/1994	2.4 U
Arsenic	SB-11	5/26/1994	1.3 J
Barium	SB-11	5/26/1994	32.9 B
Beryllium	SB-11	5/26/1994	0.2 U
Cadmium	SB-11	5/26/1994	2.2
Calcium	SB-11	5/26/1994	367 B
Chromium	SB-11	5/26/1994	5.3
Cobalt	SB-11	5/26/1994	0.93 B
Copper	SB-11	5/26/1994	98.4 J
Cyanide	SB-11	5/26/1994	0.11 U
Iron	SB-11	5/26/1994	3300
Lead	SB-11	5/26/1994	18.4

Chemical	Sample Identification	Date Collected	Result
Magnesium	SB-11	5/26/1994	372 B
Manganese	SB-11	5/26/1994	44.6
Mercury	SB-11	5/26/1994	0.15
Nickel	SB-11	5/26/1994	5.6 B
Potassium	SB-11	5/26/1994	335 B
Selenium	SB-11	5/26/1994	0.2 U
Silver	SB-11	5/26/1994	232
Sodium	SB-11	5/26/1994	29.4 U
Thallium	SB-11	5/26/1994	0.2 U
Vanadium	SB-11	5/26/1994	8.4 B
Zinc	SB-11	5/26/1994	25.3
Aluminum	SB-12	5/26/1994	3310
Antimony	SB-12	5/26/1994	2.4 U
Arsenic	SB-12	5/26/1994	2.4 J
Barium	SB-12	5/26/1994	696
Beryllium	SB-12	5/26/1994	0.2 U
Cadmium	SB-12	5/26/1994	0.77 B
Calcium	SB-12	5/26/1994	449 B
Chromium	SB-12	5/26/1994	8
Cobalt	SB-12	5/26/1994	1.7 B
Copper	SB-12	5/26/1994	496 J
Cyanide	SB-12	5/26/1994	0.11 U
Iron	SB-12	5/26/1994	4360
Lead	SB-12	5/26/1994	23.5
Magnesium	SB-12	5/26/1994	617 B
Manganese	SB-12	5/26/1994	51.3
Mercury	SB-12	5/26/1994	0.2
Nickel	SB-12	5/26/1994	6.3 B
Potassium	SB-12	5/26/1994	342.4 U
Selenium	SB-12	5/26/1994	0.2 U
Silver	SB-12	5/26/1994	282
Sodium	SB-12	5/26/1994	26.9 U
Thallium	SB-12	5/26/1994	0.2 U
Vanadium	SB-12	5/26/1994	10.2 B
Zinc	SB-12	5/26/1994	69
Aluminum	SB-13	5/26/1994	1250
Antimony	SB-13	5/26/1994	2.4 U
Arsenic	SB-13	5/26/1994	0.73 J
Barium	SB-13	5/26/1994	379
Beryllium	SB-13	5/26/1994	0.2 U
Cadmium	SB-13	5/26/1994	2
Calcium	SB-13	5/26/1994	468 B
Chromium	SB-13	5/26/1994	3.1
Cobalt	SB-13	5/26/1994	0.53 B
Copper	SB-13	5/26/1994	41.1 J
Cyanide	SB-13	5/26/1994	0.1 U
Iron	SB-13	5/26/1994	1620
Lead	SB-13	5/26/1994	4.4 U
Magnesium	SB-13	5/26/1994	348 B

Chemical	Sample Identification	Date Collected	Result
Manganese	SB-13	5/26/1994	21.5
Mercury	SB-13	5/26/1994	0.18
Nickel	SB-13	5/26/1994	2.6 B
Potassium	SB-13	5/26/1994	342.4 U
Selenium	SB-13	5/26/1994	0.2 U
Silver	SB-13	5/26/1994	234
Sodium	SB-13	5/26/1994	24.6 U
Thallium	SB-13	5/26/1994	0.2 U
Vanadium	SB-13	5/26/1994	4.2 B
Zinc	SB-13	5/26/1994	15.7
Aluminum	SB-2	5/17/1994	5690
Antimony	SB-2	5/17/1994	2.4 U
Arsenic	SB-2	5/17/1994	0.4 U
Barium	SB-2	5/17/1994	69.6
Beryllium	SB-2	5/17/1994	0.2 U
Cadmium	SB-2	5/17/1994	22.1
Calcium	SB-2	5/17/1994	365 B
Chromium	SB-2	5/17/1994	9
Cobalt	SB-2	5/17/1994	1.4 B
Copper	SB-2	5/17/1994	17.1
Cyanide	SB-2	5/17/1994	0.11 U
Iron	SB-2	5/17/1994	7360
Lead	SB-2	5/17/1994	7.4
Magnesium	SB-2	5/17/1994	754 B
Manganese	SB-2	5/17/1994	58.2
Mercury	SB-2	5/17/1994	0.4 U
Nickel	SB-2	5/17/1994	4.8 U
Potassium	SB-2	5/17/1994	342.4 U
Selenium	SB-2	5/17/1994	0.2 U
Silver	SB-2	5/17/1994	307
Sodium	SB-2	5/17/1994	46.9 B
Thallium	SB-2	5/17/1994	0.2 U
Vanadium	SB-2	5/17/1994	13.4
Zinc	SB-2	5/17/1994	36.2
Aluminum	SB-20	8/4/1994	1890
Antimony	SB-20	8/4/1994	2.4 U
Arsenic	SB-20	8/4/1994	0.63 J
Barium	SB-20	8/4/1994	6.6 B
Beryllium	SB-20	8/4/1994	0.21 U
Cadmium	SB-20	8/4/1994	0.63 UJ
Calcium	SB-20	8/4/1994	261 B
Chromium	SB-20	8/4/1994	3.3 J
Cobalt	SB-20	8/4/1994	18.7
Copper	SB-20	8/4/1994	4 B
Cyanide	SB-20	8/4/1994	0.11 U
Iron	SB-20	8/4/1994	2990
Lead	SB-20	8/4/1994	2.6 J
Magnesium	SB-20	8/4/1994	339 B
Manganese	SB-20	8/4/1994	48.1

Chemical	Sample Identification	Date Collected	Result
Mercury	SB-20	8/4/1994	0.13 U
Nickel	SB-20	8/4/1994	1.7 U
Potassium	SB-20	8/4/1994	380 B
Selenium	SB-20	8/4/1994	0.4 U
Silver	SB-20	8/4/1994	9.3
Sodium	SB-20	8/4/1994	35.3 U
Thallium	SB-20	8/4/1994	0.63 U
Vanadium	SB-20	8/4/1994	4.9 J
Zinc	SB-20	8/4/1994	8.2 J
Aluminum	SB-20R	8/4/1994	2120
Antimony	SB-20R	8/4/1994	3 U
Arsenic	SB-20R	8/4/1994	1.6 J
Barium	SB-20R	8/4/1994	5.9 B
Beryllium	SB-20R	8/4/1994	0.22 U
Cadmium	SB-20R	8/4/1994	0.63 UJ
Calcium	SB-20R	8/4/1994	766 B
Chromium	SB-20R	8/4/1994	2.8 J
Cobalt	SB-20R	8/4/1994	24.8
Copper	SB-20R	8/4/1994	4.9 B
Cyanide	SB-20R	8/4/1994	0.11 U
Iron	SB-20R	8/4/1994	3090
Lead	SB-20R	8/4/1994	5.8 J
Magnesium	SB-20R	8/4/1994	363 B
Manganese	SB-20R	8/4/1994	50.7
Mercury	SB-20R	8/4/1994	0.13 U
Nickel	SB-20R	8/4/1994	3.1 B
Potassium	SB-20R	8/4/1994	262 U
Selenium	SB-20R	8/4/1994	0.4 U
Silver	SB-20R	8/4/1994	11.3
Sodium	SB-20R	8/4/1994	36.2 U
Thallium	SB-20R	8/4/1994	0.65 U
Vanadium	SB-20R	8/4/1994	5.3 J
Zinc	SB-20R	8/4/1994	25.9 J
Aluminum	SB-3	5/18/1994	8680
Antimony	SB-3	5/18/1994	2.4 U
Arsenic	SB-3	5/18/1994	1.8 J
Barium	SB-3	5/18/1994	106
Beryllium	SB-3	5/18/1994	0.31 B
Cadmium	SB-3	5/18/1994	2.2
Calcium	SB-3	5/18/1994	24700
Chromium	SB-3	5/18/1994	8.7
Cobalt	SB-3	5/18/1994	2 B
Copper	SB-3	5/18/1994	11.4
Cyanide	SB-3	5/18/1994	0.11 U
Iron	SB-3	5/18/1994	8470
Lead	SB-3	5/18/1994	8.8
Magnesium	SB-3	5/18/1994	14900
Manganese	SB-3	5/18/1994	81.3
Mercury	SB-3	5/18/1994	0.4 U

Chemical	Sample Identification	Date Collected	Result
Nickel	SB-3	5/18/1994	4.9 U
Potassium	SB-3	5/18/1994	406 B
Selenium	SB-3	5/18/1994	0.2 U
Silver	SB-3	5/18/1994	108
Sodium	SB-3	5/18/1994	63.8 B
Thallium	SB-3	5/18/1994	0.2 U
Vanadium	SB-3	5/18/1994	19.6
Zinc	SB-3	5/18/1994	50.3
Aluminum	SB-4	5/18/1994	4310
Antimony	SB-4	5/18/1994	2.4 U
Arsenic	SB-4	5/18/1994	1.4 B
Barium	SB-4	5/18/1994	14.5 B
Beryllium	SB-4	5/18/1994	0.2 U
Cadmium	SB-4	5/18/1994	1.1
Calcium	SB-4	5/18/1994	264 B
Chromium	SB-4	5/18/1994	5.7
Cobalt	SB-4	5/18/1994	1.9 B
Copper	SB-4	5/18/1994	4.9 B
Cyanide	SB-4	5/18/1994	0.11 U
Iron	SB-4	5/18/1994	5490
Lead	SB-4	5/18/1994	5.8
Magnesium	SB-4	5/18/1994	637 B
Manganese	SB-4	5/18/1994	76.4
Mercury	SB-4	5/18/1994	0.15
Nickel	SB-4	5/18/1994	2.7 U
Potassium	SB-4	5/18/1994	374 B
Selenium	SB-4	5/18/1994	0.2 U
Silver	SB-4	5/18/1994	69.7
Sodium	SB-4	5/18/1994	23.4 U
Thallium	SB-4	5/18/1994	0.2 U
Vanadium	SB-4	5/18/1994	11.3
Zinc	SB-4	5/18/1994	12
Cadmium	SB-6A-1	3/29/1999	0.231 U
Mercury	SB-6A-1	3/29/1999	0.035
Silver	SB-6A-1	3/29/1999	17
Cadmium	SB-6A-2	3/29/1999	0.259 U
Mercury	SB-6A-2	3/29/1999	0.031
Silver	SB-6A-2	3/29/1999	23
Cadmium	SB-6A-22	3/30/1999	0.241 U
Mercury	SB-6A-22	3/30/1999	0.0149 UJ
Silver	SB-6A-22	3/30/1999	0.592 U
Cadmium	SB-6A-25	3/29/1999	0.249 U
Mercury	SB-6A-25	3/29/1999	0.0156 U
Silver	SB-6A-25	3/29/1999	0.623 U
Cadmium	SB-6A-3	3/29/1999	0.208 U
Mercury	SB-6A-3	3/29/1999	0.013 U
Silver	SB-6A-3	3/29/1999	0.521 U
Cadmium	SB-6B-1	3/30/1999	0.202 U
Mercury	SB-6B-1	3/30/1999	0.0126 U

Chemical	Sample Identification	Date Collected	Result
Silver	SB-6B-1	3/30/1999	0.504 U
Cadmium	SB-6B-2	3/30/1999	0.238 U
Mercury	SB-6B-2	3/30/1999	0.0146 U
Silver	SB-6B-2	3/30/1999	0.583 U
Cadmium	SB-6B-22	3/31/1999	0.248 U
Mercury	SB-6B-22	3/31/1999	0.0151 UJ
Silver	SB-6B-22	3/31/1999	0.617 U
Cadmium	SB-6B-25	3/30/1999	0.208 U
Mercury	SB-6B-25	3/30/1999	0.013 U
Silver	SB-6B-25	3/30/1999	0.519 U
Cadmium	SB-6B-3	3/30/1999	0.205 U
Mercury	SB-6B-3	3/30/1999	0.0128 U
Silver	SB-6B-3	3/30/1999	0.511 U
Cadmium	SB-6F-1	4/1/1999	37.4
Mercury	SB-6F-1	4/1/1999	2.41
Silver	SB-6F-1	4/1/1999	11000
Cadmium	SB-6F-10	4/1/1999	229
Mercury	SB-6F-10	4/1/1999	0.108
Cadmium	SB-6F-11	4/1/1999	170
Mercury	SB-6F-11	4/1/1999	0.283
Silver	SB-6F-11	4/1/1999	2960
Cadmium	SB-6F-12	4/1/1999	100
Mercury	SB-6F-12	4/1/1999	0.117
Silver	SB-6F-12	4/1/1999	7490
Cadmium	SB-6F-13	4/1/1999	28.6
Mercury	SB-6F-13	4/1/1999	0.041
Silver	SB-6F-13	4/1/1999	110
Cadmium	SB-6F-14	4/1/1999	186
Mercury	SB-6F-14	4/1/1999	0.16
Silver	SB-6F-14	4/1/1999	3210
Cadmium	SB-6F-15	4/1/1999	15.5
Mercury	SB-6F-15	4/1/1999	0.331
Silver	SB-6F-15	4/1/1999	171
Cadmium	SB-6F-16	4/1/1999	156
Mercury	SB-6F-16	4/1/1999	0.148
Silver	SB-6F-16	4/1/1999	3100
Cadmium	SB-6F-2	4/1/1999	240
Mercury	SB-6F-2	4/1/1999	0.27
Silver	SB-6F-2	4/1/1999	1330
Cadmium	SB-6F-3	4/1/1999	435
Mercury	SB-6F-3	4/1/1999	0.125
Silver	SB-6F-3	4/1/1999	2370
Cadmium	SB-6F-5	4/1/1999	13.1
Mercury	SB-6F-5	4/1/1999	0.068
Silver	SB-6F-5	4/1/1999	1940
Cadmium	SB-6F-6	4/1/1999	52.6
Mercury	SB-6F-6	4/1/1999	0.149
Silver	SB-6F-6	4/1/1999	4130
Cadmium	SB-6F-7	4/1/1999	47

Chemical	Sample Identification	Date Collected	Result
Mercury	SB-6F-7	4/1/1999	0.33
Silver	SB-6F-7	4/1/1999	1220
Cadmium	SB-6F-8	4/1/1999	157
Mercury	SB-6F-8	4/1/1999	0.121
Silver	SB-6F-8	4/1/1999	4120
Cadmium	SB-6F-9	4/1/1999	39.2
Mercury	SB-6F-9	4/1/1999	0.015
Silver	SB-6F-9	4/1/1999	430
Aluminum	SB-7	5/26/1994	858
Antimony	SB-7	5/26/1994	2.4 U
Arsenic	SB-7	5/26/1994	0.69 J
Barium	SB-7	5/26/1994	216
Beryllium	SB-7	5/26/1994	0.2 U
Cadmium	SB-7	5/26/1994	1
Calcium	SB-7	5/26/1994	160 B
Chromium	SB-7	5/26/1994	3.1
Cobalt	SB-7	5/26/1994	0.55 B
Copper	SB-7	5/26/1994	16 J
Cyanide	SB-7	5/26/1994	0.1 U
Iron	SB-7	5/26/1994	1380
Lead	SB-7	5/26/1994	5.5 U
Magnesium	SB-7	5/26/1994	175 B
Manganese	SB-7	5/26/1994	16
Mercury	SB-7	5/26/1994	0.4 U
Nickel	SB-7	5/26/1994	2.4 U
Potassium	SB-7	5/26/1994	342.4 U
Selenium	SB-7	5/26/1994	0.2 U
Silver	SB-7	5/26/1994	235
Sodium	SB-7	5/26/1994	33.9 U
Thallium	SB-7	5/26/1994	0.2 U
Vanadium	SB-7	5/26/1994	3.5 B
Zinc	SB-7	5/26/1994	6.4 U
Aluminum	SB-8	5/26/1994	849
Antimony	SB-8	5/26/1994	2.4 U
Arsenic	SB-8	5/26/1994	0.4 UJ
Barium	SB-8	5/26/1994	8 B
Beryllium	SB-8	5/26/1994	0.2 U
Cadmium	SB-8	5/26/1994	0.6 U
Calcium	SB-8	5/26/1994	135 U
Chromium	SB-8	5/26/1994	2.8
Cobalt	SB-8	5/26/1994	0.4 U
Copper	SB-8	5/26/1994	25.2 J
Cyanide	SB-8	5/26/1994	0.11 U
Iron	SB-8	5/26/1994	1380
Lead	SB-8	5/26/1994	5.4 U
Magnesium	SB-8	5/26/1994	151 B
Manganese	SB-8	5/26/1994	14.2
Mercury	SB-8	5/26/1994	0.4 U
Nickel	SB-8	5/26/1994	2.6 B

Chemical	Sample Identification	Date Collected	Result
Potassium	SB-8	5/26/1994	342.4 U
Selenium	SB-8	5/26/1994	0.2 U
Silver	SB-8	5/26/1994	193
Sodium	SB-8	5/26/1994	22.6 U
Thallium	SB-8	5/26/1994	0.2 U
Vanadium	SB-8	5/26/1994	3 B
Zinc	SB-8	5/26/1994	7.2 U
Aluminum	SB-9	5/26/1994	587
Antimony	SB-9	5/26/1994	2.4 U
Arsenic	SB-9	5/26/1994	0.4 UJ
Barium	SB-9	5/26/1994	12.8 B
Beryllium	SB-9	5/26/1994	0.2 U
Cadmium	SB-9	5/26/1994	0.6 U
Calcium	SB-9	5/26/1994	168 B
Chromium	SB-9	5/26/1994	6.5
Cobalt	SB-9	5/26/1994	0.4 U
Copper	SB-9	5/26/1994	36.1 J
Cyanide	SB-9	5/26/1994	0.11 U
Iron	SB-9	5/26/1994	1000
Lead	SB-9	5/26/1994	5.2 U
Magnesium	SB-9	5/26/1994	127 B
Manganese	SB-9	5/26/1994	6.9
Mercury	SB-9	5/26/1994	0.4 U
Nickel	SB-9	5/26/1994	2 B
Potassium	SB-9	5/26/1994	342.4 U
Selenium	SB-9	5/26/1994	0.38 U
Silver	SB-9	5/26/1994	244
Sodium	SB-9	5/26/1994	29.6 U
Thallium	SB-9	5/26/1994	0.2 U
Vanadium	SB-9	5/26/1994	2.1 B
Zinc	SB-9	5/26/1994	6.7 U
Aluminum	SB-9R	5/26/1994	436
Antimony	SB-9R	5/26/1994	2.4 U
Arsenic	SB-9R	5/26/1994	0.4 UJ
Barium	SB-9R	5/26/1994	6.5 B
Beryllium	SB-9R	5/26/1994	0.2 U
Cadmium	SB-9R	5/26/1994	0.6 U
Calcium	SB-9R	5/26/1994	99.6 U
Chromium	SB-9R	5/26/1994	6.4
Cobalt	SB-9R	5/26/1994	0.4 U
Copper	SB-9R	5/26/1994	22.3 J
Cyanide	SB-9R	5/26/1994	0.11 U
Iron	SB-9R	5/26/1994	726
Lead	SB-9R	5/26/1994	4.4 U
Magnesium	SB-9R	5/26/1994	81.4 B
Manganese	SB-9R	5/26/1994	5.5
Mercury	SB-9R	5/26/1994	0.14
Nickel	SB-9R	5/26/1994	2.4 U
Potassium	SB-9R	5/26/1994	342.4 U

Chemical	Sample Identification	Date Collected	Result
Selenium	SB-9R	5/26/1994	0.2 U
Silver	SB-9R	5/26/1994	265
Sodium	SB-9R	5/26/1994	29.5 U
Thallium	SB-9R	5/26/1994	0.2 U
Vanadium	SB-9R	5/26/1994	1.6 B
Zinc	SB-9R	5/26/1994	4.7 U

Appendix B

Toxicological Profiles

TOXICITY PROFILES OF CHEMICALS OF CONCERN

ALUMINUM

Aluminum (Al; CAS Registry No. 7429-90-5) has a wide variety of uses in the building, construction, photographic, and food packaging industries and in the manufacture of steel, aircraft, automobiles, printing inks, dental alloys, medications, explosives, incendiaries, and fireworks (ACGIH 1991).

Carcinogenicity

Several studies of workers in aluminum reduction facilities showed excessive deaths from lung, pancreatic, lymphatic, brain, or bladder cancers, but they were confounded by simultaneous exposures to tobacco smoke or PAHs from coal tars. (ATSDR 1990).

An inhalation study in animals by Kobayashi et al. (1968) was too seriously flawed in design and reporting to produce reliable information.

Systemic Toxicity

Health effects research in humans and animals has shown that elevated levels of aluminum in the body may be toxic, particularly to the central nervous, skeletal, and hematological systems. Much of aluminum toxicity depends on variations in absorption and chemical form of the aluminum complexes and oxidation states and interactions with dietary constituents. The toxicity of aluminum can be divided into three major categories: (1) the effect of aluminum compounds in the gastrointestinal tract following ingestion, (2) the effect in the lungs following inhalation, and (3) systemic toxicity of aluminum (ATSDR 1990a). Aluminum compounds can alter absorption of other elements in the gastrointestinal tract (i.e., fluoride, calcium, iron, cholesterol, phosphorus) and alter gastrointestinal tract mobility by inhibition of acetylcholine-induced contractions. Inhalation of aluminum dusts can lead to the development of pulmonary fibrosis, producing both restrictive and obstructive pulmonary disease. One of the greatest health concerns is aluminum's proposed association with chronic encephalopathy in humans (Ganrot 1986).

A progressive fatal neurologic syndrome has been noted in patients on long-term intermittent hemodialysis treatment for chronic renal failure and may be due to aluminum intoxication. Symptoms in these patients include a speech disorder followed by dementia, convulsions, and myoclonus. Aluminum content of brain, muscle, and bone tissues is increased in these patients. Sources of the excess aluminum may be from oral aluminum hydroxide commonly given to these patients or from aluminum in dialysis fluid derived from tap water used to prepare the dialysate fluid.

Minimal neurotoxicity in the offspring of mice exposed to aluminum lactate has been observed (Golub et al. 1995). Groups of 16 pregnant Swiss-Webster mice were fed 25 (control group), 500, or 1000 mg Al/kg of aluminum lactate throughout gestation and

lactation (Donald et al. 1989). No treatment-related changes were observed in maternal survival, body weight, food intake, toxic signs or neurobehaviour. A battery of neurobehavioral tests performed on pups showed that a significant (p=0.007) number of pups in the high dose group had impaired vertical screen climb performance. In the group receiving the lowest dose, equivalent to 100 milligrams per kilogram per day (mg/kg-day), decreased forelimb grip strength and increased foot splay distance were observed.

Colomina et al. (1992) studied the influence of lactate on the potential developmental toxicity of aluminum. Groups of 11-13 Swiss albino CD_1 mice were administered 57.5 mg Al/kg bw-day as aluminum hydroxide, aluminum lactate, or aluminum hydroxide together with lactic acid (570 mg/kg-day) by gavage on gestation days 6-15. Other groups were treated only with the same amount of lactic acid or distilled water (controls). Maternal weight gain was significantly lower in the aluminum lactate-treated mice--and significantly less food was consumed by this group--than in controls. In the offspring of these females, fetal body weight was also significantly reduced and accompanied by increased incidence of cleft palate, dorsal hyperkyphosis, and delayed parietal ossification. These developmental effects were not seen in any of the pups of the control groups or the aluminum hydroxide dosing groups. A flaw in this study was the lack of accounting for aluminum in the diet of the test animals, since commercial grain-based mouse feeds contain 200-1200 ppm aluminum and varying amounts of other trace metals (Golub et al. 1992).

Colomina et al. (1994) also administered doses of 0 or 104 mg Al/kg bw per day as aluminum hydroxide on days 6-15 of gestation to female Swiss mice. Again, dietary aluminum was not reported. A NOAEL of 104 mg Al/kg-day was reported in this study, which examined maternal body or organ weight, and the per litter number of implantations, resorptions, dead fetuses, sex ratio and fetal body weight, as well as their percentage of positive postimplantation loss. The study found no significant differences between test groups and controls. Thus, from the two Colomina studies, it can be concluded that aluminum lactate appears to be a more potent developmental toxicant in mice than the less water-soluble aluminum hydroxide.

Toxicity Values

The available systemic toxicity data on aluminum have been evaluated and found to be inadequate for quantitative risk assessment (USEPA 1999). The value for the chronic oral RfD (1 mg/kg-day) was obtained from the EPA Environmental Criteria and Assessment Office, Cincinnati, based on a LOAEL of 100 mg/kg-day for neurotoxicity, with an uncertainty factor of 100.

A dermal RfD of 0.2 mg/kg-day was estimated by multiplying the oral RfD by 0.2 (or 20%) (USEPA 1996a). EPA (USEPA 1999) has not established slope factors for aluminum.

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ANTIMONY

Antimony (Sb) is an element typically used in metal alloys, rubber, matches, ceramics, pigments, paints, lacquers, textiles, and also in historical medicines.

Therapeutic administration of antimony-containing compounds has produced cardiac effects, liver toxicity, pulmonary congestion, skin reactions, vomiting, diarrhea, gastric discomfort, and ulcers in treated humans (ATSDR, 1990).

Inhalation exposures to antimony in industrial settings have produced respiratory tract irritation, pneumoconiosis, and impaired pulmonary function in workers (Cooper et al., 1968; Potkonjak and Vishnijich, 1983). Dermatosis and ocular irritation have also been reported in humans following exposure to airborne antimony (Potkonjak and Vishnijich, 1983).

Carcinogenicity

Antimony is currently assigned a USEPA human carcinogen classification of Category D.

Toxicity Values

For antimony, USEPA has established an oral reference dose (RfD) of 0.0004 mg/kg-day, based on altered longevity, blood glucose, and cholesterol content, and including an aggregate uncertainty factor of 1000 (USEPA, 1998). The uncertainty factor accounts for interspecies and intraspecies extrapolation (10-fold each) and subchronic to chronic NOAEL extrapolation (10-fold).

Because there are insufficient data to quantify the adverse effects of exposure to antimony via the inhalation route, the oral toxicity value is used for chronic inhalation exposure scenarios.

There are no dermal RfDs for antimony. Therefore, the oral RfD for antimony was adjusted with an oral absorption fraction of 20 percent (USEPA, 1996). The chronic dermal RfDs used in this assessment is 8×10^{-5} mg/kg-day.

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BARIUM

Barium (CAS 7440-39-3) is a dense alkaline earth metal and exists in combination with other elements, primarily as barium sulfate or barium carbonate ores, in a divalent cation form. In nature barium enters the environment through the weathering of rocks, minerals and anthropogenic release mechanisms (USEPA 1998). Barium is used as a component in various alloys; in paints, soaps, paper, and rubber; and in the manufacture of ceramics and glass (HSDB 2000).

Carcinogenicity

EPA (USEPA 2000) indicates that there are insufficient data for an assessment of the carcinogenicity of barium compounds (classified Group D). Similarly, ATSDR (1992) noted a lack of carcinogenicity studies via inhalation or dermal exposure routes, but summarized two rodent studies (Schroeder and Mitchener 1975 a,b) that observed no significant differences in tumor incidence between treated and control animals. ATSDR, however, considered these rodent studies inadequate for evaluating the carcinogenic potential of barium, due to insufficient numbers of animals, no determination of an MTD, only one exposure dose, incomplete histology, and an unspecified purity of the test material.

Systemic Toxicity

Exposures to barium via the oral route are used in the majority of studies evaluating potential toxicity. ATSDR (1992) indicated a variety of effects following subchronic or chronic oral administration of barium, including mortality, respiratory, cardiovascular, hematological, musculoskeletal, hepatic, renal, neurological, and other effects. For both subchronic (15 to 364 days) and chronic (>365 days) exposures, cardiovascular effects were the most sensitive endpoint. With subchronic exposures, rodent NOAELs for all systemic effects except cardiovascular were approximately 10 mg/kg-d. For cardiovascular effects, rat NOAELs were reported as low as 0.64-0.71 mg/kg-d (Perry et al. 1983, 1985, 1989) and the human NOAEL was reported as 0.21 mg/kg-d (Wones et al. 1990).

Studies identifying the adverse effects of subchronic or chronic barium exposure via inhalation are limited in their utility for linking adverse health effects with barium exposure (ATSDR 1992). Even with the limited utility of these studies, these studies suggest a potential association between non-acute barium inhalation exposures and respiratory, cardiovascular, hematological, hepatic, developmental, and reproductive effects (ATSDR 1992).

Toxicity Values

EPA (USEPA 1998) retained its previously established oral RfD for barium of 7×10^{-2} mg/kg-day, but changed the critical effect on which it was based from increased hypertension to increased kidney weights observed in subchronic and chronic animal

drinking water studies (NTP 1994); this effect is supported by other rodent studies in which kidney weight increased (Perry et al. 1983; McCauley et al. 1985; Schroeder and Mitchener, 1975a,b; Tardiff et al. 1980). Human epidemiologic studies (Brenniman and Levy 1984; Wones et al. 1990) identified a dose at which no adverse effects were found; thus the uncertainty factor has been reduced to 3.

Although human and animal data indicate that the respiratory system is also a target for barium toxicity, EPA does not recommend an inhalation RfC at the present time (USEPA 2000), because study parameters and results were poorly reported.

Barium is presently listed by EPA in Group D (not classifiable as to human carcinogenicity); but it is considered not likely to be carcinogenic to humans via oral exposure under EPA's proposed guidelines for carcinogenic risk assessment (USEPA 1996). Its carcinogenic potential following inhalation exposure cannot be determined.

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CADMIUM

Cadmium (Cd, CAS Registry No. 7440-43-9) is not known definitively as an essential element, but there is limited evidence suggesting an essential role in experimental animals (Schwarz 1977), and some enzymes can accommodate cadmium in place of zinc (Vallee and Ulmer 1972). Because of its chemical and atomic similarities to zinc, cadmium may be cycle through homeostatic mechanisms that maintain a physiologically-balanced supply of zinc.

Carcinigenicity

The carcinogenic potential of cadmium appears to be dependent on the route of administration. In Wistar rats, cadmium inhalation produces lung tumors (Takenaka et al. 1983), while intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats, and instead produced mammary tumors in males and tumors at distant sites (Sanders and Mahaffey 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice, but not following ingestion (USEPA 1985; Kazantzis 1984; Takenaka et al. 1983). Human carcinogenicity is not as well defined, however. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers and was not attributable to co-occurrence of arsenic or to smoking (Thun et al. 1985), which are confounding aspects of several other studies (Varner 1983; Sorahan and Waterhouse 1983; Armstrong and Kazantzis 1983). Further limited evidence for carcinogenicity is provided in four studies of workers exposed to cadmium dust or fumes that exhibited a statistically significant positive association with prostate cancer (Kipling and Waterhouse 1967; Lemen et al. 1976; Holden 1980; Sorahan and Waterhouse 1983), but the total number of cases was small in each study (USEPA 1998). EPA has concluded there is no evidence of carcinogenicity via oral exposure and classifies cadmium as a probable human carcinogen (B1) for exposure via inhalation, based on limited but consistent evidence from these human studies and sufficient evidence in rats and mice by inhalation and injection. (USEPA 2001). Insufficient data were available to derive an oral cancer slope factor, however an inhalation cancer slope factor of 6.3 has been established (USEPA 2001).

Mutagenicity

Mutagenicity tests in bacteria and yeast have provided inconclusive results. Positive responses occurred in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells (Casto 1976; Ochi and Ohsawa 1983; 0berly et al. 1982), but conflicting results were obtained in chromosomal aberration assays in human lymphocytes either obtained from exposed workers or treated *in vitro*. In both *in vivo* and *in vitro* tests, cadmium appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters (Shimada et al. 1976; Watanabe et al. 1979; Gilliavod and Leonard 1975).

Systemic Toxicity

As one of the most widely-studied toxicants, effects following cadmium exposure via the oral route are well known in animals and humans. With a variety of experimental durations (from >14 d to >1 yr), adverse effects from cadmium may be expressed in virtually every organ or physiological system: respiratory, cardiovascular, hematological, musculoskeletal, hepatic, renal, immunological, neurological, developmental, or reproductive (ATSDR 1991). Chronic cadmium ingestion also results in the painfully degenerative "itai-itai" disease, affecting the skeletal system, and has been implicated in development of hypertension in exposed individuals. In general for exposures of 15 d to 1 yr, most of the animal LOAELs and NOAELs for adverse effects occur at daily ingestion of arsenic ranging from 0.014 to mg Cd kg^{-1} d^{-1}. For exposures of one year or longer, animal LOAELs and NOAELs range from 0.014 to 57 mg Cd kg^{-1} d^{-1}, while human LOAELs are reported at 0.001 to 0.0021 mg Cd kg^{-1} d^{-1} for renal effects resultant to lifetime exposures (Nogawa et al. 1989; Shiwen et al. 1990).

Chronic inhalation of cadmium produces multiple effects, such as emphysema, liver damage, anemia, proteinuria, and renal tubular damage (Goyer 1986; Lee and White 1980; Friberg et al. 1974) and affects various other organs and organ systems. LOAELs and NOAELs for experimental animals following subchronic or chronic inhalation exposures to cadmium typically range from 0.02 to 6.5 mg Cd m^3. LOAELs and NOAELs for renal effects in humans chronically exposed to cadmium-containing vapors have been reported in the range of 0.017 to 0.033 mg Cd m^3 (ATSDR 1991), while exposure at equivalent levels is carcinogenic to rats (Oldiges et al. 1989; Takenaka et al. 1983).

Toxicokinetics

Absorption of cadmium compounds through the skin is negligible. Only about 5 percent of ingested cadmium is absorbed from the gastrointestinal tract, although a low intake of calcium or iron can increase absorption to as high as 20%. From the respiratory tract, absorption is more complete, and is greater for small particles and fumes than for large particle dust. Cigarette smokers absorb 10-40 percent of inhaled cadmium. After absorption, cadmium binds to blood cells and albumin, which distribute it. Blood cadmium reaches equilibrium within a year of exposure, while the total body burden continues to increase over decades. About 50 percent of the total body burden is found in the liver and kidneys. The elimination half-life of cadmium is 16 to 33 years. Accumulations in the liver remain high, but once kidney damage occurs, excretion of cadmium via the urine greatly increases. Cadmium crosses the placental barrier in rodents (HSDB 2000).

Toxicity Values

EPA has established oral RfDs of 0.0005 mg/kg-d for cadmium in drinking water and 0.001 mg/kg-d for cadmium in food (USEPA 2001). For both exposure media, the critical effect was significant proteinuria in human subjects chronically exposed to

cadmium. Both reference doses were also based on an aggregate uncertainty factor of 10, to account for variability among humans to the toxicity of this chemical in the absence of specific data on sensitive individuals. The RfD is based on the highest level of cadmium in the human renal cortex not associated with significant proteinuria, with modeled exposures adjusted to allow for absorption differences arising from differing cadmium sources (USEPA 2001). EPA currently has no RfC for cadmium (USEPA 1998).

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COBALT

Cobalt (Co, CAS Registry No. 7440-48-4) occurs in nature in many different chemical forms. The pure metal is gray, magnetic, ductile, and somewhat malleable. Cobalt and other cobalt compounds are commonly used to make metal alloys. Small amounts of cobalt are found in food. Cobalt is a component of vitamin B_{12} and is required for good health in humans. Cobalt also can be artificially induced to become radioactive. Cobalt-60 breaks down to form beta particles and gamma radiation, and is used in cancer treatment, storage of wheat and potatoes, sterilization of spices and medical equipment, and processes to locate buried telephone and electric lines. Naturally occurring cobalt can stay in the air for a few days and will remain in water and soil for years (ATSDR 1990).

Carcinogenicity

Cobalt is not known to cause cancer by any route in humans or animals (ATSDR 1990). In a mortality study of cobalt and sodium refinery, authors concluded that the exposure to cobalt did not statistically increase the cause of death from cancer (Mur et al. 1987).

Hamsters treated with 7.9 mg/m^3 cobalt oxide, intermittently for a lifetime did not increase the incidence of tumors (Wehner et al. 1977).

Mutagenicity

No studies have been found to describe genotoxic effects in humans or animals (ATSDR 1990).

Systemic Toxicity

Cobalt has been found to produce adverse effects via the inhalation, oral, and dermal routes. Two studies have been found that suggest a relationship between cobalt exposure and death from lung cancer and cardiomyopathy. In a study by Mur et al. 1987, authors compared mortality in plant workers that worked in a cobalt and sodium refinery and processing facility. An increase in lung cancer related deaths was found in the cohort exposed to cobalt (SMR=4.66; 4 deaths in the cobalt exposed group compared to 1 death in controls). A medical case study reported the death of a metal worker due to cardiomyopathy. High levels of cobalt were found in the tissue and death was attributed to the exposure of high levels of cobalt for 4 years (Barborik and Dusek 1972). Autopsy of this worker also found congestion of the kidneys, congestion of the liver, congestion of the conjunctiva (ocular effect).

Occupational studies have also reported respiratory effects such as irritation, wheezing, asthma, pneumonia and fibrosis. The workers were exposed to 0.003 to 0.893 mg/m^3 for 2-17 years (Anttila et al. 1986; Davison et al. 1983; Demedts et al. 1984; Raffin et al. 1988; Shirakawa et al. 1988, 1989; Sprince et al. 1988; Tabatowski et al. 1988; Van Cutsem et al. 1987).

Cobalt is reported to act synergistically in combination with alcohol or antibiotics. Cardio-vascular effects were observed in people who consumed beer containing cobalt sulfate (used as a foam stabilizer) (Alexander 1969, 1972; Morin et al. 1971) The beer drinkers ingested from 0.04 mg/kg/day to 0.14 mg/kg/day of cobalt for years. Effects included cardiomyopathy characterized by: sinus tachycardia, left ventricular failure, cardiogenic shock, diminished myocardial compliance, absence of a myocardial response to exercise, enlarged heart, and extensive intracellular changes.

In animal studies the LD_{50} has been determined for Wistar rats, ranging from 91 mg/kg (for cobalt fluoride) to 190 mg/kg (for cobalt chloride) depending on the cobalt compound (Speijers et al. 1982). Sprague Dawley rats gavaged with cobalt chloride reported death at 161 mg/kg (Domingo and Llobet 1984).

Rats exposed to 26-30.2 mg/kg/day of cobalt sulfate in the diet or cobalt chloride in drinking water for 2-3 months were observed to have degenerative heart lesions and increased heart weight (Grice et al. 1969; Domingo et al. 1984).

Speijers et al. (1982) observed acute and prolonged exposure to cobalt resulted in renal tubular degeneration in rats exposed to 42 mg/kg.

Toxicokinetics

Inhaled cobalt powder is retained in the lungs and subsequently absorbed slowly. Significant amounts have been found in hair and in the liver and pancreas after exposure. About 10 percent of that absorbed persists for 5-15 years. The rest is rapidly excreted in feces and urine. Absorption of cobalt is reduced by the simultaneous administration of iron (HSDB 2000).

Metabolism

Cobalt directly induces metallothionein synthesis in hepatic tissue and stimulates the production of erythropoietin. It is thought that these are a response to tissue hypoxia resulting from an inhibition of enzymes involved in oxidative metabolism. More specifically, cobalt blocks the conversion of pyruvate to acetyl coenzyme A and of alpha-ketoglutarate to succinate (HSDB 2000).

Toxicity Values

EPA has not quantitatively estimated the risks of exposure to cobalt via any pathway and does not include it in the IRIS database (USEPA 2001). USEPA NCEA has published a provisional RfD of 0.06 mg/kg/day which has been used in this document.

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COPPER

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR, 1990; U.S. EPA, 1987).

Copper sulfate is the most common copper salt; however, other important copper salts include carbonate, cyanide, oxide, and sulfide. These are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986).

Copper is an essential trace element that is widely distributed in animal and plant tissues. It is a component of a number of metalloenzymes such as catalase, peroxidases, and cytochrome oxidase, and is essential for the utilization of iron (Goyer, 1991; Stokinger, 1981). Although most copper salts occur in two valence states, as cuprous (Cu^{+}) or cupric (Cu^{2+}) ions, the biological availability and toxicity of copper is most likely associated with the divalent state (ATSDR, 1990). The general population may be exposed to increased levels of copper in drinking water largely as a result of the corrosion of plumbing materials (U.S. EPA, 1987). Contact with copper may also result from use of copper fungicides and algicides. Workers may be exposed to copper in agriculture, and in various industries such as copper production and metal plating. The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plantr, such as windblown dust and volcanic eruptions, may be significant (ATSDR, 1990).

Carcinogenicity

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper. Therefore, U.S. EPA (1991a) has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

Systemic Toxicity

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (U.S. AF, 1990). Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu/L (Mueller-Hoecker et al., 1988; Spitalny et al., 1984). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. The clinical manifestations of Wilson's disease

include cirrhosis of the liver, hemolytic anemia, neurologic abnormalities, and corneal opacities (Goyer, 1991; ATSDR, 1990; U.S. EPA, 1987). In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of ÿ100 mg/kg/day; and hematological effects at doses of 40 mg/kg/day (U.S. EPA, 1986; Haywood, 1985; 1980; Rana and Kumar, 1978; Gopinath et al., 1974; Kline et al., 1971).

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m^3 may cause metal fume fever with symptoms such as cough, chills and muscle ache (U.S. AF, 1990). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly (Suciu et al., 1981). Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR, 1990).

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals (Lecyk, 1980; Ferm and Hanlon, 1974). Evidence also indicates that copper compounds are spermicidal (ATSDR, 1990; Battersby et al., 1982).

Metabolism

The metabolism of copper involves mainly its transfer to and from various organic ligands, most notably sulfhydryl and imidazole groups on amino acids and proteins (ATSDR, 1990). The liver, the most important organ involved in copper disposition, receives its copper from serum ÿ-globulin. It serves as a storage depot of copper; as a site for ceruloplasmin synthesis, which is then released into the blood; and as a site for the formation of various copper complexes for subsequent biliary excretion (U.S. EPA, 1987). Biliary copper is returned to the intestine and excreted in the feces (Stokinger, 1981). The half-life of injected copper was approximately 4 weeks in normal human subjects (Aaseth and Norseth, 1986).

Toxicity Values

A Reference Dose (RfD) for elemental copper is not available (U.S. EPA, 1992). However, EPA established an action level of 1300 µg/L for drinking water (56 FR 26460, June 7, 1991). Data were insufficient to derive a Reference concentration (RfC) for copper.

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IRON

Iron (Fe, CAS registry number 7439-89-6) is the fourth most abundant element in the earth's crust and the second most abundant metal. It comprises approximately 5 percent of the continental crust. Its concentration in ground water ranges from 0.5 mg/L to 10 mg/L; its concentration in soil is between 0.7 and 4.2 percent (NRC 1979). In the late 1970s, world production of iron was over 500 million metric tons, with the U.S. producing roughly 20 percent of the world total (NRC 1979). Since iron is an essential nutrient, some amount of iron is needed in the diet.

Carcinogenicity

Iron has not been reported to be mutagenic (NRC 1979). It has not been classified with respect to carcinogenicity, given the paucity of animal cancer bioassays and human cancer studies. Iron overload may be associated with carcinoma of the liver; however the data are poor and inconclusive (NRC 1979).

Systemic Toxicity

The acute effects of iron toxicity in humans are well characterized and consist of gastrointestinal, cardiovascular, metabolic, neurological and hepatic alterations (Bothwell et al. 1979; Banner and Tong 1986; Engle et al. 1987; and Mann et al. 1989, all as cited in EPA 1993). Acute effects are based mostly on observations of children who accidentally ingest therapeutic iron supplements; they are rarely, if ever, associated with ingestion of naturally occurring or other commercially produced substances (NRC 1979). Gastrointestinal toxicity is characterized by vomiting, diarrhea, and abdominal pain, caused by the direct caustic effect of iron on the mucosa of the stomach and small intestine. Gastrointestinal toxicity can progress to gastric/intestinal hemorrhage and/or necrosis and, in rare cases, to stenosis in the stomach outlet and small intestine. Cardiovascular iron toxicity is marked by severe hemodynamic alterations and can lead to shock and cardiac failure; neurological toxicity ranges from lethargy to coma. Although a rare occurrence, hepatic toxicity from iron can range from cloudy swelling of hepatocytes to necrosis. The average human lethal dose is 200B250 mg/kg body weight (NRC 1979). Thus, the average adult male would have to ingest 14 grams of elemental iron for it to be lethal; the average 2 year old, 3 grams (NRC 1979).

Chronic iron toxicity has been noted in individuals with various genetic and/or metabolic disorders, including hemochromatosis (massive iron overload together with cirrhosis and/or other tissue damage due to iron), thalassemia, and sideroblastic anemia, as well as in individuals who receive frequent blood transfusions (Jacobs 1977, and Bothwell et al. 1979, both as cited in EPA 1993). Excessive intake of iron attributed to consumption of home-brewed Kaffir beer has resulted in chronic hemochromatosis among the South African Bantu population (NRC 1979; and Bothwell and Bradlow 1960; and Bothwell et al. 1964, both as cited in EPA 1993). Pathologic findings associated with hemochromatosis include: 1) fibrosis in heavily siderotic organs, particularly the liver, 2) cirrhosis, 3) testicular atrophy, and 4) osteoporosis (NRC 1979).

Though chronic iron toxicity can occur in individuals with genetic/metabolic disorders, it is debatable whether a chronic overload via ingestion is possible in individuals with a normal ability to control iron absorption. Using values obtained from the second National Health and Nutrition Examination Survey (NHANES II), Looker et al. (1988, as cited in EPA 1993) compared dietary iron intake with biochemical indices of iron status. NHANES II consisted of a 1976B1980 sample of the U.S. population aged 6 months to 74 years. Observed intake levels of 0.15B0.27 mg/kg-day iron were found to be both great enough to prevent iron deficiency and insufficient to cause the toxic effects of iron overload (Elinder 1986; Cook 1991; Hillman and Finch 1985, all as cited in EPA 1993). Lauffer (1991, as cited in EPA 1993) and Sullivan (1992, as cited in EPA 1993) suggest that iron overload elevates the risk of acute myocardial infarction by promoting oxidation of low density lipoprotein (LDL). A 1992 Finnish study of 1,931 randomly selected men aged 42B60 years by Salonen et al. lends support to this theory in that it found that high serum ferritin concentration and high dietary iron intake were risk factors for myocardial infarction.

Animal studies attempting to model hemochromatosis have been mostly negative, as have animal studies involving parenteral administration of iron (Bothwell et al. 1979, as cited in EPA 1993; and NRC 1979).

Ingestion of iron supplements during pregnancy has not been correlated to adverse developmental effects in humans, although some women ingesting large quantities of iron (>1.2 gram) during pregnancy experienced nausea, vomiting, hematoemesis, abdominal pain, and/or diarrhea (NAS 1989, as cited in EPA 1993). No teratogenic effects have been associated with iron (NRC 1979).

No treatment-related teratogenic or embryotoxic effects were observed in rats given 2.7 mg/kg-day iron on gestation days 6-15 or rats/mice given 24-76 mg/kg-day iron for 6 days (Nolen et al. 1972; Tadokoro et al. 1979, as cited in EPA 1993).

This essential nutrient is found primarily in the form of hemoglobin in the body. The concentration of iron in the body at any given point is regulated largely through changes in the amount of iron absorbed by the gastrointestinal mucosa. The following factors influence the absorption of iron: 1) body stores, 2) the amount and nature of iron in ingested food, and 3) dietary factors that may increase or decrease the availability of iron for absorption (NRC 1979). Although the body is generally effective in regulating iron levels, it is incapable of excreting large amounts of iron following excessive accumulation resulting from acute or chronic ingestion of high levels of iron (NAS 1989, as cited in EPA 1993).

Toxicity Values

Using values obtained from the second National Health and Nutrition Examination Survey (NHANES II), Looker et al. (1988, as cited in EPA 1993) compared dietary iron intake with biochemical indices of iron status. NHANES II consisted of a 1976-1980

sample of the U.S. population aged 6 months to 74 years. Observed intake levels of 0.15-0.27 mg/kg-day iron were found to be both great enough to prevent iron deficiency and insufficient to cause the toxic effects of iron overload (Elinder 1986; Cook 1991; and Hillman and Finch 1985, as cited in EPA 1993). EPA (1993) has proposed a provisional chronic oral RfD of 0.3 mg/kg-day based on a NOAEL of 0.27 mg/kg-day (i.e., the upper-bound value in the range of mean dietary iron intakes from the NHANES II data base) and an uncertainty factor of 1. An uncertainty factor of 1 was used since: 1) iron is an essential nutrient, 2) the NHANES II data base comprised a relatively large sample size, and 3) humans exert an efficient homeostatic control over iron such that body burdens are kept constant with variations in diet. EPA's confidence in the critical study is high, while confidence in the data base is medium, resulting in medium confidence in the RfD. EPA (1993) suggests that the RfD may not be protective of people with inherited disorders of iron metabolism. In addition, EPA states that the RfD could be conservative if applied to exposure scenarios involving forms of iron with low bioavailability. The NCEA has established a provisional chronic RfD of 0.3 (mg/kg/day) (USEPA 1996, 1998). Iron has not been classified with respect to carcinogenicity. Slope factors are not available for iron.

The TLV-TWA for iron salts is 1 mg/m^3 (ACGIH 1995).

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MANGANESE

Manganese (Mn; CAS Registry No. 7439-96-5) is a pinkish gray, lustrous and brittle metal that is used in rock crushers and in the manufacture of ceramics, matches, glass, dyes, welding rods. It is a component of steel and metal alloys. (HSDB 2000).

Carcinogenicity

EPA considers manganese to be unclassifiable as a human carcinogen (Category D), based on an absence of human carcinogenicity data, inadequate evidence of carcinogenicity in animals, and inadequate genotoxicity data (EPA 1999).

Mutagenicity

Few genotoxicity assays of manganese have been conducted. No studies were located regarding genotoxic effects in humans (ATSDR 1991). Treatment of male rats with manganese at repeated oral doses of 0.014 mg/kg-day manganese for 80 days did not produce any significant chromo-somal damage in bone marrow or sperm cells (Dikshith and Chandra 1978). Results of *in vitro* genotoxicity assays have been mixed. The available data indicate that manganese may have genotoxic potential, but they are not sufficient to evaluate the genotoxic risk of manganese to humans (ATSDR 1991).

Systemic Toxicity

Humans exert an efficient homeostatic control over manganese so that body burdens are kept constant with variations in diet. Manganese is an essential element, being required for normal human growth and maintenance of health. Children may be less susceptible to manganese intoxication and may require slightly higher levels of manganese than do adults (EPA 1995).

The World Health Organization (WHO 1973) reviewed several investigations of adult diets and reported the average daily consumption of manganese to range from 2.0 to 8.8 mg/day. Higher manganese intakes are associated with diets high in whole-grain cereals, nuts, green leafy vegetables, and tea. Depending on individual diets, a normal intake may be well over 10 mg Mn/day, especially from a vegetarian diet. While the actual intake is higher, the bioavailability of manganese from a vegetarian diet is lower, thereby decreasing the actual absorbed dose. From manganese balance studies, the WHO concluded that 2-3 mg/day is adequate for adults and that 8-9 mg/day is "perfectly safe" (WHO 1973).

An epidemiologic study of manganese in drinking water was performed by Kondakis et al. (1989). Three areas in northwest Greece were chosen for this study, with manganese concentrations in natural well water of 3.6-14.6 ug/L in area A, 81.6-252.6 ug/L in area B, and 1,600-2,300 ug/L in area C. The total population of the 3 areas being studied ranged from 3,200 to 4,350 people. Although the amount of manganese in the diet was not reported, the authors indicated that most of the food was purchased from markets.

The individuals chosen were submitted to a neurologic examination, the score of which represents a composite of the presence and severity of 33 symptoms (e.g., weakness/fatigue, gait disturbances, tremors, dystonia). Whole blood and hair manganese concentrations were also determined. The authors indicate that the difference in mean scores for area C versus A was significantly increased for both sexes combined. In a subsequent analysis, logistic regression indicated that there is a significant difference between areas A and C, even when both age and sex are taken into account (Kondakis 1990). The NOAEL identified in this epidemiological study was 0.005 mg/kg-day (EPA 1995).

The major toxic effects of inhaled manganese are primarily neurological. A syndrome called "manganism" has been observed only in workers exposed to chronic, high levels of manganese. It is characterized by preliminary general weakness, anorexia and muscle pain, with psychological signs such as apathy and dullness, as well as impotence. Advanced stages include difficulty in walking, muscle tremor, and behavioral disturbances. This syndrome has not been observed for low level, chronic or sporadic exposures, nor has it been observed in studies with animals (ATSDR 1991).

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (MnO_2) dust in a Belgian alkaline battery plant. A control group of 101 male workers was matched for age, height, weight, work schedule, coffee and alcohol consumption, and smoking; educational level was slightly higher in the control group. The manganese-exposed group had been exposed to MnO_2 for an average of 5.3 years (range: 0.2-17.7 years). The geometric means of the workers' TWA airborne manganese concentrations, as determined by personal sampler monitoring at the breathing zone, were 0.215 mg/m^3 for respirable dust and 0.948 mg/m^3 for total dust. The authors noted that the personal monitoring data were representative of the usual exposure of the workers because work practices had not changed during the last 15 years of the operation of the plant.

Geometric mean concentrations of blood manganese (MnB) (0.81 ug/dL) and urinary manganese (MnU) (0.84 ug/g creatinine) were significantly higher in the Mn-exposed group than in the control group, but on an individual basis no significant correlation was found between either MnB or MnU and various external exposure parameters. A self-administered questionnaire focused on occupational and medical history, neurological complaints, and respiratory symptoms. Responses to the questionnaire indicated no significant differences between groups in either respiratory or neurological symptoms, nor were spirometric, hormonal, or calcium metabolism measurements significantly different for the two groups (Roels et al 1992).

Of particular note, manganese workers performed worse than controls on several measures of neurobehavioral function. Visual reaction time was consistently and significantly slower in the manganese-exposed workers measured in four 2-minute periods, with more pronounced slowing over the total 8-minute period and significantly greater variability in reaction times for the exposed group. Abnormal values for mean reaction times (defined as greater than or equal to

the 95th percentile of the control group) also were significantly more prevalent in the exposed group during three of four 2-minute intervals of the 8-minute testing period. Five measures of eye-hand coordination (precision, percent precision, imprecision, percent imprecision, and uncertainty) reflected more erratic control of fine hand-forearm movement in the exposed group than in the controls, with mean scores on all five measures being highly significantly
different for the two groups. There was also a significantly greater prevalence of abnormal values for these five measures in the manganese-exposed group. The hole tremormeter test of hand steadiness indicated a consistently greater amount of tremor in the exposed workers, with performance for two of the five hole sizes showing statistically significant impairment (Roels et al. 1992).

A LOAEL may be derived from the Roels et al. (1992) study by using the IRD concentration of MnO_2, expressed as mg/m^3 □years (based on 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/m^3 □years) by the average duration of the workers' exposure to MnO_2 (5.3 years) yields a LOAEL of 0.15 mg/m^3. Adjusted for continuous exposure, the LOAEL is 0.05 mg/m^3.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to MnO_2, manganese tetroxide (Mn_3O_4), and various manganese salts (sulfate, carbonate, and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable work-load and work-shift characteristics. Significant differences in mean scores between manganese-exposed and reference subjects were found for objective measures of visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory. The prevalence of abnormal scores on eye-hand coordination and hand steadiness tests showed a dose-response relationship with blood manganese levels; short-term memory scores were related to years of manganese exposure but not to blood manganese levels. The prevalence of subjective symptoms was greater in the exposed group than in controls for 20 of 25 items on the questionnaire, with four items being statistically significant: fatigue, tinnitus, trembling of fingers, and irritability. Based upon the findings of impaired neurobehavioral function in workers whose average Mn exposure was estimated by the geometric mean TWA of total airborne manganese dust at the time of the study, a LOAEL of 0.97 mg/m^3 was identified, which, when adjusted for continuous exposure, is equivalent to a LOAEL of 0.34 mg/m^3. This LOAEL is based on total manganese dust of mixed forms, whereas the LOAEL from Roels et al. (1992) study is based on the measured respirable dust fraction of MnO_2 only.

Minimal information regarding manganese and the dermal exposure route could be located. It is generally regarded that manganese uptake across intact skin is very limited, as is the case for most inorganic forms of metal ions (ATSDR 1991).

Toxicokinetics

Exposure to manganese mainly occurs via ingestion and inhalation. The extent to which manganese is absorbed across the intestine is approximated at 3-5 percent, and does not appear to be substantially influenced by the carrier medium (i.e., water versus food). Similar extents of absorption have been noted in animals as well, with typical amounts equal to 2.5-5.5 percent. Manganese distributes to various tissues following ingestion, and serves as a normal tissue constituent. Tissue levels may be somewhat higher in animal tissues than in their human tissue counterparts. Manganese which is inhaled, typically in particle form, is absorbed to some unknown extent across the lungs, and a certain percentage of inhaled manganese particles are subsequently swallowed and ingested as well (ATSDR 1991).

Metabolism

Manganese is not known to be metabolized or biotransformed, and behavior within the body would be essentially limited to absorption, distribution, potential sequestration, and excretion. The valence state of manganese is thought to undergo changes within the body (alterations in oxidation state), which may influence its ability to form complexes or serve as a co-factor for certain proteins (ATSDR 1991).

The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references.

Toxicity Values

The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references.

Two separate oral RfDs for manganese have been established by EPA: one based on ingestion of manganese in water and the other based on the ingestion of manganese in food (EPA 1999). The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references. The Food and Nutrition Board of the National Research Council (NRC 1989) determined an "adequate and safe" intake of manganese to be 2-5 mg/day for adults. This level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake. An RfD of 5×10^{-3} mg/kg-day for manganese in water is equivalent to a drinking water standard of 0.2 mg/L and is based on human chronic ingestion data (Kondakis et al. 1989). The RfD for ingestion of food is 1.4×10^{-1}

mg/kg-day and is also based on a NOAEL derived from human chronic ingestion data (NRC 1989; WHO 1973; Freeland-Graves et al. 1987). Because these RfDs are based on NOAELs identified in chronic human studies, no uncertainty factors were applied in calculating them (EPA 1999). EPA has assigned these chronic RfDs as surrogates for use with subchronic exposures without adjustment (EPA 1994).

The inhalation RfC for manganese is based on a LOAEL of 0.05 mg/m^3 as determined by Roels et al. (1992) (EPA 1999). An uncertainty factor of 1,000 reflects factors of 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less than chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of manganese. Thus, the RfC is 5×10^{-5} mg/m^3, which is equivalent to an inhalation RfD of 1.4×10^{-5} mg/kg-day. No cancer slope factors can be calculated for manganese at this time because of a lack of data.

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MERCURY

Inorganic mercury (Hg; CAS Registry number 7439-97-6) is a ubiquitous metallic element and one of the most widely studied toxicants.

Cancer

At present, EPA considers mercury to be a possible human carcinogen (Category C), based on an absence of human carcinogenicity data, inadequate evidence of carcinogenicity in animals, and inadequate genotoxicity data (USEPA 2001).

Animal carcinogenicity studies are briefly summarized by EPA (USEPA 2001):

> When 39 rats were injected *i.p.* over 2 weeks with metallic mercury and observed for their lifetimes, sarcomas were seen only in those tissues that had been in direct contact with the metal (Druckrey et al. 1957). No concurrent controls were reported.
>
> Mitsumori et al. (1981) fed groups of 60 male and 60 female SPF ICR mice 0, 15 or 30 ppm methyl mercury chloride in the diet for up to 78 weeks. The majority of the 30 ppm groups died from neurotoxicity by week 26. Histopathology on kidney tissue from all animals surviving after 53 weeks revealed renal tumors in 13 of 16 males in the 15 ppm group (2 adenomas, 11 adenocarcinomas). One adenoma was detected among 37 controls surviving to week 53 or beyond, and no tumors were seen in either control or exposed females. The possible presence of tumors at other sites was not reported in this preliminary communication.
>
> The relevance of data from studies of organic mercury to the possible carcinogenicity of inorganic mercury is uncertain.

Mutagenicity

Limited evidence has shown that exposure to mercury can cause adverse effects in the number or structure of chromosomes. In a comparison between four men exposed to mercury vapor and controls who were unexposed, the exposed group showed a statistical increase in the incidence of chromosomal aberrations in white blood cells (Popescu et al. 1979). Mabille et al. (1984), studied the chromosomal structure of occupationally exposed workers to mercury and did not find any significant increases in structural aberrations.

Methyl mercury hydroxide administered in the diet to *Drosophila melanogaster* at 5 mg/L induced chromosomal nondisjunction, while methyl and phenyl mercury produced small increases in the rate of point mutations (Ramel 1972).

Systemic Toxicity

Ingestion is one of the primary routes of exposure to mercury, but elemental mercury is only very poorly absorbed from the gastrointestinal tract (probably less than 0.01 percent) (Hammond and Beliles 1980). While CNS effects are the typical target organ effects observed following inhalation exposures, renal effects are the primary target of ingested inorganic mercury. In chronic exposures, nephrotoxicity is typically manifest as proteinuria; in severe cases, the nephrotic syndrome is observed, with subsequent edema and hypoproteinemia (Hammond and Beliles 1980).

The major toxic effects of inhaled mercury are primarily neurological. In acute exposure scenarios, clinical signs include paresthesia, ataxia, dysarthria, and deafness (Berlin 1979). Chronic exposure typically involves exposure to both mercury vapor and divalent mercury. Toxic symptoms include renal damage with nephrotic syndrome as well as increased salivation, inflammatory changes of the gums and the appearance of black lines along the gums (Skerfving and Vostal 1972).

In historical medicinal preparations, treatment with mercury compounds produced skin reactions such as erythema and dermatitis (Bhamra and Costa 1992). Other clinical signs include irritation, desquamation and loss of hair, ulcerations, hyperplasia, and hyperkeratosis (Bhamra and Costa 1992; Matheson et al. 1980).

Toxicokinetics

Exposure to mercury mainly occurs via inhalation and ingestion. Absorption of mercury from the respiratory and gastrointestinal tracts is dependent on its chemic form (Berlin 1979). Mercury vapor is very efficiently absorbed from the lungs, while elemental mercury is poorly absorbed from the gut (Bhamra and Costa 1992). After gastrointestinal absorption, elemental mercury is oxidized to a divalent form which accumulates mainly in the kidney and in part in the lung. Divalent mercury does not traverse the blood brain barrier as readily as mercury vapor. Inorganic mercury will also accumulate in the intestinal tract, skin, spleen, and testes, but to lesser degrees (Bhamra and Costa 1992). Elimination of mercury vapor is primarily by exhalation, with an estimated biological half-time of approximately 60 days (Hurst et al. 1976; Rohala et al. 1973), while mercury sequestered in the brain may take several years for a halving of retained mercury (Rossi et al. 1976). Divalent mercury, with an estimated biological half-time of 42 days, is primarily excreted in the urine and feces (Rohala et al. 1973).

Metabolism

Inorganic mercury is not known to be □metabolized□ or biotransformed, and behavior within the body would be essentially limited to absorption, distribution, potential sequestration, and excretion.

Toxicity Values

EPA has not established an oral reference dose for elemental mercury; however, for mercuric chloride, the oral RfD is 3×10^{-4} mg/kg-day based on subchronic feeding and

subcutaneous delivery studies in rats that produced LOAELS for autoimmune effects. (USEPA 2001). Neither elemental mercury nor mercuric chloride have slope factors calculated by EPA (USEPA 2001).

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SILVER

Humans are exposed to small amounts of silver from dietary sources. The oral intake of silver from a typical diet has been estimated to range from 27-88 μg/d (Kehoe et al. 1940). Tipton et al. (1966) estimated a lesser intake of 10B20 μg/d in two subjects during a 30-day observation period. Over a lifetime, a small but measurable amount of silver is accumulated by individuals having no excessive exposure. Gaul and Staud (1935) estimated that a person aged 50 years would have an average retention of 0.23-0.48 g silver.

Furchner et al. (1968) studied the absorption and retention of ingested silver in mice, rats, monkeys and dogs. In all four species, very little silver was absorbed from the GI tract. Cumulative excretion ranged from 90 to 99% on the second day after ingestion, with <1% of the dose being retained in monkeys, rats and mice. Dogs had a slightly greater retention. Furchner et al. (1968) estimated an equilibrium factor of 4.4% for ingested silver by humans. This estimate was considered to be a conservative estimate for the amount of silver which would be retained by a 70-kg human. This figure was rounded to 4% and was used in the dose conversion (i.v. dose converted to oral intake) for the calculation of the RfD.

Hepatic necrosis and ultrastructural changes of the liver have been induced by silver administration to vitamin E and/or selenium deficient rats (Wagner et al. 1975; Diplock et al. 1967; Bunyan et al. 1968). Investigators have hypothesized that this toxicity is related to a silver-induced selenium deficiency that inhibits the synthesis of the seleno-enzyme glutathione peroxidase. In animals supplemented with selenium and/or vitamin E, exposures of silver as high as 140 mg/kg-d (100 mg/L drinking water) were well-tolerated (Bunyan et al. 1968).

The critical effect in humans ingesting silver is argyria, a medically benign but permanent bluish-gray discoloration of the skin (USEPA 2001). Argyria results from the deposition of silver in the dermis and also from silver-induced production of melanin. Although silver has been shown to be uniformly deposited in exposed and unexposed areas, the increased pigmentation becomes more pronounced in areas exposed to sunlight due to photoactivated reduction of the metal. Although the deposition of silver is permanent, it is not associated with any adverse health effects.

Silver compounds have been employed for medical uses for centuries. In the nineteenth and early twentieth centuries, silver arsphenamine was used in the treatment of syphillis; more recently it has been used as an astringent in topical preparations. Gaul and Staud (1935) reported 70 cases of generalized argyria following organic and colloidal silver medication, including 13 cases of generalized argyria following intravenous silver arsphenamine injection therapy and a biospectrometric analysis of 10 cases of generalized argyria classified according to the quantity of silver present. In the i.v. study, data were presented for 10 males and two females who were administered 31 to 100 i.v. injections of silver arsphenamine (total dose was 4-20 g) over a 2 to 9.75 year period. Argyria developed after a total dose of 4, 7 or 8 g in some patients, while in others,

argyria did not develop until after a total dose of 10, 15 or 20 g. In the biospectrometric analysis of skin biopsies from 10 cases of generalized argyria, the authors confirmed that the degree of the discoloration is directly dependent on the amount of silver present. The authors concluded that argyria may become clinically apparent after a total accumulated i.v. dose of approximately 8 g of silver arsphenamine. However, since body accumulates silver throughout life, it is theoretically possible for amounts less than this (for example, 4 g silver arsphenamine) to result in argyria. Therefore, based on cases presented in this study, the lowest i.v. dose resulting in argyria in one patient, 1 g metallic silver (4 g silver arsphenamine) is considered to be a minimal effect level for this study.

Limited studies exist evaluating the effects of subchronic or chronic inhalation exposure of silver. ATSDR (1989b) notes that occupational exposure has lead to respiratory irritation and gastrointestinal distress (Rosenman et al. 1979, 1987) from exposure periods of 1 to 10 years. Actual exposure levels were not quantified, but a limited personal monitoring study associated with the Rosenman studies determined 8-hr time-weighted average concentrations ranging from 0.039 to 0.378 mg/m^3.

One study regarding subchronic or chronic dermal exposures to silver is discussed by ATSDR (1989b). Wahlberg (1965) exposed guinea pigs continuously for 8 weeks to a skin depot of silver nitrate, deriving a mortality NOAEL of 137.13 mg/kg-d, and a weight loss LOAEL at the same concentration. Other effects arising from chronic dermal exposure to silver include localized skin discoloration and allergic responses (ATSDR 1989b).

USEPA (2001) considers silver to be unclassifiable as a human carcinogen (Category D). In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas. No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years.

In about 700 untreated rats the rate of spontaneous tumor formation of any site was 1 to 3%. Furst and Schlauder (1977) evaluated silver and gold for carcinogenicity in a study designed to avoid solid-state carcinogenesis. Metal powder was suspended in trioctanoin and injected monthly intra-muscularly into Fischer 344 rats. The dose was 5 mg each for 5 treatments and 10 mg each for 5 more treatments, for a total dose of 75 mg silver. The treatment regimen included a vehicle control (a reportedly inert material), and cadmium as a positive control. Injection site sarcomas were found only in vehicle control (1/50), gold (1/50) and cadmium (30/50); no tumors (0/50) appeared at the site of injection in the silver-treated animals. A complete necropsy was performed on all animals. The authors mentioned the existence of spontaneous tumors in Fischer 344 rats, but reported only injection site tumors. They concluded that finely divided silver powder injected i.m. does not induce cancer.

No evidence of the mutagenicity of silver was shown in two available studies. Demerec et al. (1951) studied silver nitrate for the possible induction of back-mutations from

streptomycin dependence to nondependence in *Eschericha coli*. Silver nitrate was considered nonmutagenic in this assay. Nishioka (1975) screened silver chloride with other chemicals for mutagenic effects using a method called the *rec*-assay. Silver chloride was considered nonmutagenic in this assay.

Toxicity Values

USEPA (2001) has established an oral RfD for silver of 0.005 mg/kg-d based on argyria in humans exposed to silver intravenously (Gaul and Staud, 1935). This RfD is based upon a LOAEL of 1 g total dose of silver, converted to an oral dose of 0.014 mg/kg-d, and an aggregate uncertainty factor of 3. The uncertainty factor accounts for minimal effects in a subpopulation which has exhibited an increased propensity for the development of the cosmetic effect of argyria. A dermal RfD is calculated to be 0.001 mg/kg-day, based on the oral RfD multiplied by an absorption efficiency factor of 0.2 (USEPA 1996a).

USEPA has not established an inhalation RfC (USEPA 2001).

Argyria, the critical effect upon which the RfD for silver is based, occurs at levels of exposure much lower than those levels associated with other effects of silver. Argyrosis, resulting from the deposition of silver in the eye, has also been documented, but generally involves the use of eye drops or make-up containing silver (Greene and Su 1987). Silver has been found to be deposited in the cornea and the anterior capsule of the lens. The same deposition pattern was seen in the eyes of male Wistar rats following administration of a 0.66% silver nitrate solution to the eyes for 45 days (Rungby 1986). No toxicological effects were reported.

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VANADIUM

Vanadium (V; CAS Registry No. 7440-66-6) is a gray-to-lustrous-white elemental metal (pliable solid or powder) used primarily as an alloying agent in metals; as an ingredient in pesticides, dyes, and pigments; and as an industrial catalyst. Vanadium undergoes violent reactions in the presence of bromine trifluoride, chlorine, lithium, or oxidants; powdered vanadium explodes with chlorine even at 0°C. While functional roles for vanadium in humans have yet to be confirmed, evidence supports the concept that vanadium is an essential micronutrient for animals (French and Jones 1992).

Carcinogenicity

No studies regarding the carcinogenic effects of vanadium or vanadium compounds in humans or animals were located by ATSDR (1990) during that Agency's initial review of the toxicological literature. The International Agency for Research on Cancer reported negative experimental evidence of the carcinogenicity of metallic vanadium, vanadyl ions, and trivalent vanadium 2,4-pentanedione (Boffetta 1993). Leonard and Gerber (1994) noted the lack of data for a carcinogenic determination, but mentioned that vanadium is mitogenic with the concomitant potential for associated carcinogenicity (e.g., Ames and Gold 1990). In a study by Bishayee et al. (1997), ammonium vanadate appeared to modulate several factors associated with erythropoiesis under carcinogenic challenge by diethylnitrosamine. On the other hand, recent evidence indicates that vanadium is a tumor promoter when it is able to transactivate AP-1-dependent gene expression. With vanadium, AP-1 transactivation is dependent on the generation of O_2- and H_2O_2, but not OH (Ding et al. 1999).

Mutagenicity

In vitro genotoxicity assays have shown that vanadium or vanadium compounds are generally positive in bacterial (Kada et al. 1980; Kanematsu et al. 1980), yeast (Sora et al. 1986), rodent (Smith 1983), and human cell (Birnboim 1988; Hanauske et al. 1987) studies. However, there are no *in vivo* assays which have assessed the genotoxicity of vanadium compounds (as reported by ATSDR 1990). In 1994 Leonard and Gerber concluded that vanadium compounds are not clastogenic, but can be weakly mutagenic. In a Syrian hamster embryo assay (Kerckaert et al. 1996), vanadium pentoxide was negative with a 24-hr exposure, but positive with a 7-day exposure. This pattern of response (24-hr SHE negative/7-day SHE positive) has been seen with other chemicals which have tumor promotion-like characteristics.

Systemic Toxicity

Study volunteers were fed capsules containing 0.47-1.3 mg V (as ammonium vanadyl tartrate) per kilogram of body weight for a duration of 3 months. No hematological, hepatic, or renal effects were found. Subjects reported intestinal cramping and diarrhea (Dimond et al. 1963); but without concurrent experimental controls, the reported effects are not necessarily directly attributable to vanadium (ATSDR 1990).

The major effects in humans from exposure to vanadium vapors, aerosols, and dusts are irritant in nature, as observed from a variety of occupational epidemiological studies, case reports, and clinical studies. In general, irritation to the respiratory tract occurs at lower concentrations than to skin or eyes (Calabrese and Kenyon 1991). Workers exposed to vanadium compound dust have reported dry mouth, rhinitis, epistaxis, tracheitis, metallic taste, green tongue, and irritated eyes. One of the primary acute effects of vanadium exposure is peripheral vasoconstriction of lungs, spleen, kidneys, and intestines. Prolonged exposure can cause cardiac arrythmias and bradycardia.

Rodent mortality (LD_{50}) occurred at doses of approximately 30-40 mg V (as $NaVO_3$) kg^{-1} (Llobet and Domingo 1984), but chronic dietary exposures at 4.1 mg V (as $VOSO_4$) were not lethal (Schroeder and Balassa 1967; Schroeder et al. 1970). A three-month exposure to $NaVO_3$ in drinking water produced mononuclear cell infiltration in rat lungs, primarily in the perivascular region (Domingo et al. 1985). Cardiovascular effects in rodents have also been reported (Susic and Kentera 1986).

Exposure of male white rats (n=11 per exposure group) to a 70-day continuous fumigation with 0, 0.002, or 0.27 mg V (as V_2O_5) m^{-3} produced significant systemic effects in the high-exposure group that were not observed in the low-exposure group (Pazynich 1966). Effects included alterations in motor chronaxy, decreased oxyhemoglobin content, effects on leukocyte nuclei, and pathological conditions in several organ systems (lungs, liver, kidneys, and heart). ATSDR (1990) summarizes a variety of inhalation exposure studies, but presents quantitative exposure information only for acute studies; subchronic or chronic studies were apparently too deficient in detail to provide the Agency with quantitative toxicological information, and the effects presented in the studies are only cursorily discussed.

The effects of dermal exposure to vanadium or vanadium compounds appear to be largely unstudied. Dermal absorption and skin irritation were reported following the application to rabbit skin of a 20 percent solution of sodium metavanadate (Stokinger 1967); human skin absorption, however, may be very low (EPA 1977), as evidenced by a lack of skin penetration during an *in vitro* study using radiolabelled vanadium (Roshchin et al. 1980).

Toxicokinetics

For the general populace, exposures to vanadium compounds occur largely through food, while industrial workers are more commonly exposed to vanadium-containing dusts, fumes, and aerosols.

Vanadium is absorbed from a variety of foods with a relatively low efficiency, but in sufficient quantities to be stored at detectable levels in many body tissue (French and Jones 1992). Generally, less than 5 percent of the ingested dose is absorbed through the gastrointestinal tract (Byrne and Kosta 1978; Curran et al. 1959; Nielsen 1994), while airborne vanadium is absorbed very efficiently by the lungs (Boyd and Kustin 1985). The ICRP (1960) indicated that approximately 25 percent of soluble vanadium compounds may be absorbed via the respiratory tract. Although the body burden of

vanadium is typically very small (French and Jones 1992), the element distributes throughout the body with preferential accumulation usually observed in the liver, kidney, and bone (Byrne and Kosta 1978; Nechay et al. 1986; Mongold et al 1990). Blood is the medium for the distribution of vanadium, of which about 95 percent is bound to transferrin as the vanadyl ion (V^{+4} as VO^{+2}) (Patterson et al. 1986). Because of the low level of absorption via the gastrointestinal tract, the majority of ingested vanadium is excreted via the feces; absorbed vanadium is excreted primarily in the urine (IPCS 1988).

Metabolism

As an elemental molecule, vanadium *per se* is unmetabolizable, however, metabolic incorporations of vanadium have been studied. In the biological tissues, vanadium occurs largely as interconversions between two oxidation states: tetravalent vanadyl (V^{+4}) or pentavalent vanadate (V^{+5}) (ATSDR 1990). Within the organism, the role of vanadium has yet to be definitively understood, although the following effects occur under conditions of vanadium deficiency: increased abortion and perinatal death rates, decrease milk production, hepatic lipid and phospholipid changes, growth impairment (of bone, tooth, and cartilage), nutritional edema, thyroid metabolism changes, and depressed overall growth (French and Jones 1992). The widely varying pharmacological actions of vanadium have been poorly understood and are receiving increasing attention, particularly with respect to insulinomimetic properties (French and Jones 1992).

Toxicity Values

A 1953 chronic oral study in rats by Stokinger et al. was used by EPA to determine an oral RfD for vanadium pentoxide (EPA 1999). An unspecified number of rats were dosed via the diet with 17.85 or 178.5 ppm vanadium pentoxide for 2.5 years. Vanadium toxicity was evaluated by examining growth rate, survival and hair cystine content. The only significant effect was decreased hair cystine at the higher dose, and EPA extrapolated from a no-adverse-effect level of 17.85 ppm, converted to 0.89 mg/kg/day, to calculate a reference dose of 9×10^{-3}, or 0.009 mg/kg-day. An uncertainty factor of 100 was used (10 for interspecies extrapolation and 10 to allow for sensitive subpopulations); confidence in this RfD is low because of the lack of details reported in the study and the lack of available data on vanadium pentoxide.

EPA has not provided an inhalation RfC for vanadium or any of its compounds in the IRIS record. EPA has not classified vanadium as to its carcinogenic potential (EPA 1999). No oral or inhalation slope factors exist (EPA 1999).

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ZINC

Zinc (Zn) is a nutritionally essential trace element that also plays various industrial roles in galvanizing processes; paint formulations; and rubber, glass, or paper production. Because it does not rust, zinc provides a protective coating on other metals; it is also used in alloys such as bronze and brass to make electrical equipment, and with copper to make U.S. coins. Zinc salts are used in ceramics, textiles, batteries, and also in the pharmaceutical industry as a solubilizing agent in many drugs. Zinc is ubiquitous in the environment and in foods; it is released to the environment from such anthropogenic activities as mining, steel production, burning of coal and wastes, and from its own purification processes. Zinc is deleterious to human health in too little or too great quantities (HSDB 2000).

Systemic Toxicity

The toxic effects observed in humans following oral exposure to zinc as zinc oxide included abdominal pain, vomiting, anemia (ATSDR 1992), decreased HDL cholesterol (Chandra 1984; Hooper et al. 1980), and pancreatic damage (Chobanian 1981; Murphy 1970) at a dose greater than 10 times the recommended dietary allowance for zinc. High doses of Zn in humans cause decreased levels of hemoglobin and hematocrit, and similar effects were observed in other experimental animals species (ATSDR 1992). Ulceration of the forestomach and intestinal bleeding were reported for mice dosed with 1,110 mg Zn/kg-day and ferrets dosed with 390 mg/kg-day (Maita et al. 1981, Straube et al. 1980).

As a dietary requirement, oral exposure to zinc has been well-studied in animals and humans. With a variety of experimental durations (from >14 days to >1 year), adverse effects from zinc excess include mortality, gastrointestinal, hematological, musculoskeletal, hepatic, renal, immunological, and developmental changes (ATSDR 1992). In general, most of the animal NOAELS for these effects occur at daily ingestion of zinc ranging from 10 to 100 mg/kg, although dog NOAELs of 2 mg/kg-day for gastrointestinal and musculo-skeletal toxicity were obtained by Anderson and Danylchuk (1979) in a 9-month study of dogs given zinc in drinking water. For the same types of adverse effects, the typical range of human LOAELs and NOAELs is from 1 to 5 mg/kg-day, although Kynast and Saling (1986) report a NOAEL of 0.09 mg/kg-day for developmental effects in humans administered daily doses of zinc aspartate in capsules for 11 weeks duration.

Toxicity Values

EPA has established an oral reference dose (RfD) of 0.3 mg zinc (as a soluble salt) ingested per kilogram body weight per day, based on a decrease in erythrocytic superoxide dismutase content in adult human females after 10 weeks of zinc exposure (Yadrick et al. 1989). This RfDis based upon a dietary supplement LOAEL dosage of approximately 60 mg/kg, converted to 1 mg/kg-day, and by applying an aggregate uncertainty factor of 3. The uncertainty factor accounts for the use of a minimal LOAEL

from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient (USEPA 1999).

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Appendix C

Risk Calculations

TABLE C-1
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging time - Noncancer = AT	8760 days
Averaging Time - Cancer = AT	25550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	2.45E-06	8.40E-07	4.00E-04	NA	6.13E-03	--
BARIUM	1.24E+03	1.70E-03	5.82E-04	7.00E-02	NA	2.43E-02	--
CADMIUM	1.46E+02	2.00E-04	6.87E-05	5.00E-04	NA	4.01E-01	--
COPPER	2.05E+02	2.81E-04	9.65E-05	4.00E-02	NA	7.03E-03	--
MERCURY	4.12E-01	5.65E-07	1.94E-07	1.00E-04	NA	5.65E-03	--
SILVER	1.10E+04	1.51E-02	5.17E-03	5.00E-03	NA	3.01E+00	--
ZINC	5.47E+01	7.49E-05	2.57E-05	3.00E-01	NA	2.50E-04	--
Cumulative Risk						**3.46E+00**	**0.00E+00**

TABLE C-2
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	5,700	cm2/event
Adherence Factor = AF	0.07	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	350	event/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging time - Noncancer = AT	8,760	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.0E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	7.99E-07	1.50E-15	1.25E-05	NA	6.39E-02	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**6.39E-02**	**0.00E+00**

TABLE C-3
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	200 mg/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging time - Noncancer = AT	2190 days
Averaging Time - Cancer = AT	25550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	2.29E-05	1.96E-06	4.00E-04	NA	5.72E-02	--
BARIUM	1.24E+03	1.59E-02	1.36E-03	7.00E-02	NA	2.26E-01	--
CADMIUM	1.46E+02	1.87E-03	1.60E-04	5.00E-04	NA	3.74E+00	--
COPPER	2.05E+02	2.63E-03	2.25E-04	4.00E-02	NA	6.56E-02	--
MERCURY	4.12E-01	5.27E-06	4.52E-07	1.00E-04	NA	5.27E-02	--
SILVER	1.10E+04	1.41E-01	1.21E-02	5.00E-03	NA	2.81E+01	--
ZINC	5.47E+01	7.00E-04	6.00E-05	3.00E-01	NA	2.33E-03	--
Cumulative Risk						**3.23E+01**	**0.00E+00**

TABLE C-4
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	2,800	cm2/event
Adherence Factor = AF	0.20	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	350	event/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging time - Noncancer = AT	2,190	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.0E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	5.23E-06	4.49E-07	1.25E-05	NA	4.19E-01	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**4.19E-01**	**0.00E+00**

TABLE C-5
ESTIMATES OF CANCER AND NONCANCER RISKS FOR YOUTH TRESPASSERS DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	50 mg/day
Exposure Frequency = EF	117 day/yr
Exposure Duration = ED	10 yr
Body Weight = BW	50 kg
Averaging time - Noncancer = AT	3650 days
Averaging Time - Cancer = AT	25550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	5.74E-07	8.19E-08	4.00E-04	NA	1.43E-03	--
BARIUM	1.24E+03	3.97E-04	5.68E-05	7.00E-02	NA	5.68E-03	--
CADMIUM	1.46E+02	4.69E-05	6.70E-06	5.00E-04	NA	9.37E-02	--
COPPER	2.05E+02	6.58E-05	9.40E-06	4.00E-02	NA	1.65E-03	--
MERCURY	4.12E-01	1.32E-07	1.89E-08	1.00E-04	NA	1.32E-03	--
SILVER	1.10E+04	3.53E-03	5.04E-04	5.00E-03	NA	7.05E-01	--
ZINC	5.47E+01	1.75E-05	2.51E-06	3.00E-01	NA	5.85E-05	--
Cumulative Risk						**8.09E-01**	**0.00E+00**

TABLE C-6
ESTIMATES OF CANCER AND NONCANCER RISKS FOR YOUTH TRESPASSERS DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	4,690	cm2/event
Adherence Factor = AF	0.07	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	117	event/yr
Exposure Duration = ED	10	yr
Body Weight = BW	50	kg
Averaging Time (Noncancer) = AT	3,650	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	3.08E-07	4.40E-08	1.25E-05	NA	2.46E-02	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**2.46E-02**	**0.00E+00**

TABLE C-7
ESTIMATES OF CANCER AND NONCANCER RISKS FOR PARK GROUNDSKEEPER DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	250 day/yr
Exposure Duration = ED	25 yr
Body Weight = BW	70 kg
Averaging time - Noncancer = AT	9125 days
Averaging Time - Cancer = AT	25550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	1.75E-06	6.25E-07	4.00E-04	NA	4.38E-03	--
BARIUM	1.24E+03	1.21E-03	4.33E-04	7.00E-02	NA	1.73E-02	--
CADMIUM	1.46E+02	1.43E-04	5.11E-05	5.00E-04	NA	2.86E-01	--
COPPER	2.05E+02	2.01E-04	7.18E-05	4.00E-02	NA	5.02E-03	--
MERCURY	4.12E-01	4.03E-07	1.44E-07	1.00E-04	NA	4.03E-03	--
SILVER	1.10E+04	1.08E-02	3.84E-03	5.00E-03	NA	2.15E+00	--
ZINC	5.47E+01	5.35E-05	1.91E-05	3.00E-01	NA	1.78E-04	--
Cumulative Risk						**2.47E+00**	**0.00E+00**

TABLE C-8
ESTIMATES OF CANCER AND NONCANCER RISKS FOR PARK GROUNDSKEEPER DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	5,700	cm2/event
Adherence Factor = AF	0.07	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	250	event/yr
Exposure Duration = ED	25	yr
Body Weight = BW	70	kg
Averaging time - Noncancer = AT	9,125	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	5.71E-07	2.04E-07	1.25E-05	NA	4.57E-02	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**4.57E-02**	**0.00E+00**

TABLE C-9
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT PARK VISITORS DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	78 day/yr
Exposure Duration = ED	30 yr
Body Weight = BW	70 kg
Averaging time - Noncancer = AT	10,950 days
Averaging Time - Cancer = AT	25,550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	5.46E-07	2.34E-07	4.00E-04	NA	1.37E-03	--
BARIUM	1.24E+03	3.79E-04	1.62E-04	7.00E-02	NA	5.41E-03	--
CADMIUM	1.46E+02	4.46E-05	1.91E-05	5.00E-04	NA	8.93E-02	--
COPPER	2.05E+02	6.27E-05	2.69E-05	4.00E-02	NA	1.57E-03	--
MERCURY	4.12E-01	1.26E-07	5.39E-08	1.00E-04	NA	1.26E-03	--
SILVER	1.10E+04	3.36E-03	1.44E-03	5.00E-03	NA	6.72E-01	--
ZINC	5.47E+01	1.67E-05	7.16E-06	3.00E-01	NA	5.57E-05	--
Cumulative Risk						**7.71E-01**	**0.00E+00**

TABLE C-10
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT PARK VISITORS DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	5,700	cm2/event
Adherence Factor = AF	0.07	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	78	event/yr
Exposure Duration = ED	30	yr
Body Weight = BW	70	kg
Averaging time - Noncancer = AT	10,950	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	1.78E-07	7.63E-08	1.25E-05	NA	1.42E-02	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**1.42E-02**	**0.00E+00**

TABLE C-11
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD PARK VISITORS DUE TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	200 mg/day
Exposure Frequency = EF	78 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging time - Noncancer = AT	2,190 days
Averaging Time - Cancer = AT	25,550 days
Conversion Factor = CF	1.0E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	5.10E-06	4.37E-07	4.00E-04	NA	1.27E-02	--
BARIUM	1.24E+03	3.53E-03	3.03E-04	7.00E-02	NA	5.05E-02	--
CADMIUM	1.46E+02	4.17E-04	3.57E-05	5.00E-04	NA	8.33E-01	--
COPPER	2.05E+02	5.85E-04	5.02E-05	4.00E-02	NA	1.46E-02	--
MERCURY	4.12E-01	1.17E-06	1.01E-07	1.00E-04	NA	1.17E-02	--
SILVER	1.10E+04	3.13E-02	2.69E-03	5.00E-03	NA	6.27E+00	--
ZINC	5.47E+01	1.56E-04	1.34E-05	3.00E-01	NA	5.20E-04	--
Cumulative Risk						**7.19E+00**	**0.00E+00**

TABLE C-12
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD PARK VISITORS DUE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	2,800	cm2/event
Adherence Factor = AF	0.2	mg/cm2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	78	event/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging time - Noncancer = AT	2,190	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ANTIMONY	1.79E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
BARIUM	1.24E+03	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
CADMIUM	1.46E+02	1.17E-06	1.00E-07	1.25E-05	NA	9.33E-02	--
COPPER	2.05E+02	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MERCURY	4.12E-01	0.00E+00	0.00E+00	1.00E-04	NA	0.00E+00	--
SILVER	1.10E+04	0.00E+00	0.00E+00	2.00E-04	NA	0.00E+00	--
ZINC	5.47E+01	0.00E+00	0.00E+00	3.00E-01	NA	0.00E+00	--
Cumulative Risk						**9.33E-02**	**0.00E+00**

TABLE C-13
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Ingestion of Groundwater**

Ingestion Rate = CR	2 L/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging time - Noncancer = AT	8760 days
Averaging Time - Cancer = AT	25550 days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ALUMINUM	1.94E+00	5.31E-02	1.82E-02	1.00E+00	NA	5.31E-02	--
CADMIUM	1.36E-01	3.73E-03	1.28E-03	5.00E-04	NA	7.45E+00	--
COBALT	7.88E-03	2.16E-04	7.40E-05	2.00E-02	NA	1.08E-02	--
MANGANESE	1.13E+00	3.10E-02	1.06E-02	2.00E-02	NA	1.55E+00	--
VANADIUM	8.49E-03	2.33E-04	7.97E-05	7.00E-03	NA	3.32E-02	--
Cumulative Risk						**9.10E+00**	**0.00E+00**

TABLE C-14
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Groundwater**

Surface Area for Contact = SA	18,000	cm2
Event Time = ET	0.20	hr/day
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging time - Noncancer = AT	8,760	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.0E-03	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ALUMINUM	1.94E+00	9.55E-05	3.28E-05	1.00E+00	NA	9.55E-05	--
CADMIUM	1.36E-01	6.71E-06	2.30E-06	1.25E-05	NA	5.37E-01	--
COBALT	7.88E-03	3.89E-07	1.33E-07	2.00E-02	NA	1.94E-05	--
MANGANESE	1.13E+00	5.59E-05	1.91E-05	8.00E-04	NA	6.98E-02	--
VANADIUM	8.49E-03	4.19E-07	1.44E-07	1.82E-04	NA	2.30E-03	--
Cumulative Risk						**6.09E-01**	**0.00E+00**

TABLE C-15
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Ingestion of Groundwater**

Ingestion Rate = CR	2 L/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging time - Noncancer = AT	2190 days
Averaging Time - Cancer = AT	25550 days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ALUMINUM	1.94E+00	2.48E-01	2.12E-02	1.00E+00	NA	2.48E-01	--
CADMIUM	1.36E-01	1.74E-02	1.49E-03	5.00E-04	NA	3.48E+01	--
COBALT	7.88E-03	1.01E-03	8.64E-05	2.00E-02	NA	5.04E-02	--
MANGANESE	1.13E+00	1.45E-01	1.24E-02	2.00E-02	NA	7.24E+00	--
VANADIUM	8.49E-03	1.09E-03	9.30E-05	7.00E-03	NA	1.55E-01	--
Cumulative Risk						**4.25E+01**	**0.00E+00**

TABLE C-16
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

*** RME- Dermal Contact with Groundwater**

Surface Area for Contact = SA	6,600	cm2
Event Time = ET	0.2	hr/day
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging time - Noncancer = AT	2,190	days
Averaging Time - Cancer = AT	25,550	days
Conversion Factor = CF	1.0E-03	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ALUMINUM	1.94E+00	1.63E-04	1.40E-05	1.00E+00	NA	1.63E-04	--
CADMIUM	1.36E-01	1.15E-05	9.84E-07	1.25E-05	NA	9.18E-01	--
COBALT	7.88E-03	6.65E-07	5.70E-08	2.00E-02	NA	3.33E-05	--
MANGANESE	1.13E+00	9.56E-05	8.19E-06	8.00E-04	NA	1.19E-01	--
VANADIUM	8.49E-03	7.16E-07	6.14E-08	1.82E-04	NA	3.94E-03	--
Cumulative Risk						**1.04E+00**	**0.00E+00**

Appendix D

Risk Assessment Guidance for Superfund
D Tables

TABLE D-1.1
SELECTION OF EXPOSURE PATHWAYS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Soil	Surface Soil	On-site	Resident	Adult	Ingestion	On-Site	Quant	Future residents are may contact surface soil.
						Dermal	On-Site	Quant	Future residents are may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
					Child	Ingestion	On-Site	Quant	Future residents are may contact surface soil.
						Dermal	On-Site	Quant	Future residents are may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
			On-site	Park Visitor	Adult	Ingestion	On-Site	Quant	Future park visitors may contact surface soil.
						Dermal	On-Site	Quant	Future park visitors may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
					Child	Ingestion	On-Site	Quant	Future park visitors may contact surface soil.
						Dermal	On-Site	Quant	Future park visitors may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
			On-site	Trespasser	Youth	Ingestion	On-Site	Quant	Future trespassers may contact surface soil.
						Dermal	On-Site	Quant	Future trespassers may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
		Subsurface Soil	On-site	Resident	Adult	Ingestion	On-Site	Qual	Future residents are may contact subsurface soil during gardening or other yard activities.
						Dermal	On-Site	Qual	Future residents are may contact subsurface soil during gardening or other yard activities.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
					Child	Ingestion	On-Site	Qual	Future residents are may contact subsurface soil during gardening or other yard activities.
						Dermal	On-Site	Qual	Future residents are may contact subsurface soil during gardening or other yard activities.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
			On-site	Groundskeeper	Adult	Ingestion	On-Site	Qual	Future groundskeepers may contact subsurface soil.
						Dermal	On-Site	Qual	Future groundskeepers may contact subsurface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
			On-site	Groundskeeper	Adult	Ingestion	On-Site	Quant	Future groundskeepers may contact surface soil.
						Dermal	On-Site	Quant	Future groundskeepers may contact surface soil.
						Inhalation	On-Site	None	Inhalation is a negligable pathway based on lack of volatiles.
	Ground Water	Ground Water	On-site	Resident	Adult	Ingestion	On-Site	Quant	Future residents may ingest ground water as tap water if not connected to public water.
						Dermal	On-Site	Quant	Future residents may contact ground water if not connected to public water.
						Inhalation	On-Site	None	Volatiles are not of concern at this site.
					Child	Ingestion	On-Site	Quant	Future residents may ingest ground water as tap water if not connected to public water.
						Dermal	On-Site	Quant	Future residents may contact ground water if not connected to public water.
						Inhalation	On-Site	None	Volatiles are not of concern at this site.
			On-site	Park Visitor	Adult	Ingestion	On-Site	None	It is unlikely that park visitors would contact groundwater.
						Dermal	On-Site	None	It is unlikely that park visitors would contact groundwater.
					Child	Ingestion	On-Site	None	It is unlikely that park visitors would contact groundwater.
						Dermal	On-Site	None	It is unlikely that park visitors would contact groundwater.
			On-site	Trespasser	Youth	Ingestion	On-Site	None	It is unlikely that trespassers would contact groundwater.
						Dermal	On-Site	None	It is unlikely that trespassers would contact groundwater.
			On-site	Park Groundskeeper	Adult	Ingestion	On-Site	Quant	Groundskeepers may ingest groundwater as tap water if not connected to public water.
						Dermal	On-Site	Qual	Groundskeepers may contact groundwater during irrigation activities.
						Inhalation	On-Site	None	Volatiles are not of concern at this site.

TABLE D-2.1
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK
ON-SITE - SURFACE SOIL

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products (On-site)

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value		Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							Inorganics										
7429-90-5	Aluminum	511.5		10200		mg/kg	SB-1	14/14	113 - 113	10200	N/A	ND	N	N/A	N/A	Yes	ASL
7440-36-0	Antimony	5	J	5	J	mg/kg	SB-1	1/14	2.4 - 2.7	5	N/A	ND	N	N/A	N/A	Yes	ASL
7440-38-2	Arsenic	0.69	J	2.4	J / J	mg/kg	SB-1 / SB-12	9/14	0.4 - 0.42	2.4	N/A	7.50E+00	C	N/A	N/A	No	BSL
7440-39-3	Barium	6.25	B/B	1240		mg/kg	B-2	14/14	0.42 - 0.42	1240	N/A	3.00E+02	N	N/A	N/A	Yes	ASL
7440-41-7	Beryllium	0.31	B	0.35	B	mg/kg	SB-1	2/14	0.2 - 0.215	0.35	N/A	1.60E-01	N	N/A	N/A	Yes	ASL
7440-43-9	Cadmium	0.39	B	435		mg/kg	SB-6F-3	47/72	0.205 - 1.6	435	N/A	1.00E+00	N	N/A	N/A	Yes	ASL
7440-70-2	Calcium	160	B	24700		mg/kg	SB-3	11/14	120 - 175	24700	N/A	ND		N/A	N/A	N/A	NUT
7440-47-3	Chromium	2.8		9.8		mg/kg	SB-1	14/14	175 - 175	9.8	N/A	1.00E+01	N	N/A	N/A	No	BSL
7440-48-4	Cobalt	0.53	B	21.75		mg/kg	SB-20	12/14	0.4 - 0.4	21.75	N/A	3.00E+01	N	N/A	N/A	No	BSL
7440-50-8	Copper	3.5	B	496	J	mg/kg	SB-12	13/14	6.4 - 6.4	496	N/A	2.50E+01	N	N/A	N/A	Yes	ASL
7439-89-6	Iron	863		10800		mg/kg	SB-1	14/14	0.11 - 0.11	10800	N/A	ND		N/A	N/A	No	NUT
7439-92-1	Lead	4.2	J/J	45.8		mg/kg	B-7	9/14	4.4 - 5.5	45.8	N/A	4.00E+02		N/A	N/A	No	BSL
7439-95-4	Magnesium	104.2	B/B	14900		mg/kg	SB-3	14/14		14900	N/A	ND		N/A	N/A	N/A	NUT
7439-96-5	Manganese	6.2		81.3		mg/kg	SB-3	14/14	5.5 - 5.5	81.3	N/A	ND	N	N/A	N/A	Yes	ASL
7439-97-6	Mercury	0.015		2.41		mg/kg	SB-6F-1	24/37	0.0128 - 0.4	2.41	N/A	1.00E-01		N/A	N/A	Yes	ASL
7440-02-0	Nickel	2	B	6.6	B	mg/kg	SB-1	7/14	2.4 - 4.9	6.6	N/A	1.30E+01	N	N/A	N/A	No	BSL
7440-09-7	Potassium	335	B	406	B	mg/kg	SB-3	5/14	342.4 - 434	406	N/A	ND		N/A	N/A	N/A	NUT
7782-49-2	Selenium	0.3	B	0.4	B	mg/kg	B-7	2/14	0.2 - 0.4	0.4	N/A	2.00E+00	N	N/A	N/A	No	BSL
7440-22-4	Silver	0.23	B	11000		mg/kg	SB-6F-1	56/71	0.22 - 351	11000	N/A	ND	N	N/A	N/A	Yes	ASL
7440-23-5	Sodium	46.9	B	63.8	B	mg/kg	SB-3	2/14	20.5 - 37.1	63.8	N/A	ND		N/A	N/A	N/A	NUT
7440-62-2	Vanadium	1.85	B/B	19.7		mg/kg	SB-1	14/14	3 - 3	19.7	N/A	1.50E+02	N	N/A	N/A	No	BSL
7440-66-6	Zinc	12		69		mg/kg	SB-12	9/14	5.7 - 25.3	69	N/A	2.00E+01	N	N/A	N/A	Yes	ASL

(1) Minimum/maximum detected concentration.

(2) Maximum concentration used as screening value.

(3) Screening Toxicity Value - Taken from New York State Technical and Administrative Guidance Memorandum (TAGM) Recommended Soil Cleanup Objectives (RSCO) (NYSDEC 1994b)

(4) Rationale Codes

Selection Reason: Above Screening Toxicity (ASL)

Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions: N/A = Not Applicable
ND = No Data
COPC = Chemical of Potential Concern

N = Noncarcinogen; C = Carcinogen
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered

Qualifiers: B=Reported value is less than the Reporting Limit but greater than the Instrument Detection Limit or Method Detection Limit
J=Indicates an estimated value.

TABLE D-2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK
GROUNDWATER

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Peerless Photo Products

CAS Number	Chemical	Minimum [1] Concentration	Minimum Qualifier	Maximum [1] Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration [2] Used for Screening	Background Value	Screening [3] Toxicity Value		Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for [4] Contaminant Deletion or Selection
							Inorganics										
7429-90-5	Aluminum	95.5	B	4880		ug/L	MW-1	26/34	72.3 - 409	4880	N/A	ND	N	N/A	N/A	Yes	ASL
7440-36-0	Antimony	14.4	B	16.6	B/B	ug/L	MW-9	5/31	3 - 12	16.6	N/A	3.00E+00	N	N/A	N/A	Yes	ASL
7440-38-2	Arsenic	4.3	J	7	J	ug/L	MW-1	3/31	2 - 5	7	N/A	2.50E+01	C	N/A	N/A	No	BSL
7440-39-3	Barium	13.6	B	329		ug/L	MW-1	33/34	7.9 - 7.9	329	N/A	1.00E+03	N	N/A	N/A	No	BSL
7440-41-7	Beryllium	1.1	B	1.35	B/B	ug/L	MW-9	2/15	1 - 1	1.35	N/A	3.00E+00	N	N/A	N/A	No	BSL
7440-43-9	Cadmium	0.68	B	269		ug/L	MW-6	64/80	0.05 - 3	269	N/A	5.00E+00	N	N/A	N/A	Yes	ASL
7440-70-2	Calcium	5020		51300		ug/L	MW-1	34/34	0.05 - 0.05	51300	N/A	ND		N/A	N/A	N/A	NUT
7440-47-3	Chromium	2.1	B / B	72.3		ug/L	MW-2	55/84	1 - 11.1	72.3	N/A	5.00E+01	N	N/A	N/A	Yes	ASL
7440-48-4	Cobalt	1	B	23.95	B/B	ug/L	MW-9	18/30	1 - 4	23.95	N/A	ND	N	N/A	N/A	Yes	ASL
7440-50-8	Copper	2.3	B	35.2		ug/L	MW-1	22/32	1 - 9	35.2	N/A	2.00E+02	N	N/A	N/A	No	BSL
7439-89-6	Iron	79	B	14800		ug/L	MW-1	31/34	70.9 - 199	14800	N/A	ND		N/A	N/A	Yes	NUT
7439-92-1	Lead	1.8	B	34		ug/L	MW-1	39/74	1.6 - 26.3	34	N/A	2.50E+01	N	N/A	N/A	Yes	ASL
7439-95-4	Magnesium	1520	B	7540		ug/L	MW-7S	34/34	ND	7540	N/A	3.50E+04		N/A	N/A	No	NUT
7439-96-5	Manganese	5.7	B	1680		ug/L	MW-1	32/34	1.6 - 172	1680	N/A	3.00E+02	N	N/A	N/A	Yes	ASL
7439-97-6	Mercury	0.07	B	0.19	J	ug/L	MW-1	3/68	0.05 - 0.5	0.19	N/A	7.00E-01	N	N/A	N/A	No	BSL
7440-02-0	Nickel	5.1	B	50.1		ug/L	MW-1	18/31	2.8 - 12	50.1	N/A	1.00E+02	N	N/A	N/A	No	BSL
7440-09-7	Potassium	823	B	12700		ug/L	MW-4	30/34	12 - 1710	12700	N/A	ND		N/A	N/A	N/A	NUT
7440-22-4	Silver	1	B	6.3	B	ug/L	MW-9	14/57	1 - 2	6.3	N/A	5.00E+01	N	N/A	N/A	No	BSL
7440-23-5	Sodium	1100		31100		ug/L	MW-4	34/34	2 - 2	31100	N/A	2.00E+03		N/A	N/A	Yes	NUT
7440-62-2	Vanadium	1.1	B	18	B	ug/L	MW-1	18/30	1 - 3	18	N/A	ND	N	N/A	N/A	Yes	ASL
7440-66-6	Zinc	20.8		423		ug/L	MW-4	24/34	19.6 - 65.6	423	N/A	2.00E+03	N	N/A	N/A	No	BSL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from New York State Water Quality Standards for Class GA Ground Water (NYS GQS) (NYSDEC 1998)
(4) Rationale Codes

Selection Reason: Above Screening Toxicity (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data
COPC = Chemical of Potential Concern
N = Noncarcinogen; C = Carcinogen
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered

Qualifiers:
B=Reported value is less than the Reporting Limit but greater than the Instrument Detection Limit or Method Detection Limit.
J=Indicates an estimated value.

TABLE D-3.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK
ON-SITE - SURFACE SOIL

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products (On-site)

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Inorganics												
Aluminum*	mg/kg	3.41E+03	7.56E+03	1.02E+04		mg/kg	7.56E+03	95% UCL_T		3.41E+03	Mean	Regional Guidance
Antimony	mg/kg	1.50E+00	1.79E+00	5.00E+00	J	mg/kg	1.79E+00	95% UCL_T		1.50E+00	Mean	Regional Guidance
Barium	mg/kg	2.05E+02	1.90E+03	1.24E+03		mg/kg	1.24E+03	Max		2.05E+02	Mean	Regional Guidance
Beryllium*	mg/kg	1.33E-01	1.67E-01	3.50E-01	B	mg/kg	1.67E-01	95% UCL_T		1.33E-01	Mean	Regional Guidance
Cadmium	mg/kg	2.97E+01	1.46E+02	4.35E+02		mg/kg	1.46E+02	95% UCL_T		2.97E+01	Mean	Regional Guidance
Copper	mg/kg	5.88E+01	2.05E+02	4.96E+02	J	mg/kg	2.05E+02	95% UCL_T		5.88E+01	Mean	Regional Guidance
Manganese*	mg/kg	3.77E+01	6.50E+01	8.13E+01		mg/kg	6.50E+01	95% UCL_T		3.77E+01	Mean	Regional Guidance
Mercury	mg/kg	1.87E-01	4.12E-01	2.41E+00		mg/kg	4.12E-01	95% UCL_T		1.87E-01	Mean	Regional Guidance
Silver	mg/kg	7.03E+02	1.44E+05	1.10E+04		mg/kg	1.10E+04	Max		7.03E+02	Mean	Regional Guidance
Zinc	mg/kg	2.05E+01	5.47E+01	6.90E+01		mg/kg	5.47E+01	95% UCL_T		2.05E+01	Mean	Regional Guidance

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Normal Data (Mean).
* = Eliminated as a COPC based on background. See Table D-3.3.
(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.

TABLE D-3.2
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK
GROUNDWATER

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Peerless Photo Products

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Inorganics												
Aluminum	ug/L	9.74E+02	1.94E+03	4880		ug/L	1.94E+03	95% UCL_T		9.74E+02	Mean	Regional Guidance
Antimony*	ug/L	5.41E+00	7.39E+00	16.6	B/B	ug/L	7.39E+00	95% UCL_T		5.41E+00	Mean	Regional Guidance
Cadmium	ug/L	3.42E+01	1.36E+02	269		ug/L	1.36E+02	95% UCL_T		3.42E+01	Mean	Regional Guidance
Chromium*	ug/L	9.37E+00	1.26E+01	72.3		ug/L	1.26E+01	95% UCL_T		9.37E+00	Mean	Regional Guidance
Cobalt	ug/L	4.43E+00	7.88E+00	23.95	B/B	ug/L	7.88E+00	95% UCL_T		4.43E+00	Mean	Regional Guidance
Lead*	ug/L	5.16E+00	6.51E+00	34		ug/L	6.51E+00	95% UCL_T		5.16E+00	Mean	Regional Guidance
Manganese	ug/L	3.75E+02	1.13E+03	1680		ug/L	1.13E+03	95% UCL_T		3.75E+02	Mean	Regional Guidance
Vanadium	ug/L	4.42E+00	8.49E+00	18	B	ug/L	8.49E+00	95% UCL_T		4.42E+00	Mean	Regional Guidance

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Normal Data (Mean).
* = Eliminated as a COPC based on background. See Table D-3.4. Lead was still evaluated as a COPC as a conservative measure.
(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.

TABLE D-3.3
SUMMARY OF THE COMPARISON OF ON-SITE SURFACE SOIL DATA TO BACKGROUND DATA
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Site	Analyte	Units	Background		Site RME Medium EPC Value	COPC	Reason
			Max	Max + 10%			
on-site	Aluminum	mg/kg	8990	9889	7.56E+03	No	maximum background concentration plus 10% > RME Medium EPC Value
on-site	Antimony	mg/kg	0.35	0.385	1.79E+00	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Barium	mg/kg	26.1	28.71	1.24E+03	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Beryllium	mg/kg	0.32	0.352	1.67E-01	No	maximum background concentration plus 10% > RME Medium EPC Value
on-site	Cadmium	mg/kg	0.55	0.605	1.46E+02	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Copper	mg/kg	15.6	17.16	2.05E+02	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Manganese	mg/kg	89.5	98.45	6.50E+01	No	maximum background concentration plus 10% > RME Medium EPC Value
on-site	Mercury	mg/kg	0.07	0.077	4.12E-01	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Silver	mg/kg	72.1	79.31	1.10E+04	Yes	maximum background concentration plus 10% < RME Medium EPC Value
on-site	Zinc	mg/kg	25.6	28.16	5.47E+01	Yes	maximum background concentration plus 10% < RME Medium EPC Value

TABLE D-3.4
SUMMARY OF THE COMPARISON OF GROUNDWATER DATA TO BACKGROUND DATA
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Site	Analyte	Units	Background			Site RME	COPC	Reason
			Mean	Standard Deviation	Mean + 2*STDEV	Medium EPC Value		
Monitoring Well	Aluminum	ug/L	291.625	132.3879243	556.4008486	1.94E+03	Yes	mean plus two standard deviations < RME Medium EPC Value
Monitoring Well	Antimony	ug/L	7.525	8.264532655	24.05406531	7.39E+00	No	mean plus two standard deviations > RME Medium EPC Value
Monitoring Well	Cadmium	ug/L	1.86	1.505988048	4.871976096	1.36E+02	Yes	mean plus two standard deviations < RME Medium EPC Value
Monitoring Well	Chromium	ug/L	4.48	5.610436703	15.70087341	1.26E+01	No	mean plus two standard deviations > RME Medium EPC Value
Monitoring Well	Cobalt	ug/L	1.425	1.534872416	4.494744832	7.88E+00	Yes	mean plus two standard deviations < RME Medium EPC Value
Monitoring Well	Lead	ug/L	6.375	8.90487928	24.18475856	6.51E+00	No	mean plus two standard deviations > RME Medium EPC Value
Monitoring Well	Manganese	ug/L	148.3	125.1205818	398.5411637	1.13E+03	Yes	mean plus two standard deviations < RME Medium EPC Value
Monitoring Well	Vanadium	ug/L	1.55	1.508862706	4.567725413	8.49E+00	Yes	mean plus two standard deviations < RME Medium EPC Value

TABLE D-4.1
VALUES USED FOR RESIDENT ADULT DAILY SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	100	U.S. EPA 1989
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED-NC	Exposure Duration = ED	yr	24	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	8,760	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	5,700	U.S. EPA 2001
	AF	Adherence Factor = AF	mg/cm2	0.07	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	350	U.S. EPA 1997
	ED-NC	Exposure Duration = ED	yr	24	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	8,760	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.2
VALUES USED FOR RESIDENT CHILD DAILY SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	200	U.S. EPA 1989
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	2,800	U.S. EPA 2001
	AF	Adherence Factor = AF	mg/cm2	0.2	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	350	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.3
VALUES USED FOR ADULT PARK VISITOR SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Park Visitor
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	100	U.S. EPA 1989
	EF	Exposure Frequency = EF	day/yr	78	BPJ
	ED	Exposure Duration = ED	yr	30	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	10,950	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	5,700	U.S. EPA 1992
	AF	Adherence Factor = AF	mg/cm2	0.07	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	78	BPJ
	ED	Exposure Duration = ED	yr	30	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	10,950	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.4
VALUES USED FOR CHILD PARK VISITOR SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Park Visitor
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	200	U.S. EPA 1989a
	EF	Exposure Frequency = EF	day/yr	78	BPJ
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	2,800	U.S. EPA 1992
	AF	Adherence Factor = AF	mg/cm2	0.2	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	78	BPJ
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.5
VALUES USED FOR YOUTH TRESPASSER SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Youth Trespasser
Receptor Age: Youth (9-18 years)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	50	U.S. EPA 1991
	EF	Exposure Frequency = EF	day/yr	117	BPJ
	ED	Exposure Duration = ED	yr	10	U.S. EPA 1991
	BW	Body Weight = BW	kg	50	U.S. EPA 1990
	AT-NC	Averaging time - Noncancer = AT	days	3,650	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	4,690	U.S. EPA 1992
	AF	Adherence Factor = AF	mg/cm2	0.07	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	117	BPJ
	ED	Exposure Duration = ED	yr	10	U.S. EPA 1991
	BW	Body Weight = BW	kg	50	U.S. EPA 1990
	AT-NC	Averaging time - Noncancer = AT	days	3,650	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.6
VALUES USED FOR PARK GROUNDSKEEPER SURFACE SOIL INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Peerless Photo Products Site
Receptor Population: Park Groundskeeper
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	mg/day	100	U.S. EPA 1991
	EF	Exposure Frequency = EF	day/yr	250	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	25	U.S. EPA 1991
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	9,125	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2/event	5,700	U.S. EPA 1992
	AF	Adherence Factor = AF	mg/cm2	0.07	U.S. EPA 1992
	EF	Exposure Frequency = EF	event/yr	250	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	25	U.S. EPA 1991
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	9,125	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	kg/mg	1.00E-06	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.7
VALUES USED FOR RESIDENT ADULT INCIDENTAL GROUNDWATER INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Peerless Photo Products Site
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	L/day	2	U.S. EPA 1991
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED-NC	Exposure Duration = ED	yr	24	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	8,760	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2	18,000	U.S. EPA 1992
	PC	Permeability Coefficient	cm/hr	chemical-specific	
	ET	Event Time = ET	hr/day	0.20	U.S. EPA 1992
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED-NC	Exposure Duration = ED	yr	24	U.S. EPA 1989
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	8,760	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	L/cm3	1.00E-03	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.8
VALUES USED FOR RESIDENT CHILD INCIDENTAL GROUNDWATER INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Peerless Photo Products Site
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	L/day	2	U.S. EPA 1991
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
Dermal	SA	Surface Area for Contact = SA	cm2	6,600	U.S. EPA 1992
	PC	Permeability Coefficient	cm/hr	chemical-specific	
	ET	Event Time = ET	hr/day	0.2	U.S. EPA 1992
	EF	Exposure Frequency = EF	day/yr	350	U.S. EPA 1997
	ED	Exposure Duration = ED	yr	6	U.S. EPA 1991
	BW	Body Weight = BW	kg	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	2,190	U.S. EPA 1991
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989
	CF	Conversion Factor = CF	L/cm3	0.001	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-4.9
VALUES USED FOR PARK GROUNDSKEEPER INCIDENTAL GROUNDWATER INTAKE EQUATIONS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Peerless Photo Products Site
Receptor Population: Park Groundskeeper
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference
Ingestion	CR	Ingestion Rate = CR	L/day	1	U.S. EPA 1991
	EF	Exposure Frequency = EF	day/yr	250	U.S. EPA 1997
	ED-NC	Exposure Duration = ED	yr	25	U.S. EPA 1991
	BW	Body Weight = BW	kg	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer = AT	days	8,760	U.S. EPA 1989
	AT-C	Averaging Time - Cancer = AT	days	25,550	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

TABLE D-5
NON-CANCER TOXICITY DATA - ORAL//DERMAL
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Value (mg/kg-day)	Oral to Dermal Adjustment Factor (GI ABS) (1)	Adjusted Dermal RfD (2) (mg/kg bw-day)	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD: Target Organ	Dates of RfD: Target Organ (3) (mm/dd/yy)
Inorganics								
ALUMINUM	Subchronic	1.00E+00	1	1.00E+00	Central Nervous System	100/3	EPA-NCEA	5/30/1997
ANTIMONY	Chronic	4.00E-04	0.15	6.00E-05	Blood glucose and cholesterol	1000/1	IRIS	3/21/2003
BARIUM	Subchronic	7.00E-02	0.07	4.90E-03	Kidneys	3/1	IRIS	3/21/2003
CADMIUM	Chronic	5.00E-04	0.025	1.25E-05	Kidneys	10/1	IRIS	3/21/2003
COBALT	Chronic	2.00E-02	1	2.00E-02	Respiratory System	NA/NA	EPA-NCEA	8/7/2001
COPPER	Chronic	4.00E-02	1	4.00E-02	Gastrointestional System	None	HEAST	7/25/1997
MANGANESE	Chronic	2.00E-02	0.04	8.00E-04	Central Nervous System	1/1	IRIS	3/21/2003
MERCURY	Chronic	1.00E-04	1	1.00E-04	None	30/1	IRIS	3/21/2003
SILVER	Subchronic	5.00E-03	0.04	2.00E-04	Skin	3/1	IRIS	3/21/2003
VANADIUM	Chronic	7.00E-03	0.026	1.82E-04	Gastrointestinal System, Kidney	100/1	HEAST	7/25/1997
ZINC	Chronic	3.00E-01	1	3.00E-01	Blood	3/1	IRIS	3/21/2003

N/A= Not Applicable

(1) Taken from USEPA 2000 Guidance. USEPA. 2000. *Risk Assessment Guidance for Superfund, volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*. Interim Guidance.

(2) Dermal toxicological values adjusted from oral values using USEPA 2000 recommended chemical-specific gastrointestinal absorption factors(GI ABS). RfDs are multiplied by the GI ABS.

(3) IRIS - Integrated Risk Information System. For IRIS values, the date IRIS was searched is provided.
HEAST - Health Effects Assessment Summary Tables. For HEAST values, the date of HEAST is provided.
EPA-NCEA - National Center for Environmental Assessment. For EPA-NCEA values, the date of the article provided by EPA-NCEA is provided.

TABLE D-6
CANCER TOXICITY DATA - ORAL/DERMAL
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Chemical of Potential Concern	Oral Cancer Slope Factor	Oral to Dermal Adjustment Factor (GI ABS)[1]	Adjusted Cancer Slope Factor [2]	Units	Weight of Evidence/Cancer Guideline Description	Source	Date [3] (mm/dd/yy)
Inorganics							
ALUMINUM	NA	1	NA	per (mg/kg-day)	D	EPA-NCEA	5/30/1997
ANTIMONY	NA	0.15	NA	per (mg/kg-day)	NA	IRIS	3/21/2003
BARIUM	NA	0.07	NA	per (mg/kg-day)	D	IRIS	3/21/2003
CADMIUM	NA	0.025	NA	per (mg/kg-day)	B1	IRIS	3/21/2003
COBALT	NA	1	NA	per (mg/kg-day)	NA	EPA-NCEA	8/7/2001
COPPER	NA	1	NA	per (mg/kg-day)	D	IRIS	3/21/2003
MANGANESE	NA	0.04	NA	per (mg/kg-day)	D	IRIS	3/21/2003
MERCURY	NA	1	NA	per (mg/kg-day)	D	IRIS	3/21/2003
SILVER	NA	0.04	NA	per (mg/kg-day)	D	IRIS	3/21/2003
VANADIUM	NA	0.026	NA	per (mg/kg-day)	D	HEAST	7/25/1997
ZINC	NA	1	NA	per (mg/kg-day)	D	IRIS	3/21/2003

N/A= Not Applicable

(1) Taken from USEPA 2000 Guidance.

(2) Dermal Toxicological values adjusted from oral values using USEPA 2000 recommended chemical-specific gastrointestinal absorption factors(GI ABS). CSFs are divided be the GI ABS.

(3) For IRIS values, the date IRIS was searched is provided.
For HEAST values, the date of HEAST is provided.
For EPA-NCEA values, the date of the article provided by NCEA is provided.

Weight of Evidence: A - Human carcinogen
B1 - Probable human carcinogen - indicate that limited human data are available
B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
C - Posible human carcinogen
D - Not classifiable as a human carcinogen
E - Evidence of noncarcinogenicity

TABLE D-7
CHEMICAL-SPECIFIC PARAMETERS
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Chemical of Potential Concern	Absorption Factor	Reference	Permeability Constant (cm/hr)	Reference
Inorganics				
ALUMINUM	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
ANTIMONY	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
BARIUM	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
CADMIUM	0.001	U.S. EPA, 2000	1.00E-03	USEPA 2000
COBALT	NA	U.S. EPA, 2000	1.00E-03	On-line Database[1]
COPPER	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
MANGANESE	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
MERCURY	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000
SILVER	NA	U.S. EPA, 2000	1.00E-03	
VANADIUM	NA	U.S. EPA, 2000	1.00E-03	On-line Database[1]
ZINC	NA	U.S. EPA, 2000	1.00E-03	USEPA 2000

(1) Toxicity and Chemical-Specific Factors Database. Http://risk.lsd.ornl.gov/cgi-bin/tox. July 2001

Table D-8
Intentionally not included in this report

TABLE D-9.1
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil Child	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	5.7E-02	0.0E+00	5.7E-02
			BARIUM	--	--	NA	BARIUM	Kidneys	2.3E-01	0.0E+00	2.3E-01
			CADMIUM	--	--	NA	CADMIUM	Kidneys	3.7E+00	4.2E-01	4.2E+00
			COPPER	--	--	NA	COPPER	Gastrointestional System	6.6E-02	0.0E+00	6.6E-02
			MERCURY	--	--	NA	MERCURY	None	5.3E-02	0.0E+00	5.3E-02
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	2.8E+01	0.0E+00	2.8E+01
			ZINC	--	--	NA	ZINC	Blood	2.3E-03	0.0E+00	2.3E-03
			(Total for Child)	---	---	---		(Total for Child)	3.2E+01	4.2E-01	3.3E+01
	Surface Soil Adult	Peerless Photo Products Site	**Inorganics**				**Inorganics**				NA
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	6.1E-03	0.0E+00	6.1E-03
			BARIUM	--	--	NA	BARIUM	Kidneys	2.4E-02	0.0E+00	2.4E-02
			CADMIUM	--	--	NA	CADMIUM	Kidneys	4.0E-01	6.4E-02	4.6E-01
			COPPER	--	--	NA	COPPER	Gastrointestional System	7.0E-03	0.0E+00	7.0E-03
			MERCURY	--	--	NA	MERCURY	None	5.6E-03	0.0E+00	5.6E-03
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	3.0E+00	0.0E+00	3.0E+00
			ZINC	--	--	NA	ZINC	Blood	2.5E-04	0.0E+00	2.5E-04
			(Total for Adult)	---	---	---		(Total for Adult)	3.5E+00	6.4E-02	3.5E+00
			(Total for Child + Adult)	---	---	---				**Total Hazard Index Across Surface Soil (Child)**	**3.3E+01**
				Total Risk Across Surface Soil		---				**Total Hazard Index Across Surface Soil (Adult)**	**3.5E+00**
Groundwater	Groundwater	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ALUMINUM	--	--	NA	ALUMINUM	Central Nervous System	2.5E-01	1.6E-04	2.5E-01
			CADMIUM	--	--	NA	CADMIUM	Kidneys	3.5E+01	9.2E-01	3.6E+01
			COBALT	--	--	NA	COBALT	Respiratory System	5.0E-02	3.3E-05	5.0E-02
			MANGANESE	--	--	NA	MANGANESE	Central Nervous System	7.2E+00	1.2E-01	7.4E+00
			VANADIUM	--	--	NA	VANADIUM	Gastrointestinal System, Kidney	1.6E-01	3.9E-03	1.6E-01
			(Total for Child)	---	---	---		(Total for Child)	4.2E+01	1.0E+00	4.4E+01
	Groundwater Adult	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ALUMINUM	--	--	NA	ALUMINUM	Central Nervous System	5.3E-02	9.55E-05	5.3E-02
			CADMIUM	--	--	NA	CADMIUM	Kidneys	7.5E+00	5.37E-01	8.0E+00
			COBALT	--	--	NA	COBALT	Respiratory System	1.1E-02	1.94E-05	1.1E-02
			MANGANESE	--	--	NA	MANGANESE	Central Nervous System	1.6E+00	6.98E-02	1.6E+00
			VANADIUM	--	--	NA	VANADIUM	Gastrointestinal System, Kidney	3.3E-02	2.30E-03	3.6E-02
			(Total for Adult)	---	---	---		(Total for Adult)	9.1E+00	6.1E-01	9.7E+00
			(Total for Child + Adult)	---	---	---				**Total Hazard Index Across Groundwater (Child)**	**4.4E+01**
						---				**Total Hazard Index Across Groundwater (Adult)**	**9.7E+00**
					Total Risk Across All Media and All Exposure Routes	**0.0E+00**				**Total Hazard Index Across All Media and All Exposure Routes (Child)**	**7.6E+01**
										Total Hazard Index Across All Media and All Exposure Routes (Adult)	**1.3E+01**

TABLE D-9.2

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future
Receptor Population: Park Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	1.4E-03	0.0E+00	1.4E-03
			BARIUM	--	--	NA	BARIUM	Kidneys	5.4E-03	0.0E+00	5.5E-03
			CADMIUM	--	--	NA	CADMIUM	Kidneys	8.9E-02	1.4E-02	1.0E-01
			COPPER	--	--	NA	COPPER	Gastrointestional System	1.6E-03	0.0E+00	1.6E-03
			MERCURY	--	--	NA	MERCURY	None	1.3E-03	0.0E+00	1.3E-03
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	6.7E-01	0.0E+00	6.7E-01
			ZINC	--	--	NA	ZINC	Blood	5.6E-05	0.0E+00	5.6E-05
			(Total)	---	---	---		(Total)	7.7E-01	1.4E-02	7.8E-01
Total Risk Across Surface Soil						---		**Total Hazard Index Across Surface Soil**			**7.8E-01**
Total Risk Across All Media and All Exposure Routes						---		**Total Hazard Index Across All Media and All Exposure Routes**			**7.8E-01**

TABLE D-9.3
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future Receptor Population: Park Visitor Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	1.3E-02	0.0E+00	1.3E-02
			BARIUM	--	--	NA	BARIUM	Kidneys	5.0E-02	0.0E+00	5.1E-02
			CADMIUM	--	--	NA	CADMIUM	Kidneys	8.3E-01	9.3E-02	9.3E-01
			COPPER	--	--	NA	COPPER	Gastrointestional System	1.5E-02	0.0E+00	1.5E-02
			MERCURY	--	--	NA	MERCURY	None	1.2E-02	0.0E+00	1.2E-02
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	6.3E+00	0.0E+00	6.3E+00
			ZINC	--	--	NA	ZINC	Blood	5.2E-04	0.0E+00	5.2E-04
			(Total)	---	---	---		(Total)	7.2E+00	9.3E-02	7.3E+00
Total Risk Across Surface Soil						---	**Total Hazard Index Across Surface Soil**				**7.3E+00**
Total Risk Across All Media and All Exposure Routes						---	**Total Hazard Index Across All Media and All Exposure Routes**				**7.3E+00**

TABLE D-9.4
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Current/Future
Receptor Population: Youth Trespasser
Receptor Age: Youth (9-18 years)

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	1.4E-03	0.0E+00	1.4E-03
			BARIUM	--	--	NA	BARIUM	Kidneys	5.7E-03	0.0E+00	5.7E-03
			CADMIUM	--	--	NA	CADMIUM	Kidneys	9.4E-02	2.5E-02	1.2E-01
			COPPER	--	--	NA	COPPER	Gastrointestional System	1.6E-03	0.0E+00	1.6E-03
			MERCURY	--	--	NA	MERCURY	None	1.3E-03	0.0E+00	1.3E-03
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	7.1E-01	0.0E+00	7.1E-01
			ZINC	--	--	NA	ZINC	Blood	5.8E-05	0.0E+00	5.8E-05
			(Total)	---	---	---		(Total)	8.1E-01	2.5E-02	8.3E-01
Total Risk Across Surface Soil						---	**Total Hazard Index Across Surface Soil**				**8.3E-01**
Total Risk Across All Media and All Exposure Routes						---	**Total Hazard Index Across All Media and All Exposure Routes**				**8.3E-01**

TABLE D-9.5
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future
Receptor Population: Park Groundskeeper
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ANTIMONY	--	--	NA	ANTIMONY	Blood glucose and cholesterol	4.4E-03	0.0E+00	4.4E-03
			BARIUM	--	--	NA	BARIUM	Kidneys	1.7E-02	0.0E+00	1.7E-02
			CADMIUM	--	--	NA	CADMIUM	Kidneys	2.9E-01	4.6E-02	3.3E-01
			COPPER	--	--	NA	COPPER	Gastrointestional System	5.0E-03	0.0E+00	5.0E-03
			MERCURY	--	--	NA	MERCURY	None	4.0E-03	0.0E+00	4.0E-03
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	2.2E+00	0.0E+00	2.2E+00
			ZINC	--	--	NA	ZINC	Blood	1.8E-04	0.0E+00	1.8E-04
			(Total)	---	---	---		(Total)	2.5E+00	4.6E-02	2.5E+00
					Total Risk Across Surface Soil	---			**Total Hazard Index Across Surface Soil**		**2.5E+00**
Groundwater	Groundwater	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ALUMINUM	--	--	NA	ALUMINUM	Central Nervous System	1.9E-02	--	1.9E-02
			CADMIUM	--	--	NA	CADMIUM	Kidneys	2.7E+00	--	2.7E+00
			COBALT	--	--	NA	COBALT	Respiratory System	3.9E-03	--	3.9E-03
			MANGANESE	--	--	NA	MANGANESE	Central Nervous System	5.5E-01	--	5.5E-01
			VANADIUM	--	--	NA	VANADIUM	Gastrointestinal System, Kidney	1.2E-02	--	1.2E-02
			(Total)	---	--	---		(Total)	3.3E+00	--	3.3E+00
						---			**Total Hazard Index Across Groundwater**		**3.3E+00**
			Total Risk Across All Media and All Exposure Routes			---			**Total Hazard Index Across All Media and All Exposure Routes**		**5.8E+00**

TABLE D-10.1
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil Child	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			BARIUM	--	--	NA	BARIUM	Kidneys	2.3E-01	0.0E+00	2.3E-01
			CADMIUM	--	--	NA	CADMIUM	Kidneys	3.7E+00	4.2E-01	4.2E+00
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	2.8E+01	0.0E+00	2.8E+01
			(Total for Child)	---	---	---		(Total for Child)	3.2E+01	4.2E-01	3.2E+01
	Surface Soil Adult	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			CADMIUM	--	--	NA	CADMIUM	Kidneys	4.0E-01	6.4E-02	4.6E-01
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	3.0E+00	0.0E+00	3.0E+00
			(Total for Adult)	---	---	---		(Total for Adult)	3.4E+00	6.4E-02	3.5E+00
	Surface Soil Adult + Child	Peerless Photo Products Site	**Inorganics**								
			CADMIUM	NA	NA	NA					
			SILVER	NA	NA	NA					
			(Total for Child + Adult)	---	---	---				**Total Hazard Index Across Surface Soil (Child)**	**3.2E+01**
				Total Risk Across Surface Soil		---				**Total Hazard Index Across Surface Soil (Adult)**	**3.5E+00**
Groundwater	Groundwater Child	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			ALUMINUM	--	--	NA	ALUMINUM	Central Nervous System	2.5E-01	1.6E-04	2.5E-01
			CADMIUM	--	--	NA	CADMIUM	Kidneys	3.5E+01	9.2E-01	3.6E+01
			MANGANESE	--	--	NA	MANGANESE	Central Nervous System	7.2E+00	1.2E-01	7.4E+00
			VANADIUM	--	--	NA	VANADIUM	Gastrointestinal System, Kidney	1.6E-01	3.9E-03	1.6E-01
			(Total for Child)	---	---	---		(Total for Child)	4.2E+01	1.0E+00	4.3E+01
	Groundwater Adult	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			CADMIUM	--	--	NA	CADMIUM	Kidneys	7.5E+00	5.37E-01	8.0E+00
			MANGANESE	--	--	NA	MANGANESE	Central Nervous System	1.6E+00	6.98E-02	1.6E+00
			(Total for Adult)	---	---	---		(Total for Adult)	9.0E+00	6.1E-01	9.6E+00
			(Total for Child + Adult)	---	---	---				**Total Hazard Index Across Groundwater (Child)**	**4.3E+01**
						---				**Total Hazard Index Across Groundwater (Adult)**	**9.6E+00**
					Total Risk Across All Media and All Exposure Routes	**0.0E+00**				**Total Hazard Index Across All Media and All Exposure Routes (Child)**	**7.6E+01**
										Total Hazard Index Across All Media and All Exposure Routes (Adult)	**1.3E+01**

Total Hazard Index for CNS (Child)	**3.6E+01**
Total Hazard Index for Skin (Child)	**2.8E+01**
Total Hazard Index for Kidneys (Child)	**4.0E+01**
Total Hazard Index for GI System (Child)	**1.6E-01**

Total Hazard Index for CNS (Adult)	**4.6E+00**
Total Hazard Index for Skin (Adult)	**3.0E+00**
Total Hazard Index for Kidneys (Adult)	**8.5E+00**
Total Hazard Index for GI System (Adult)	**NA**

TABLE D-10.2

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site

Scenario Timeframe: Future
Receptor Population: Park Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			CADMIUM	--	--	NA	CADMIUM	Kidneys	8.9E-02	1.4E-02	1.0E-01
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	6.7E-01	0.0E+00	6.7E-01
			(Total)	---	---	---		(Total)	7.6E-01	1.4E-02	7.8E-01
			Total Risk Across Surface Soil			---		**Total Hazard Index Across Surface Soil**			**7.8E-01**
			Total Risk Across All Media and All Exposure Routes			---		**Total Hazard Index Across All Media and All Exposure Routes**			**7.8E-01**

Total Hazard Index for CNS	**6.7E-01**
Total Hazard Index for Skin	**6.7E-01**
Total Hazard Index for Kidneys	**1.0E-01**

TABLE D-10.3

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future Receptor Population: Park Visitor Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			CADMIUM	--	--	NA	CADMIUM	Kidneys	8.3E-01	9.3E-02	9.3E-01
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	6.3E+00	0.0E+00	6.3E+00
			(Total)	---	---	---		(Total)	7.1E+00	9.3E-02	7.2E+00
Total Risk Across Surface Soil						---	**Total Hazard Index Across Surface Soil**				7.2E+00
Total Risk Across All Media and All Exposure Routes						---	**Total Hazard Index Across All Media and All Exposure Routes**				7.2E+00

Total Hazard Index for CNS	6.3E+00
Total Hazard Index for Skin	6.3E+00
Total Hazard Index for Kidneys	9.3E-01

TABLE D-10.4
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Current/Future
Receptor Population: Youth Trespasser
Receptor Age: Youth (9-18 years)

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk			Chemical	Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**				**Inorganics**				
			CADMIUM	--	--	NA	CADMIUM	Kidneys	9.4E-02	2.5E-02	1.2E-01
			SILVER	--	--	NA	SILVER	Central Nervous System, Skin	7.1E-01	0.0E+00	7.1E-01
			(Total)	---	---	---		(Total)	8.0E-01	2.5E-02	8.2E-01
Total Risk Across Surface Soil						---	**Total Hazard Index Across Surface Soil**				**8.2E-01**
Total Risk Across All Media and All Exposure Routes						---	**Total Hazard Index Across All Media and All Exposure Routes**				**8.2E-01**

Total Hazard Index for CNS	**7.1E-01**
Total Hazard Index for Skin	**7.1E-01**
Total Hazard Index for Kidneys	**1.2E-01**

TABLE D-10.5
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
PEERLESS PHOTO PRODUCTS SITE, SHOREHAM, NEW YORK

Location: Peerless Photo Products Site
Scenario Timeframe: Future
Receptor Population: Park Groundskeeper
Receptor Age: Adult

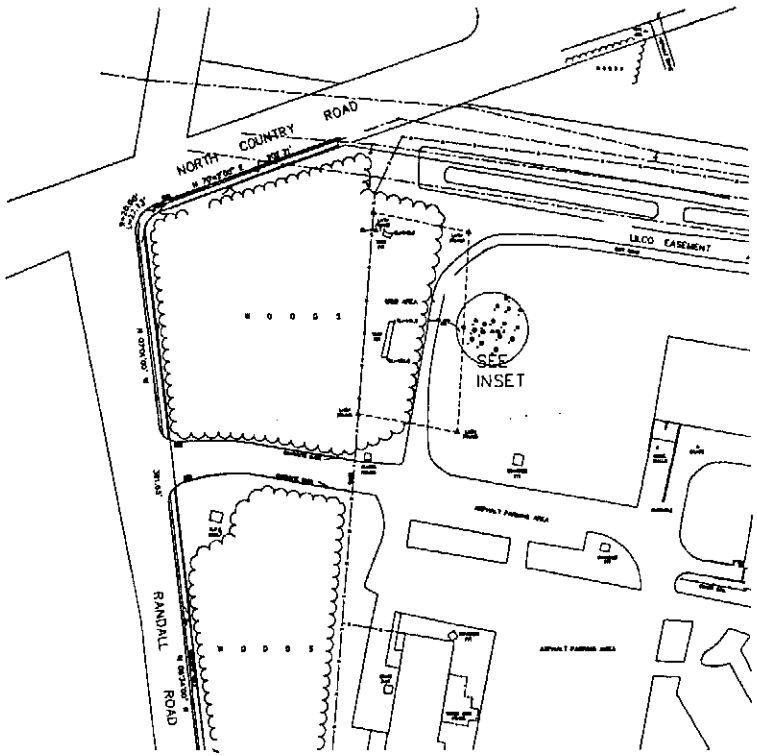
Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk		
				Ingestion	Dermal	Exposure Routes Total
Soil	Surface Soil	Peerless Photo Products Site	**Inorganics**			
			CADMIUM	--	--	NA
			SILVER	--	--	NA
			(Total)	---	---	---
Total Risk Across Surface Soil						---
Groundwater	Groundwater	Peerless Photo Products Site	**Inorganics**			
			CADMIUM	--	--	NA
			MANGANESE	--	--	NA
			(Total)	--	--	NA
						NA
Total Risk Across All Media and All Exposure Routes						---

Chemical	Non-Carcinogenic Hazard Quotient			
	Primary Target Organ	Ingestion	Dermal	Exposure Routes Total
Inorganics				
CADMIUM	Kidneys	2.9E-01	4.6E-02	3.3E-01
SILVER	Central Nervous System, Skin	2.2E+00	0.0E+00	2.2E+00
	(Total)	2.4E+00	4.6E-02	2.5E+00
Total Hazard Index Across Surface Soil				**2.5E+00**
Inorganics				
CADMIUM	Kidneys	2.7E+00	--	2.7E+00
MANGANESE	Central Nervous System	5.5E-01	--	5.5E-01
	(Total)	3.2E+00	--	3.2E+00
Total Hazard Index Across Groundwater				**3.2E+00**
Total Hazard Index Across All Media and All Exposure Routes				**5.7E+00**

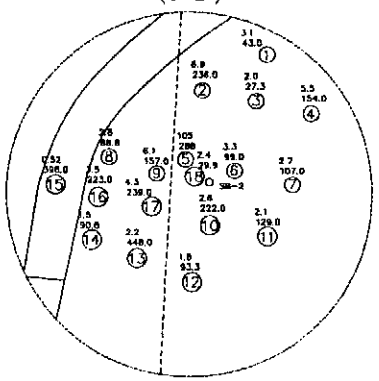
Total Hazard Index for CNS	**2.7E+00**
Total Hazard Index for Skin	**2.2E+00**
Total Hazard Index for Kidneys	**3.0E+00**

Appendix E

Sample Location Maps



SHALLOW SOIL SAMPLES
(0-2")

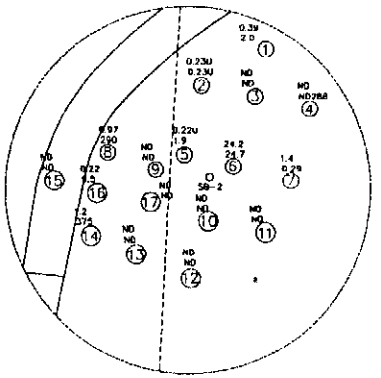


"INSET"
SCALE: 1"= 7.5'

Sample Results from SB-2

Sample Depth (ft)	0-2	4-6	8-10	15-17	20-22
Cadmium	22.1	5.3	0.83	0.73	0.95
Silver	307	73.5	0.41	0.41	6.2

DEEP SOIL SAMPLES
(2-2.5")



"INSET"
SCALE: 1"= 7.5'

LEGEND

CADMIUM	5.5
SILVER	154.0
SOIL SAMPLE LOCATION	(12)

NOTE: ALL RESULTS IN PPM (PARTS PER MILLION)

150 75 0 150
GRAPHIC SCALE IN FEET

FILE: F:\PROJ\13711.02\AGFA\FIGURE\FIG6.DWG

EA ENGINEERING, SCIENCE, AND TECHNOLOGY	AGFA DIVISION OF BAKER CORP. SHOREHAM, NEW YORK	APC 6 - PHASE 1 & 2 SOIL SAMPLING LOCATIONS AND ANALYTICAL RESULTS	DESIGNED BY: CN	DRAWN BY: CN	DATE: 3-16-00	PROJECT NO.: 13711.02	FILE NAME: FIGURE 6
			CHECKED BY: CN	PROJECT MGR.: CK	SCALE: AS SHOWN	DRAWING NO.: -	FIGURE: -

SB-6E

SAMPLE DEPTH (FT)	4.0-6.0	8.0-10.0	12.0-14.0	16.0-18.0
Ag	23.5	34	8.8	ND
Cd	ND	ND	ND	ND

SB-6G

SAMPLE DEPTH (FT)	4.0-6.0	4.0-6.0 DUP	8.0-10.0	12.0-14.0	16.0-18.0	20.0-22.0
Ag	9.6	ND	41.1	5.8	ND	ND
Cd	ND	ND	ND	ND	ND	ND

SB-6A

SAMPLE DEPTH (FT)	4.0
Ag	406 J
Cd	37.9

SB-6B

SAMPLE DEPTH (FT)	4.0
Ag	376 J
Cd	4

SB-6F

SAMPLE DEPTH (FT)	4.0-6.0	8.0-10.0	12.0-14.0
Ag	269	254	250
Cd	3.9	182	293

SB-6C

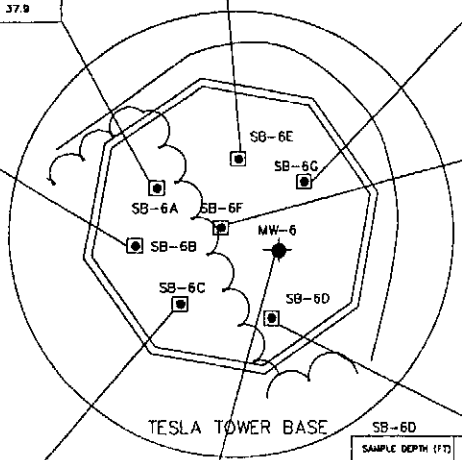
SAMPLE DEPTH (FT)	4.0-6.0	8.0-10.0	12.0-14.0
Ag	ND	18.6	0.73 B
Cd	ND	ND	ND

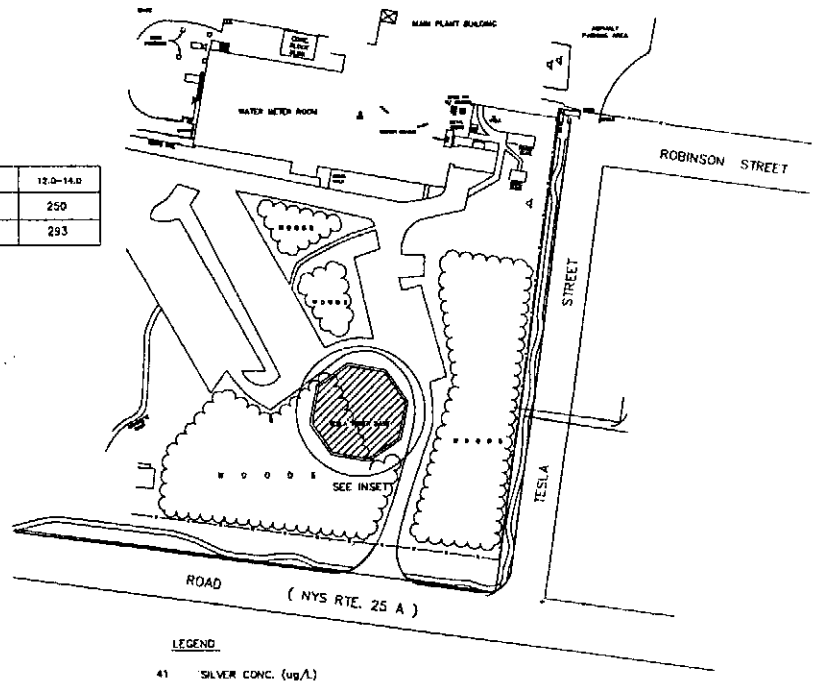
SB-6D

SAMPLE DEPTH (FT)	4.0-6.0	8.0-10.0	12.0-14.0
Ag	ND	ND	ND
Cd	ND	ND	ND

MW-6

SAMPLE DEPTH (FT)	3.5-5.5	40-41	59-61	54-56	109-111
Ag	36.4	ND	ND	0.46	1.9
Cd	ND	1.9	1.4	1.2	0.70





N

LEGEND

41	SILVER CONC. (ug/L)
62	CADMIUM CONC. (ug/L)
J	CONC. IS BETWEEN THE MDL AND THE MCQL
B	CONC. IS ESTIMATED
	MONITORING WELL
	SOIL BORING
	APC - AREA OF POTENTIAL CONCERN

50 25 0 50
GRAPHIC SCALE IN FEET
(INSET)

150 75 0 150
GRAPHIC SCALE IN FEET

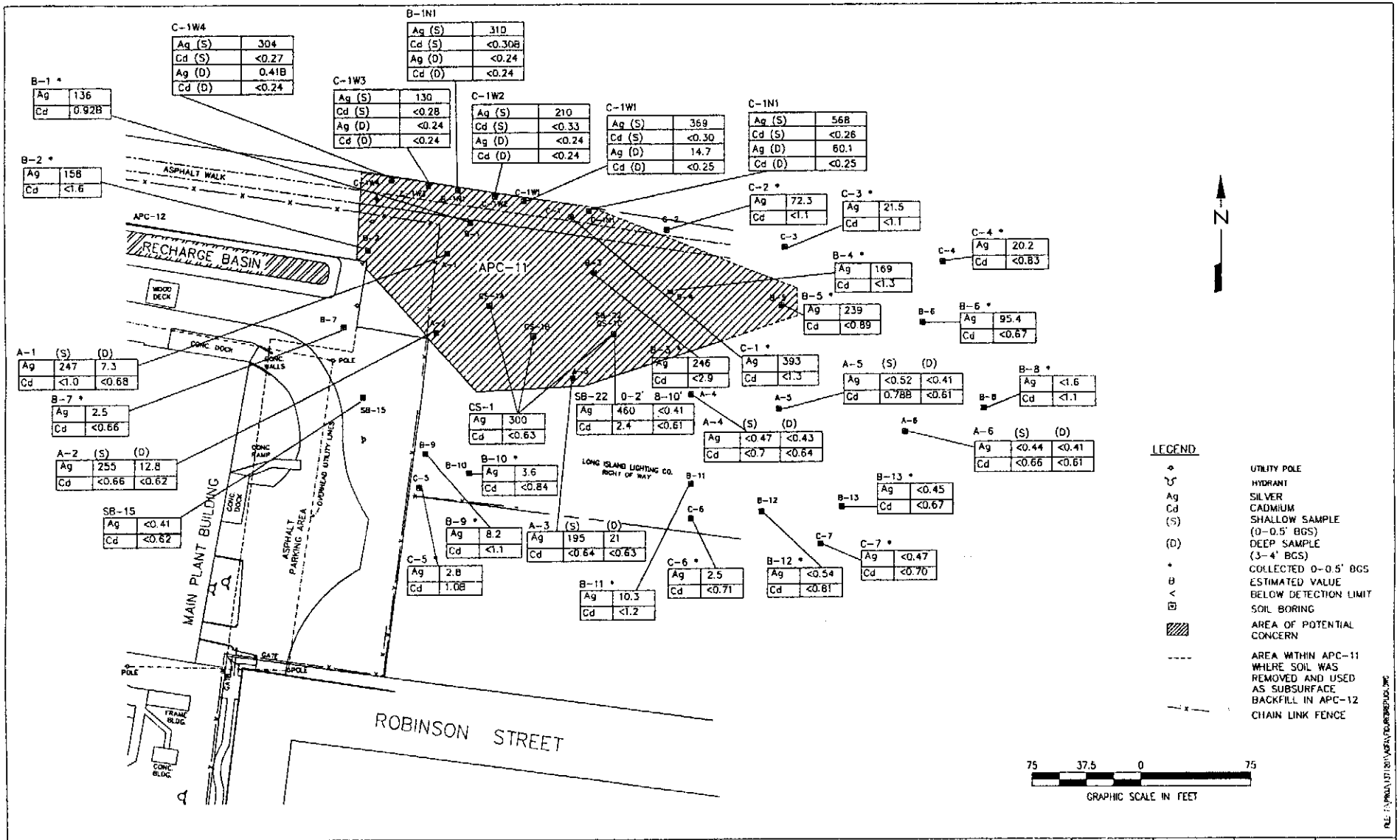
FILE: F:\PROJ\13712\01\AGFA\FIGURE7PERLD.DWG

EA ENGINEERING, SCIENCE, AND TECHNOLOGY

AGFA DIVISION OF BAYER CORP.
SHOREHAM, NEW YORK

APC-10 PHASE 1 AND 2 SOIL SAMPLING LOCATIONS AND ANALYTICAL RESULTS

DESIGNED BY	DRAWN BY	DATE	PROJECT NO.	FILE NAME
CN	CN	5-2-00	13712.01	FIGURE 7
CHECKED BY	PROJECT MGR.	SCALE	DRAWING NO.	FIGURE
CK	CK	AS SHOWN	-	7

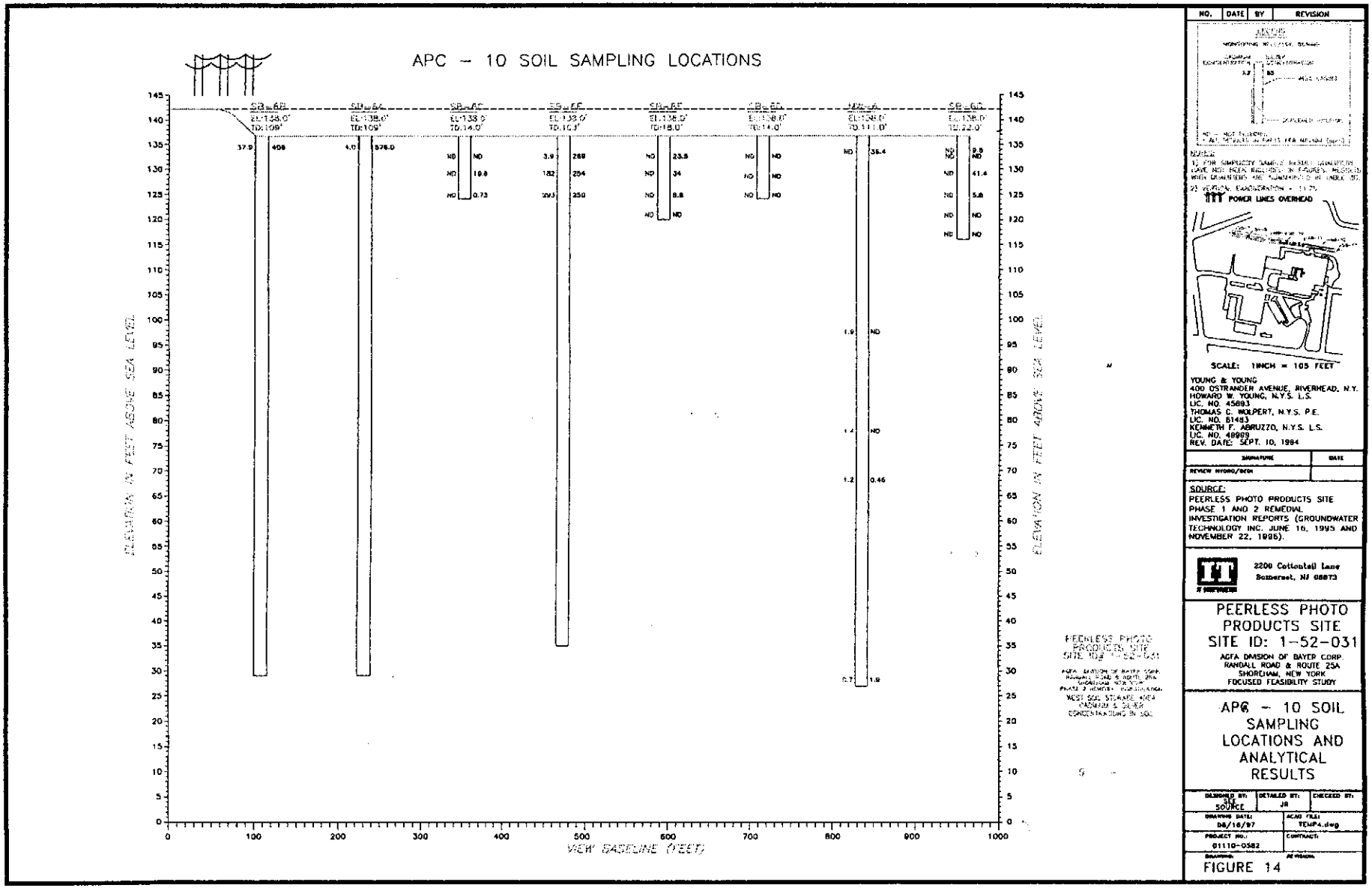


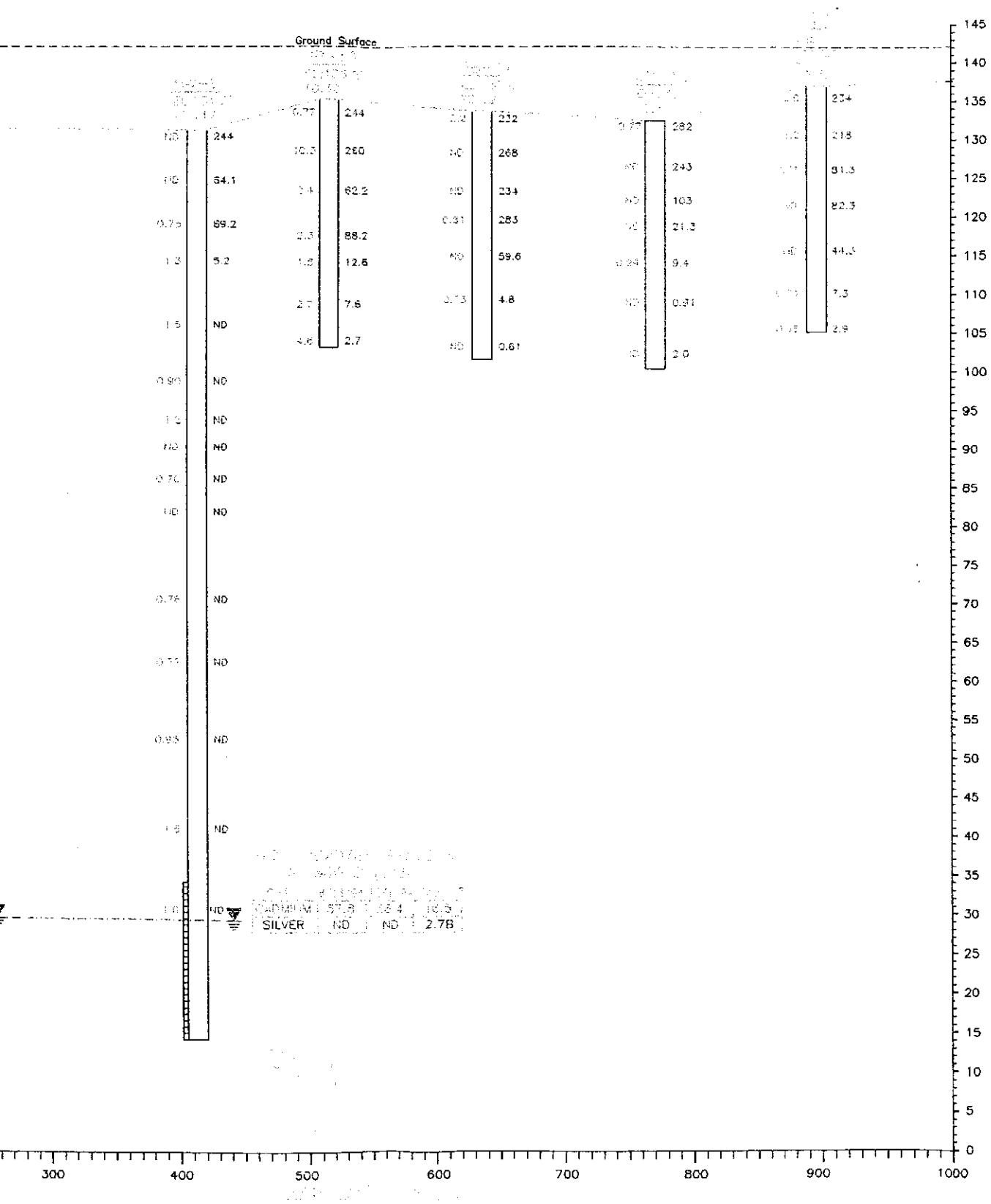
EA ENGINEERING, SCIENCE, AND TECHNOLOGY

AGFA DIVISION OF BAYER CORP.
SHOREHAM, NEW YORK

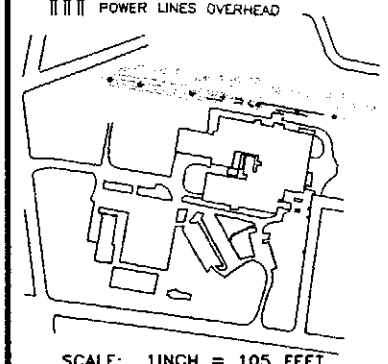
APC-11 PHASE 1 AND 2
SOIL SAMPLING LOCATIONS AND ANALYTICAL RESULTS

DESIGNED BY	DRAWN BY	DATE	PROJECT NO.	FILE NAME
CN	CN	5-3-00	13712.01	FIGURE 8
CHECKED BY	PROJECT MGR.	SCALE	DRAWING NO.	FIGURE
CK	CK	AS SHOWN	-	8


APC – 10 SOIL SAMPLING LOCATIONS
ELEVATION IN FEET ABOVE SEA LEVEL
VIEW BASELINE (FEET)
0
100
200
300
400
500
600
700
800
900
1000
PEERLESS PHOTO PRODUCTS SITE
SITE ID: 1-52-031
AGFA DIVISION OF BAYER CORP.
RANDALL ROAD & ROUTE 25A
SHOREHAM, NEW YORK
FOCUSED FEASIBILITY STUDY
APC – 10 SOIL SAMPLING LOCATIONS AND ANALYTICAL RESULTS
POWER LINES OVERHEAD
SCALE: 1INCH = 105 FEET
YOUNG & YOUNG
400 OSTRANDER AVENUE, RIVERHEAD, N.Y.
HOWARD W. YOUNG, N.Y.S. L.S.
LIC. NO. 45893
THOMAS C. WOLPERT, N.Y.S. P.E.
LIC. NO. 61483
KENNETH F. ABRUZZO, N.Y.S. L.S.
LIC. NO. 48989
REV. DATE: SEPT. 10, 1994
SOURCE:
PEERLESS PHOTO PRODUCTS SITE
PHASE 1 AND 2 REMEDIAL
INVESTIGATION REPORTS (GROUNDWATER
TECHNOLOGY INC. JUNE 16, 1995 AND
NOVEMBER 22, 1995).
2200 Cottontail Lane
Somerset, NJ 08873
DRAWN BY: SOURCE
DETAILED BY: JR
CHECKED BY:
DRAWING DATE: 08/16/97
ACAD FILE: TEMP4.dwg
PROJECT NO.: 01110-0582
CONTRACT:
FIGURE 14



POWER LINES OVERHEAD



SCALE: 1INCH = 105 FEET

YOUNG & YOUNG
400 OSTRANDER AVENUE, RIVERHEAD, N.Y.
HOWARD W. YOUNG, N.Y.S. L.S.
LIC. NO. 45893
THOMAS C. WOLPERT, N.Y.S. P.E.
LIC. NO. 61483
KENNETH F. ABRUZZO, N.Y.S. L.S.
LIC. NO. 49999
REV. DATE: SEPT. 10, 1994

SIGNATURE	DATE
REVIEW HYDRO/GEO:	

SOURCE:
PEERLESS PHOTO PRODUCTS SITE PHASE 1 AND 2 REMEDIAL INVESTIGATION REPORTS (GROUNDWATER TECHNOLOGY INC. JUNE 16, 1995 AND NOVEMBER 22, 1996).

485-C ROUTE 1 SOUTH SUITE 200
ISELIN, NJ 08830

PEERLESS PHOTO PRODUCTS SITE
SITE ID: 1-52-031

AGFA DIVISION OF BAYER CORP.
RANDALL ROAD & ROUTE 25A
SHOREHAM, NEW YORK
FOCUSED FEASIBILITY STUDY

APC - 12 SOIL SAMPLING LOCATIONS AND ANALYTICAL RESULTS

DESIGNED BY:	DETAILED BY:	CHECKED BY:
SEE SOURCE	JR	
DRAWING DATE: 08/18/97		ACAD FILE: TEMP4.dwg
PROJECT NO.: 01110-0582		CONTRACT:
DRAWING: FIGURE 9		REVISION: