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TECHNICAL MEMORANDUM
SUBTASK 7.2
PUBLIC HEALTH AND
ENVIRONMENTAL EFFECTS ASSESSMENT
REMEDIAL INVESTIGATION/FEASIBILITY STUDY

First Piedmont Rock Quarry/Route 719 Site

Prepared By:

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9 March 1990

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January 24, 1991

Mr. Andrew Palestini
Enforcement Project Manager
U.S. Environmental Protection Agency
Region III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

Reference: Technical Memorandum - Subtask 7.2 Round Two Revisions
Risk Assessment Report
First Piedmont Rock Quarry/Route 719 Site
Westinghouse Project No. 4112-88-960

Dear Mr. Palestini:

On behalf of Goodyear Tire and Rubber Company, Corning Glass Works, and First Piedmont Corporation (Respondents), Westinghouse Environmental and Geotechnical Services, Inc. (Westinghouse) is pleased to submit two copies of Revised Sections of the referenced document for your use. These Revisions are submitted as a project deliverable in accordance with the Consent Order (Docket No. III-83-13-DC) and the RI/FS Work Plan.

In addition, we have enclosed two copies of our Response to Comments on the Risk Assessment received from you 10 October 1990.

Please provide written acknowledgement of receipt of these materials at your earliest convenience.

We trust that this information meets your needs. If you have any questions, please call.

Sincerely

WESTINGHOUSE ENVIRONMENTAL AND
GEOTECHNICAL SERVICES, INC.

Jan P. Sassaman, PhD
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March 9, 1990

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841 Chestnut Building
Philadelphia, Pennsylvania 19107

Reference: Technical Memorandum - Subtask 7.2
Public Health and Environmental Effects Assessment
First Piedmont Rock Quarry/Route 719 Site
Westinghouse Project No. 4112-88-907B

Dear Mr. Palestini:

On behalf of Goodyear Tire and Rubber Company, Corning Glass Works, and First Piedmont Corporation (Respondents), Westinghouse Environmental and Geotechnical Services, Inc. (Westinghouse) is pleased to submit two copies of the referenced document for your use. This document is submitted as a project deliverable in accordance with the Consent Order (Docket No. III-83-13-DC) and the RI/FS Work Plan.

In addition, we have enclosed two copies of Section 9 of the Final Remedial Investigation Report, replacement tables (28-1 through 28-17) and a replacement sheet for the Table of Contents for the RI Report in accord with our transmittal of February 13, 1990.

Please provide written acknowledgement of receipt of this document at your earliest convenience.

We trust this information meets your needs. If you have any questions, please call.

Sincerely,
WESTINGHOUSE ENVIRONMENTAL AND
GEOTECHNICAL SERVICES, INC.

Jan F. Sassaman, Ph.D.
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TECHNICAL MEMORANDUM
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9 March 1990

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EXECUTIVE SUMMARY

This technical memorandum presents the results of Subtask 7.2: Public Health and Environmental Effects Assessment, for the Remedial Investigation/Feasibility Study (RI/FS) of the First Piedmont Rock Quarry/Route 719 (FPRQ) site. It is provided to the U.S. Environmental Protection Agency as a project deliverable in accordance with a Consent Order between Goodyear Tire and Rubber Company, Corning Glass Works, First Piedmont Corporation, the U.S. Environmental Protection Agency, and the Commonwealth of Virginia.

BACKGROUND

The risk assessment provides an analysis of the potential risk to human health and the environment posed by the site in the absence of remedial action. In addition, the risk assessment provides an analysis of the chemicals and exposure pathways of concern to focus the Feasibility Study.

Disposal activities occurred in approximately two acres of the site during a period from April 1970 to July 1972. The U.S. Environmental Protection Agency listed the site on the National Priorities List on July 21, 1987. Under the terms of a Consent Order, the PRP's agreed to carry out a RI/FS at the site. Westinghouse Environmental and Geotechnical Services, Inc. of Cary, North Carolina, was contracted by the PRP's to perform the RI/FS at the site.

The majority of material disposed of at the site consisted of industrial solid waste. Approximately 65,000 cubic yards of solid waste, 630,000 gallons of diatomaceous earth slurry, and 15,000 gallons of a waste water and residual degreaser mixture were disposed of at the site.

SITE ENVIRONMENT

The FPRQ site is located within the Piedmont Physiographic Province of southern Virginia. This area consists of rounded uplands dissected by numerous streams. Land surfaces in the vicinity of the site slope gently eastward with local relief between the uplands and valleys on the order of approximately 100 feet. Surface drainage from the site is to Lawless Creek, about 1,400 feet to

the northwest of the site. Lawless Creek is a tributary to Fall Creek, which in turn is a tributary to the Dan River.

The regional geology in the vicinity of the FPRQ site consists of zero to three feet of weathered residuum and saprolite overlying Precambrian metamorphic rocks. Triassic-age dikes occur sporadically within the Precambrian rocks. Recent unconsolidated sediments occur along major stream valleys. Bedrock in this area consists of massive, lineated gneiss. No faults have been mapped in the immediate vicinity of the site. Geophysical survey data indicate that the shallow bedrock is fractured, but that fractures probably do not occur at depths much greater than 50 to 100 feet.

Most soils in the area are moderately to well drained sandy loams. The soils range from well developed on flat slopes to thin and poorly developed on steep slopes.

Local ground water systems occur within small areas coinciding with surface water basins. Little, if any, interbasinal flow occurs. Ground water recharge occurs throughout the area from infiltration of precipitation. Ground water occurrence in the Piedmont Province is limited principally to a depth of less than 150 feet. Most water occurs within 30 feet of the surface. Flow is primarily horizontal. Ground water at the FPRQ site occurs at depths ranging from the land surface to about 30 feet below land surface. The private wells in the Beaver Park community are hydraulically upgradient from the landfill.

Surface water run-off from the site enters the northern and southern drainages. Run-off from the site is generated in response to precipitation falling within the landfill area and as a result of ground water discharge.

The FPRQ site is in the mixed deciduous forest biome of North America, characterized by a species-rich mixed mezophytic forest. The quarry proper has undergone recent disturbance and the vegetation reflects early stages of secondary succession. Streams in the vicinity of the site are classified as upper perennial, first or second order streams. None of the streams are trout waters.

A preliminary wetland delineation was performed at the FPRQ site to determine if any areas in the vicinity of the site would be classified as wetlands. Only a cat-tail marsh located at the confluence of the northern

drainage and the Lawless Creek flood plain is considered to be a wetland. This area and the other areas of hydrophytic vegetation that were identified are probably due to changes in the hydrology of the area caused by man's activities.

There are no threatened or endangered species known to occur in the immediate area of the FPRQ site.

Crop production and pasture land account for approximately 20 percent of the land use in the vicinity of the site. There are several clusters of unincorporated residential areas within one mile of the site, which account for approximately 10 percent of the land use. Beaver Park, a community of approximately 260 persons, is the largest of these residential areas and is located immediately southeast of the site. No historical or archaeological resources have been identified in the vicinity of the FPRQ site.

SITE CONTAMINANTS

Sampling activities conducted during the FPRQ RI consisted of multimedia sampling for chemical analysis of soils in and near the quarry, surface water and sediments, and ground water. In addition, surface water and sediment samples were obtained for biological toxicity testing of both water and sediment elutriate. Fourteen compounds of particular concern were identified as indicator chemicals representing constituents of "highest risk", thus permitting a public health evaluation that focuses on the chemicals of greatest potential concern at the site.

TOXICOLOGICAL CHARACTERISTICS

A brief review of the toxicological characteristics was prepared for each of the fourteen indicator chemicals. Toxicological profiles of each of the fourteen indicator chemicals are presented in Appendix B.

TRANSPORT PATHWAYS AND POTENTIAL EXPOSURE POINTS

Simple exposure pathways are of primary significance at the FPRQ site. Of these, the ingestion pathways are the most important, based on site constituents and contaminant distribution.

The potential extent of ground water exposure is limited. Private wells in the vicinity of the FPRQ site are upgradient of the quarry. In general,

contaminants found in the quarry and source areas are primarily metals, which have a very low solubility and are readily adsorbed onto the soil matrix.

Surface water migration pathways include the northern and southern drainages. Discharge from the North Pond and from leachate seeps enters the northern drainage. Most of the landfill is internally drained and therefore does not contribute to runoff from the site. The concentration of soluble contaminants that may leave the site diminish as they become diluted by surface water run-off from the remaining portions of the drainage basin. The potential for surface water dilution, particularly as the northern and southern drainages flow into Lawless Creek, is significant.

The potential for soil being carried from the site is minimal. Soil could migrate from the site if carried off by individuals or on the tires of vehicles entering and leaving the site. However, access to the site is limited by a chain link fence and no trespassing signs.

Potential exposure via the air transport pathway is negligible. The prevailing wind direction in the vicinity of the site is from the southwest. Negligible amounts of contaminants carried by a southwest wind would move away from the community of Beaver Park to forested and agricultural land northeast of the site, where particulates would be deposited or otherwise filtered out by vegetation.

Potential human exposure to contaminants could be by five exposure routes: source area contact, ground water, surface water, soil, and air. Presently, there is minimal exposure to source contamination. There are no ground water wells within the confines of the site. Much of the landfill material is covered with a cap. No activities occur at the site that would result in direct contact, with the exception of the ongoing RI/FS. There is no exposure to contaminants in ground water from the FPRQ site at present, nor is such exposure likely to occur in the future since the most likely areas for future development in the vicinity of the site are not located in areas hydraulically downgradient from the quarry. Potential exposure via the surface water pathway is limited. Migration of contaminants from the quarry via the surface water pathway extends primarily to the northern and southern drainage with negligible, if any, contamination of the wetland or of Lawless Creek. Surface water down gradient of the site is not used for potable water. Fish species observed in Lawless and Fall Creeks are not

likely to be used as a food source by the local human population. Inhalation of volatilized compounds is unlikely since concentrations of volatiles in samples obtained from the site are very low to below detectable levels and volatiles were not found in near-surface samples.

A total of seventeen scenarios were developed for the FPRQ site for the no-action remedial alternative. The scenarios considered include potential exposure to source material and leachate in the quarry, ground water, surface water and sediments, soils in and near the quarry, and particulate air emissions.

EXPOSURE ASSESSMENT

Development of exposure scenarios was based on a series of general assumptions, as well as on specific assumptions for the different scenarios. These assumptions lend a degree of conservatism to the exposure estimates. It is unlikely that the FPRQ site would be developed for residential use, unlikely that individuals would live there for 70 years, and unlikely that individuals would be exposed to the maximum concentrations of contaminants in any particular environmental media.

Actual exposure estimates for each of the exposure scenarios are detailed in Tables 6.1 through 6.17. Specific assumptions and calculation methods for each of these exposure scenarios are provided with the appropriate table.

HUMAN HEALTH BASED STANDARDS AND CRITERIA

Human health based standards and criteria as established by the U.S. Environmental Protection Agency are presented for the fourteen indicator chemicals, including such standards as reference doses, health advisories, and cancer potencies where relevant.

RISK SUMMARY

Risk requires that there is exposure to toxic or otherwise harmful conditions. Without such exposure, there is no risk. Currently, there is no exposure to the FPRQ site, therefore, the risks estimated are potential risks, based on the assumption that the site is developed or otherwise used in the future such that there will be some degree of exposure. The risk assessment is

based on a chain of conservative assumptions that, taken together, provide a significant margin of safety in estimating potential risk from the site.

Under the assumptions inherent in the "average case" calculations for the various scenarios, the following areas of potential concern are identified.

- Quarry Leachate - exposure to antimony, barium, lead, arsenic, and benzene as a consequence of future use of the quarry leachate as a source of potable water.
- Source Material - exposure to lead as a consequence of soil ingestion by children playing in source material.
- Northern Drainage Sediments - exposure to arsenic as a consequence of incidental ingestion of soil by children playing in sediments.
- Quarry Soil - exposure to lead and arsenic as a consequence of soil ingestion by children playing in quarry soil.
- Northern Drainage Soil - exposure to arsenic as a result of ingestion of soil by children playing and adults gardening in Northern Drainage soil.

Given the extremely conservative assumptions inherent in the "reasonable worst case" calculations for the various scenarios, the following additional areas of concern were identified.

- Source Material - exposure to arsenic as a result of ingestion by children playing and adults gardening in source material and exposure to lead as a consequence of adults ingesting soil while gardening in source material.
- North and South Ponds - exposure to arsenic as a consequence of incidental ingestion of water by children during play.
- Northern Drainage Sediments - exposure to barium and lead as a consequence of incidental ingestion of soil by children playing in sediments.
- Quarry Soils - exposure to lead and arsenic by adults as a consequence of ingestion of soil while gardening in quarry soils and exposure to arsenic by children during play.
- Northern Drainage Soils - exposure to lead by children as a consequence of incidental ingestion of soil while playing in Northern Drainage soils.

o - Southern Drainage Soils (Disturbed Area) - exposure to arsenic as a result of ingestion by children playing and adults gardening in Southern Drainage soils.

The following are summaries of the risks associated with each suite of exposure scenarios, given the conservatism inherent in the analyses.

ENVIRONMENTAL EFFECTS ASSESSMENT

Potential effects are qualitatively discussed for physical, biological, and human environments. However, few such effects, if any, are anticipated.

The FPRQ site, given the no-action alternative, would result in little to no adverse environmental effects on the physical environment. The ground and surface water components have the highest potential for adverse effects. Given the no-action alternative, few adverse environmental effects on the biological environment are expected. Habitat areas influenced by surface or ground water or animals that might come into contact with contaminated surface water or soils within the site itself would have the highest potential to be adversely affected. The potential effects on the human environment were discussed in the risk assessment.

Samples of surface water and sediments were collected for aquatic toxicity testing. Surface water in the northern drainage and in Lawless Creek were not acutely or subchronically toxic to the test organisms. However, surface water from the upper reaches of the northern drainage did result in statistically significant decreases in growth among fathead minnows and reproduction among Ceriodaphnia at concentrations above 30 percent dilution. Undiluted water from the lower northern drainage resulted in decreased reproduction of Ceriodaphnia. It is likely that effluent from the upper northern drainage would always be diluted to concentrations significantly less than 30 percent and thus there would

be no expected adverse effects on reproduction or growth of biota as a result of drainage water from the FPRQ site. In addition, some surface water samples from the Southern drainage exceeded Clean Water Act criteria for protection of aquatic life for cadmium and zinc.

SECTION 1

INTRODUCTION

This technical memorandum presents the results of Subtask 7.2: Public Health and Environmental Effects Assessment, for the Remedial Investigation/Feasibility Study (RI/FS) of the First Piedmont Rock Quarry/Route 719 (FPRQ) site. Selection of a cost effective remedy for remedial action at Superfund sites requires comparison of alternatives based on an evaluation of public health and environmental impacts, as well as other factors such as technological and engineering feasibility, cost, and institutional constraints (U.S. Environmental Protection Agency, 1986). Evaluation of public health and environmental impacts is accomplished by a risk assessment, which serves both to evaluate potential adverse effects associated with the site and to determine the likelihood that these potential adverse effects could actually pose a significant risk to human health or the environment.

This technical memorandum is provided to the U.S. Environmental Protection Agency (EPA) Region III as a project deliverable in accordance with the Consent Order (Docket No. III-88-13-DC) between Goodyear Tire and Rubber Company, Corning Glass Works, First Piedmont Corporation, the U.S. Environmental Protection Agency, and the Commonwealth of Virginia, dated December 31, 1988.

The risk assessment process for the FPRQ site, as described in the RI/FS Work Plan (BCM Converse, 1987), is divided into four subtasks.

1. Preliminary Health and Environmental Effects Assessment
2. Public Health Assessment of No-Action Alternative

3. Public Health and Environmental Initial Screening of Alternatives (Subtask 2.1 of Phase II Feasibility Study)

4. Detailed Analysis of Alternatives (Phase III Feasibility Study)

This technical memorandum covers item number 2 above, Public Health Assessment of No-Action Alternative. Item number 1, the Preliminary Health and Environmental Health Effects Assessment, was presented in a prior memorandum as a deliverable for Subtask 7.1 (Westinghouse, 1989d). Additional material has been presented in the technical memorandum for the Phase II FS regarding health based criteria and levels of environmental contamination that are protective of the environment. Subsequent analyses will be provided at the completion of the Phase III Feasibility Study (Detailed Analysis).

1.1 OBJECTIVES

According to the RI/FS Work Plan, the objectives of Subtask 7.2 are two-fold. The risk assessment serves to provide an analysis of the potential risk to human health and the environment posed by the site absent remedial action. In addition, the risk assessment also serves to provide an analysis of the chemicals and exposure pathways of concern to focus the Feasibility Study.

1.2 ORGANIZATION OF TECHNICAL MEMORANDUM

This risk assessment is prepared as a separate Technical Memorandum in addition to material provided in the FPRQ Remedial Investigation Report. Much of the material herein is repeated from other memoranda, including the Subtask 7.1 Technical Memorandum: Preliminary Health and Environmental Effects Assessment (Westinghouse, 1989d). The results of the risk assessment will be summarized

in the FPRQ Remedial Investigation Report, which is being submitted to the U.S. Environmental Protection Agency as a deliverable under Task 8.

Section 1 provides information on the risk assessment process and the specific scope of the Public Health and Environmental Effects Assessment. Also presented in this section is a specific discussion of the organization of this risk assessment and a summary of background information on the FPRQ site.

Section 2 provides a description of the environment of the FPRQ site and its environs in terms of physical, biological, and human elements of the environment.

Section 3 discusses site contaminants in terms of both studies prior to the RI/FS and results of sampling during the RI/FS. This section also discusses the selection of a suite of indicator chemicals for risk assessment at the FPRQ site.

Section 4 provides information on the toxicology and environmental fate and behavior of these selected indicator chemicals. Section 4 also provides summary information on dose-response relationships for the selected indicator chemicals.

Section 5 provides a discussion of contaminant transport routes from the FPRQ site, a brief description of potential exposure routes, and identification of exposure scenarios.

Section 6 provides estimates of potential human exposures to site contaminants for selected exposure routes and scenarios.

Section 7 discusses the human health related standards and criteria and regulations.

Section 8 provides a summary of risks associated with the no-action alternative at the FPRQ site.

Section 9 summarizes the potential environmental effects associated with the FPRQ site.

Section 10 lists the references used in preparing this risk assessment.

1.3 FIRST PIEDMONT ROCK QUARRY/ROUTE 719 SITE

The FPRQ site is located at 36°39'28" north latitude and 70°19'22" west longitude, in Pittsylvania County, Virginia. The site, located along Route 719 approximately six miles north of Danville, Virginia, is bordered by wooded areas to the north and east, by Lawless Creek to the west, and by Route 719 and the community of Beaver Park to the south. Figure 1 is a regional site location map. Figure 2 is a map of the immediate environs of the site showing the Phase II monitoring locations.

The four-acre FPRQ site is part of a 182-acre parcel of land owned by Mr. and Mrs. Richard Lacey Compton of Blairs, Virginia. The site was leased from the Comptons by Mr. Ben Davenport, President of First Piedmont Corporation (FPC), located in Chatham, Virginia. FPC leased the four-acre site as First Piedmont Rock Quarry from April 1, 1970 until April 1, 1975. Disposal activities occurred on approximately two acres of the site during a period from April 1970 to July 1972 under the supervision of the Pittsylvania County Health Department.

In a letter dated June 1, 1981, Mr. Davenport of FPC was notified by Goodyear Tire and Rubber Company that some of the wastes generated at Goodyear's Danville plant and deposited at the FPRQ site were hazardous. Mr. Davenport filed a "Notification of Hazardous Waste Site" form with the U.S. Environmental Protection Agency on June 5, 1981, listing solvents as one of the wastes disposed of at the site.

Subsequently, the U.S. Environmental Protection Agency investigated the site pursuant to the National Contingency Plan (NCP). The site was scored in accord with the Hazard Ranking System (HRS) and received an initial hazardous ranking score of 37.51 in 1985. Goodyear Tire and Rubber Company, FPC, and Corning Glass Works were identified as the Principal Responsible Parties (PRP's). The site was subsequently rescored to 30.16, just above the minimum score of 28.5 required for listing on the National Priorities List (NPL), on the basis of comments received by the U.S. Environmental Protection Agency. The site was subsequently listed on the NPL on July 21, 1987 pursuant to Section 105 of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 as amended by the Superfund Amendments and Reauthorization Act (SARA) of 1986.

A consent order (Docket No. III-88-13-DC) was signed in December 1987. Under the terms of this order, the PRP's agreed to carry out a RI/FS at the site in accord with U.S. Environmental Protection Agency guidelines and a workplan prepared by a U.S. Environmental Protection Agency contractor (BCM Converse, 1987). In February 1988, Westinghouse Environmental and Geotechnical Services, Inc. of Cary, North Carolina, was contracted by the PRP's to perform the RI/FS at the site.

1.4 SITE HISTORY

This section presents a summary of the site history, which was described in greater detail in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b).

1.4.1 Ownership History

The property on which the FPRQ site is located was owned by Mr. J.F. Strictland until his death in 1963 (Richard Compton, personal communication, 1988). Mr. Richard Lacey Compton and his wife, Mae Milan Compton of Blairs, Virginia purchased the 182-acre farm, which included the quarry, from a bank in 1963 following Mr. Strictland's death. Mae Milan Compton's interest in the property passed to her husband, Richard, upon her death in 1965. Mr. Compton is the current owner of the property and was the sole owner at the time disposal operations were taking place.

Quarrying operations took place at the site when it was owned by Mr. Strictland (Richard Compton, personal communication, 1988). Although detailed information on the operation of the quarry is not available, it is known that the site was a source of crushed stone (Henika and Thayer, 1977).

By lease dated March 13, 1970, Richard L. Compton and Lois H. Compton, his wife (re-married), leased approximately four acres, including the FPRQ site, to Ben Davenport of the First Piedmont Corporation. The lease ran from April 1, 1970 to April 1, 1975, and stipulated that the site would be used as an industrial waste landfill. Of the four acres of the quarry site, about two acres were landfilled from April 1970 to July 1972. During the lease period, Goodyear Tire and Rubber Company, Corning Glass Works, and Southern Processor contributed industrial waste to the site.

1.4.2 Disposal History

At the time disposal operations began, the quarry contained approximately six inches of standing water. The actual floor of the quarry is relatively flat

at an elevation of about 630 feet above mean sea level (ft. msl). The east edge of the quarry floor is approximately 65 feet below current grade, whereas the western edge, which was the route of entry for trucks hauling industrial waste to the site (Ben Davenport, personal communication, 1988), is about two feet below grade.

The majority of material disposed of at the site consisted of industrial solid waste. Approximately 65,000 cubic yards of solid waste, 630,000 gallons of diatomaceous earth slurry, and 15,000 gallons of a waste water and residual degreaser mixture were disposed of at the site.

The quarry was not filled in a systematic fashion, that is, there were no cells or segregated disposal areas for specific wastes. Material brought to the site was generally landfilled in the most accessible location. Drums were repeatedly landfilled in random fashion with other solid waste. Slurried diatomaceous earth was transported by FPC in tanker trucks and discharged onto the solid waste. Solid waste, some which had been compacted, was typically hauled in 40 cubic yard containers. Since site security was minimal, unauthorized disposal is likely to have occurred. Due to the scarcity of cover material in the vicinity of the site, waste was not covered at the end of each day. As a result, the quarry contains almost exclusively industrial waste.

Waste sent by Goodyear consisted mainly of general plant refuse, such as cardboard and wooden pallets, but also included rubber buildup and residual MS-20 (a floor degreaser) that prior to use contained tetrachlororethylene. Corning shipped mainly industrial waste, such as paper and cardboard, along with off-specification glass to the FPRQ site. Southern Processors' waste included dirt removed from tobacco leaves, tobacco scrap, paper, and wood.

A fire occurred at the site in 1972. The Virginia State Health Department inspected the site and issued an order for its closure. FPC covered the site with a one- to two-foot thick cap. The cover material consisted of overburden taken from above the east edge of the quarry. No material tests were performed on the cover material. No drawings or documentation describing the cover or capping procedures are available.

In the early 1980's, FPC and Goodyear repaired parts of the cover where it had eroded. This was done with soil from the west side of the quarry using a front-end loader.

SECTION 2

DESCRIPTION OF SITE ENVIRONMENT

This section presents a description of the FPRQ site and its surrounding environment as a background for evaluation of potential human health and environmental effects associated with the site. The environment is described in terms of physical, biological, and human components. Figure 2 illustrates the immediate environs of the site, along with the Phase II monitoring locations.

2.1 PHYSICAL ENVIRONMENT

The following discussion of the physical environment summarizes information important to an understanding of the environmental dynamics of site contaminants. This information has been submitted previously in various technical memoranda and is discussed in more detail in the RI Report.

2.1.1 Physiography/Topography

The FPRQ site is located within the Piedmont Physiographic Province of southern Virginia. This area consists of rounded uplands dissected by numerous streams. Land surfaces in the vicinity of the site slope gently eastward at a grade of 0 to 8 percent (County of Pittsylvania, 1986). Local relief between the uplands and valleys is on the order of approximately 100 feet.

The FPRQ site itself is located along the northwest slope of a southwest-northeast trending ridge. Site topography varies from about 600 to 660 ft. msl. Local relief is high due to prior quarrying operations.

Surface drainage from the site is to Lawless Creek, about 1,400 feet to the northwest of the site. Lawless Creek is a tributary to Fall Creek, which in turn is a tributary to the Dan River.

2.1.2 Climate

Pittsylvania County has a temperate climate. The major factors which influence the climate include the County's mid-latitude location, prevailing southwesterly winds, and warm air off the Atlantic Ocean. Detailed climate data for Chatham, Virginia, located about 10 miles north of the site, as well as summaries of mean monthly climate information, were provided in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b). Tables 1 and 2 provide summary information on climatological data for Chatham, Virginia. The mean daily temperature varies from about 36°F in January to 76°F in July. Annual precipitation averages 43.48 inches and is relatively evenly distributed throughout the year. About 15 inches of snow falls per year. Wind direction is primarily from the south west throughout the year, except for the autumn months (August through October) when it is from the north.

Potential evapotranspiration, as calculated by the Thornthwaite method, is about 32 inches per year. Pan evaporation totals about 52 inches per year. Based on the precipitation and potential evapotranspiration data, there is a potential moisture excess of about 11 inches per year. This excess constitutes surface water runoff, changes in soil moisture storage, and ground water recharge. The precipitation excess occurs primarily during the winter months, when potential for evapotranspiration is low.

2.1.3 Geology

The regional geology in the vicinity of the FPRQ site consists of zero to three feet of weathered residuum and saprolite overlying Precambrian metamorphic rocks. Triassic-age dikes occur sporadically within the Precambrian rocks. Recent Quaternary-age unconsolidated sediments occur along major stream valleys. Bedrock in this area is the Precambrian Shelton Formation which consists of massive, lineated gneiss ranging in composition from quartz monzonite to granite (Henika and Thayer, 1977).

Two Triassic-age diabase dikes occur in the immediate vicinity of the site. One, along the southern edge of the site, appears to trend northwest-southeast. The other occurs downgradient of the site and trends more north-south.

Although major faults occur in the region, no faults have been mapped in the immediate vicinity of the site (Henika and Thayer, 1977). Geophysical survey data obtained during the Phase II RI indicate that the shallow bedrock is fractured, but that fractures probably do not occur at depths much greater than 50 to 100 feet.

Based on the response of the drilling rig during installation of site piezometers, the bedrock encountered was moderately fractured. The frequency of fractures encountered during drilling varied from 1 to 12 per foot. The water yield from the fractures was variable. No major fractures or fracture groups have been identified intersecting the landfill.

2.1.4 Soils

Most soils in the area are moderately to well drained sandy loams. The soils range from well developed on flat slopes to thin and poorly developed on

steep slopes. The U.S. Soil Conservation Service (1988) is currently completing a soil survey for Pittsylvania County. Preliminary U.S. Soil Conservation Service (SCS) soil data for the vicinity of the site has been provided in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b).

The soils in the vicinity of the FPRQ site have little sorptive capacity, as indicated by the results of the cation exchange capacity (CEC) and total organic carbon (TOC) analyses presented in the RI Report. The CEC values, which are a measure of inorganic sorptive capacity, range from 2.84 to 12.30 milliequivalents per 100 grams. These CEC values are typical of low clay soils and kaolinite clay minerals. The TOC values, which are a measure of organic sorptive capacity, range from 53 to 324 mg per kg. These TOC values are typical for soils developed from granitic parent material. The highest CEC values were reported in the northern drainage. The highest TOC values were reported from background soils north of the landfill.

2.1.5 Ground Water

Local ground water systems in the Piedmont Province typically occur within small areas coinciding with surface water basins. Little, if any, interbasinal flow occurs (LeGrand 1960 and 1985).

Ground water recharge occurs throughout the area as a result of infiltration of precipitation. Recharge rates are generally about 10 to 15 percent of mean annual precipitation (Heath, 1980). Based on a mean annual precipitation of 45 inches for the Danville area, this is equivalent to about 0.38 to 0.55 feet per year.

Ground water occurrence in the Piedmont Province is limited principally to a depth of less than 150 feet, with most water occurring within 30 feet of the surface (LeGrand, 1960). Flow is primarily horizontal. Within each ground water basin, most ground water is discharged to the surface at low points in the topography (springs) and/or along surface drainages.

Ground water at the FPRQ site occurs at depths ranging from 0 (at the land surface) to about 30 feet below land surface. Several areas within the landfill, such as the north and south ponds and the leachate seep, as well as springs along the adjacent site drainages, are indicative of the intersection of the water table and land surfaces.

Water level data for piezometers installed during Phase I of the RI show ground water to occur generally at depths of 3 to 25 feet below the land surface in areas adjacent to the landfill. These data indicate that in most locations ground water occurs only in the bedrock. In upland areas east of the site, where the overburden is thicker, or near ground water discharge areas, the lower portion of the residuum-saprolite may also be saturated.

Shallow ground water flow in the vicinity of the site generally follows topography. Recharge occurs principally in the upland areas with eventual discharge to local stream channels, as evidenced by the springs previously mentioned. Flow trends generally to the northwest, towards Lawless Creek.

Since most of the ground water flow is contained in the bedrock, it is controlled by bedrock fractures. Geophysical data indicate that the fractures are generally limited to the upper 50 to 100 feet of the bedrock. On a large scale, it is hypothesized that the shallow bedrock is sufficiently fractured so that the hydraulic regime of the shallow bedrock approaches that of a porous

medium. The local effect of the fractures may, however, allow ground water movement in preferential directions dictated by the fracture trends.

The private wells in the Beaver Park community are hydraulically upgradient from the landfill. Therefore, ground water cannot flow to the private wells from the landfill itself. In order for ground water at the landfill (elevation about 642 ft. msl) to flow toward the nearest private wells, the potentiometric elevations in the area between the landfill and the private wells would have to be lowered by 10 to 30 feet. This event is not possible given estimated private well use.

Shallow horizontal hydraulic gradients range from approximately 0.1 feet per feet in the upland area east and up gradient of the site to 0.15 to 0.18 feet per feet to the west and down gradient of the site. In the vicinity of the landfill itself, horizontal gradients are as low as 0.007 feet per feet, and shallow ground water is located just below the surface. Vertical hydraulic gradients are downward in the upland areas and upward along the discharge area (i.e., north and south drainages, and Lawless Creek). Site data indicate that the hydraulic conductivity of the shallow flow regime averages 0.16 feet per day and of the fractured bedrock averages 0.13 feet per day.

In the vicinity of the site, ground water discharges to the surface water system both up and down gradient of the landfill. The seeps and springs up gradient of the landfill and south of the Beaver Park community serve as ground water discharge points. The landfill is also a ground water discharge area for local ground water. The south and north ponds, and leachate seep, represent discharge areas where water is lost from the ground water system via evaporation and transpiration. However, most ground water at the site that is discharged

to the surface is likely discharged from the north pond to the surface water in the northern site drainage.

The hydraulic function of the landfill may be conceptualized as a small reservoir with a relatively impermeable bottom and slightly impermeable sides. The main outlet for this conceptual reservoir is at the north pond. Much of the ground water flow from upgradient areas, and ground water from recharge at the site, is likely discharged (i.e., spilled) to the surface water in the northern drainage at the north pond. Observations at the site indicate that most ground water discharged to the northern drainage re-infiltrates or is lost to the atmosphere via evapotranspiration. Figure 3 illustrates this concept.

There is little available specific information on regional ground water quality. Data presented in LeGrand (1960) for Pittsylvania County indicate that shallow ground water in the Precambrian rocks is usually a calcium-magnesium-bicarbonate type with low total dissolved solids. The ground water is generally hard, with a neutral to slightly acidic pH. Iron and manganese concentrations may be elevated. No data exist on trace element concentrations.

Ground water quality in the region of the FPRQ site was evaluated as part of the Phase II RI activities. Detailed data are provided in the Subtask 4.13 Technical Memorandum: Ground Water Sampling and Analysis (Westinghouse 1989e) and in the RI Report.

Up gradient, or background ground water samples, obtained from three wells, indicated a mixed cation-bicarbonate type water, with total dissolved solids (TDS) values of 143 to 175 mg per liter. In general, trace inorganic constituents were low or non-detectable in the background samples, with the exception of total iron concentrations in one sample.

Down gradient water quality was evaluated based on samples from eleven monitor wells. Down gradient ground water is also a mixed cation-bicarbonate type with TDS values generally equivalent to, or less than, those of the upgradient wells. There are some elevated inorganic constituents compared to background in some of the downgradient wells.

Within Pittsylvania County, 15 percent of the residents are served by central water systems that use a surface water source. The remaining residents, most of whom are in rural settings, rely on either individual ground water wells or privately owned surface or ground water systems (County of Pittsylvania, 1986). The City of Danville and adjacent areas use the Dan River as a primary water source for residential consumption and industrial applications.

2.1.6 Surface Water

The FPRQ site is located in a small drainage basin tributary to Lawless Creek. Surface water runoff from the site flows about 1400 feet to Lawless Creek, which is located to the north west of the site. Lawless Creek is a tributary to Fall Creek, which in turn discharges to the Dan River. Detailed information regarding surface water flow regimes and surface water quality in the vicinity of the FPRQ site is provided in the RI Report. A summary is provided below.

Surface water run-off from the site enters one of two drainages, the northern and southern drainages. Run-off from the site is generated in response to precipitation falling within the landfill area and as a result of ground water discharge. Some run-on to the site occurs from a small area immediately adjacent to the eastern highwall.

Mean annual flow from the northern and southern drainage basins, and at three locations along Lawless Creek, indicate that surface water discharge from the northern and southern drainages is very small and comprises less than one percent of the mean annual flow of Lawless Creek at the Route 719 bridge. Most of the discharge from the southern drainage originates upgradient of the site.

Mean annual flow estimates for the northern quarry and southern quarry drainages show that the northern quarry drainage contributes about 8.5 and 0.06 percent of the mean annual flow for the northern drainage and Lawless Creek at the Route 719 bridge, respectively. Similarly, the southern quarry drainage contributes about 1.6 and 0.03 percent of the mean annual flow for the southern drainage and Lawless Creek at the Route 719 bridge, respectively. These percentages represent significant dilution factors which may be the dominant factor controlling the fate of potential surface water contaminants and resulting adverse effects.

Sediment yield from the quarry is dependent on peak flow rates, soil erodability, length and steepness of slopes, and vegetative cover. Visual observations indicate that little sediment is transported beyond 500 feet west of the quarry in the northern drainage. Some of this initial sedimentation is the result of filtration by vegetation within the drainage area. Approximately 15.6 tons of soil per acre annually are estimated to be eroded from the site and transported to Lawless Creek. This sediment load undergoes considerable dilution from additional sediment bed load from upstream Lawless Creek. Detailed calculations of sediment loading are provided in the RI Report.

Lawless Creek, Fall Creek, and the Dan River are part of the Roanoke River Basin. Lawless Creek, Fall Creek, and the Dan River near Danville, Virginia are

Class III, Non-tidal Waters (Coastal and Piedmont) according to the Virginia Water Control Board (1987). The Dan River is classified as a public water supply approximately 15 miles upstream of Danville. The only significance of the Virginia Water Control Board classification is the variable dissolved oxygen, pH, and temperature standards assigned to different classes of water. None of these streams are recognized by the Virginia Water Control Board as "high quality surface water, public water supplies, or scenic rivers." The Virginia Department of Game and Inland Fisheries also has established a classification system for trout waters. Lawless Creek, Fall Creek, and the Dan River near Danville, Virginia are not recognized trout streams (Virginia Department of Game and Inland Fisheries, 1988). The Virginia Department of Game and Inland Fisheries reports that Lawless Creek is a warm water stream inhabited by sunfish, blue gills, catfish, and sucker. Lawless Creek does not appear to be used as a potable water supply. It is used for watering livestock, however.

2.2 BIOLOGICAL ENVIRONMENT

The following discussion of the biological environment provides a summary of information contained in the Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a) and in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b). This information is important to an understanding of the environmental dynamics of site contaminants.

The FPRQ site lies in the mixed deciduous forest biome of North America. This biome is characterized by a species-rich mixed mezophytic forest. More specifically, the site is located within an oak-pine forest association. Oaks and pines are the dominant tree species. Subdominant species include hickories,

beech, tulip poplar, and maples. The environmental factors which tend to control the species present are soil and topographic factors which affect soil moisture levels. The other controlling factor is succession, as species composition changes over time.

The physiography of the quarry itself has been discussed earlier in this memorandum. The quarry proper has undergone recent disturbance. Consequently, the vegetation reflects early stages of secondary succession. Small Virginia pine saplings (Pinus virginiana), goldenrods (Solidago spp.), asters, and grasses are prevalent. The vegetation is concentrated in moist areas and is sparse in areas with only a thin veneer of soil or in areas exposed to erosion.

2.2.1 Aquatic/Wetland Habitat

The Virginia Department of Game and Inland Fisheries classified streams in the vicinity of the site (Edwards Branch, Fall Creek, and Lawless Creek) as upper perennial, first or second order streams. None of the streams are trout waters.

A preliminary wetland delineation was performed at the FPRQ site to determine if any areas in the vicinity of the site would be classified as wetlands. The purpose of the preliminary delineation was not to define the exact wetland-upland boundaries, but to determine the location and type of wetlands, if any, in the vicinity of the site which may be influenced by actions occurring at the quarry. The methods and findings of this preliminary wetland delineation are presented in the Subtask 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a).

The simple jurisdictional approach outlined in the U.S. Environmental Protection Agency (1988) wetland manual was used in performing the wetland delineation. Three criteria were considered in determining if an area was to be classified as a wetland; hydrophytic vegetation, hydric soils, and wetland hydrology. All three of these criteria need to be met for classification as wetland. Only the cat-tail marsh located at the confluence of the northern drainage and the Lawless Creek flood plain meet all three criteria. This area and the other areas of hydrophytic vegetation that were identified are probably due to changes in the hydrology of the area caused by man's activities. Figure 4 presents the results of the wetland mapping.

Within the quarry, hydrophytic vegetation occurs around the north and south pond. This vegetation is due to the high ground water levels at these locations. The high ground water levels are in turn a result of the past quarrying and landfill operations. These areas do not exhibit hydric soils and the presence of hydrophytic vegetation is a consequence of anthropogenic (human) activities at the quarry site.

Similarly, the area of hydrophytic vegetation within the northern drainage is due to changes in the hydrologic regime caused by the quarrying and landfilling operations. This area was once upland woods. Landfilling at the quarry and discharge from the north pond changed the hydrologic regime and caused the vegetation in these area to adapt. Changes in the hydrology of this area killed the upland trees, creating a more open habitat in which the emergent hydrophytic vegetation developed on the wetter sites.

Those portions of the Lawless Creek flood plain not in pasture contain two types of areas. The flood plain is primarily a bottomland hardwood forest. A

small cat-tail marsh occurs at the confluence of the northern drainage and the Lawless Creek floodplain where it is locally wetter than the surrounding flood plain. Examination of aerial photographs of the vicinity of the FPRQ site indicate that the cat-tail marsh area was not present prior to landfilling. For this reason, it may also have been influenced by the change in the hydrologic regime.

The Virginia Department of Inland Fisheries and Game (1988) listed 56 fish species, 17 amphibians, 10 reptiles, 44 birds, 20 mammals, one aquatic mollusk, and four aquatic crustaceans occurring within the U.S.Geological Survey, Blairs quadrangle. The entire list of species was presented in the Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a). Fish collection records are also provided for Lawless, Fall, and Little Fall Creeks. Fish collected in these creeks are primarily shiners and chubs, which are considered to be non-game species. Though these species are not likely to be consumed by humans, they probably are an important source of food for non-human carnivores such as hawks, egrets, herons, osprey, turtles, minks, and otters which may inhabit the area.

2.2.2 Woodland Habitat

The surrounding plant community is primarily upland mesic mixed deciduous forest. These areas are dominated by white oak (Quercus alba), black oak (Q. nigra), red oak (Q. rubra), beech (Fagus grandifolia), tulip poplar (Liriodendron tulipifera), sweet gum (Liquidambar stryaciflua), and Virginia pines (Pinus virginiana). In the understory, sourwood (Oxydendrum arboreum), dogwood (Cornus

florida), ironwood (Carpinus caroliniana), and holly (Ilex opaca) are prevalent with a shrub layer of Vibernums and Vaccinium.

In areas which have been logged or in old abandoned agricultural fields or pasture land, there are often dense stands of Virginia pine. There are several dense stands of these pines within the vicinity of the quarry. As the pines mature and die, they are replaced by a variety of deciduous tree species to form mixed pine-hardwood stands.

Among the animals, there are a number of game, non-game, and pest species found in the woodland habitats of the Blairs quadrangle. A field tally by the Virginia Department of Inland Fisheries and Game (1988) listed 17 amphibians, 16 reptiles, 60 birds, 38 mammals, and four terrestrial invertebrates. A complete list of species that have been observed in the area was provided in the Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a).

2.2.3 Edge Habitat

The edge habitats are dominated by thick stands of aggressive and weedy trees, shrubs, and herbacious perennials. These include winged elm, (Ulmus alata), sweetgum (Liquidambar stryaciflua), raspberries (Rubus spp.), honeysuckles (Lonicera), and poison ivy (Rhus radicans).

The Virginia Department of Inland Fisheries and Game (1988) provided a breakdown for animal species associated with the pasture-woodland interface of the Blairs quadrangle. The dominant species in this habitat were birds, with 63 tallied. Other species included ten amphibians, 12 reptiles, 36 mammals, one terrestrial insect, and five other terrestrial invertebrates. A list of species

found in edge habitats was provided in the Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a).

2.2.4 Agricultural/Farm Species

Pasture land in the vicinity of the site is commonly comprised of various fescu grasses (Festuca spp.), legumes, and clovers. Many weeds, such as poke (Phytolacca americana), thistles, asters, and ragweed (Ambrosia spp.) are also significant components of the pasture habitat. Agricultural crops consist primarily of tobacco, with corn and soybeans as secondary crops.

Personnel of the Soil Conservation Service state that there are no dairy, sheep, or hog farms along Lawless Creek in the vicinity of the site. There are an estimated 100 head of beef cattle which may consume water from Lawless Creek. Most of these cattle are located on a farm owned by John Lumsford.

2.2.5 Threatened and/or Endangered Species

There are no state (Virginia) or federally listed threatened or endangered species known to occur in the immediate area of the FPRQ site according to the Virginia Department of Game and Inland Fisheries (1988).

2.3 HUMAN ENVIRONMENT

The following discussion of the human environment provides a summary of information contained in the Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a) and in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b) that is important to an understanding of the environmental dynamics of the site vicinity.

2.3.1 Land Use

The FPRQ site is located in a rural setting just north of Danville, Virginia. The vicinity of the site is relatively sparsely populated and much of the land is wooded, or is in pasture or cropland.

Aerial photographs and records maintained by the SCS (U.S. Soil Conservation Service, 1988) indicate that farmland, forests, and residential areas occur in the vicinity of the site. The largest land use areas are forested, which cover approximately 70 percent of the land within one mile of the FPRQ site. Some of the forested areas are timbered and some are abandoned pasture and farmland.

Crop production and pasture land account for approximately 20 percent of the land use in the vicinity of the site. Tobacco and corn are the primary crops and cattle the primary livestock.

There are several clusters of unincorporated residential areas within one mile of the site. These account for approximately 10 percent of the land use. Beaver Park, a community of approximately 260 persons, is the largest of these residential areas and is located immediately southeast of the site.

2.3.2 Human Populations

The Task 2.2 Technical Memorandum: Sensitive Receptor Survey (Westinghouse 1988a) provides a detailed discussion of demographic data and estimates of the human population in the Beaver Park Community, for areas within one and two miles of the site, and for Pittsylvania County. In addition, the memorandum includes a drawing illustrating property lines and structures within two miles of the site and provides a listing of property owners within two-miles of the site, with references to tax map and parcel numbers.

The population density of Pittsylvania County is estimated to be 2.99 persons per household (County of Pittsylvania, 1986). The County has experienced an average growth rate of 1.2 percent over the ten year period 1970 to 1980 (County of Pittsylvania, 1986). Table 3 provides a breakdown of the County population by age bracket and gender. The percentage of the County population within an age bracket may be used to estimate the number of people in the same bracket living within the area of concern. Sensitive populations, for purposes of risk assessment, would be those under five and over 65 years of age.

Within one mile of the site, the human population is estimated to range from 455 to 600 people. Within two-miles of the site, the human population is estimated to range from 1,893 to 2,225 people.

The community of Beaver Park is the residential area in the immediate vicinity of the site. Numerous methods were employed to estimate the population of Beaver Park, including aerial photographs, U.S. Geological Survey maps, County tax records, and a site reconnaissance. Table 4 presents the various estimates which are based on counting of residential structures, and the demographic data presented above. The estimates based on the site reconnaissance and the 1988 photograph are considered to be the most accurate.

2.3.3 Historical and Archaeological Resources

No historical or archaeological resources have been identified in the vicinity of the FPRQ site.

SECTION 3

SITE CONTAMINANTS

This section briefly reviews the history of sampling activities at the FPRQ site and the process by which a suite of indicator compounds was selected for assessment of potential human health risk associated with the site.

3.1 PRE-RI/FS STUDIES

Based on source sampling results (Subtask 4.5 Technical Memorandum: Source Area Sampling (Westinghouse 1988e)) and historical site data (Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988a)), target constituents were identified for detailed characterization in the Subtask 4.6 Technical Memorandum: Waste Characterization (Westinghouse 1989b). These constituents, listed in Table 5, include:

1. all organic constituents detected, including those below the contract required quantitation limits (CRQL) but above the instrument detection limits (IDL);
2. all inorganic compounds detected above the contract required detection limits (CRDL); and
3. those constituents suspected of being present at the site.

It should be noted that many of the organic constituents were detected only once or twice and/or were below the CRQL.

The only exception to these criteria for selecting the initial suite of target constituents are for the major ions aluminum, calcium, magnesium, sodium, and potassium. These cations are not included among the target constituents

because they are the major constituents in most waters, natural soils, and sediments; generally not found in elevated concentrations above background levels; and have a low toxicity. Although aluminum is toxic to aquatic life it was not found in high concentrations at the FPRQ site. In addition, no tentatively identified compounds outside the Target Analyte List (TAL) and Target Compound List (TCL) parameters are included.

3.2 RI/FS SAMPLING RESULTS

Sampling efforts conducted during the FPRQ RI/FS are summarized below. RI sampling events consisted of source area sampling conducted by Westinghouse during the Phase I portion of the RI, private well sampling in the community of Beaver Park conducted by CDM Federal Systems under contract to U.S. Environmental Protection Agency Region III, and two rounds of multimedia sampling conducted by Westinghouse during Phase II of the RI. In addition, surface water and sediment samples collected by Westinghouse during Phase II Round 2 sampling of the RI were tested for toxicity to aquatic organisms by means of bioassay testing. The results of this latter effort are discussed in Section 8.0 of this technical memorandum as well as in the Subtask 4.14 Technical Memorandum: Bioassessment Sampling and Analysis (Westinghouse 1989f).

3.2.1 Source Area Sampling Results

During September 1988, eight environmental samples (five solid and three aqueous) were collected from selected locations at the site, including the north and south ponds, a leachate seep at the head of the north drainage, a waste pile (solid), and two drums on site as part of the RI Phase I sampling. In addition,

duplicate samples and various sample blanks were collected to evaluate data quality.

The source area sampling results are generally consistent with historical data. However, fewer inorganic constituents were detected, and those detected were found in generally lower concentrations in these later analyses compared to previous analyses.

The inorganic analytical data indicate that the compounds arsenic, barium, cadmium, lead, and zinc were found in the greatest number of samples and in the most elevated concentrations compared to background. Arsenic, barium, and zinc concentrations in the leachate sediment, as well as cadmium, lead, and zinc values in the north pond sediment, waste pile, and the two drums were above the concentration range of natural soils. The same metals found in the solids samples also were found at concentrations elevated above background in the north pond and leachate aqueous samples. However, only arsenic and barium concentrations exceeded Federal Primary Drinking Water Standards. These inorganic data indicate that, except for arsenic and barium, trace metals present in the solid matrix are not being appreciably mobilized into the aqueous matrix.

The organic data for the solid samples indicate only one compound detected above the CRQL; xylene at 1,300 ug per liter in one sample, taken from one of the drums. The results of the aqueous sample analyses indicate only 9 ug per liter of benzene, 13 ug per liter of xylene, and 80 ug per liter of n-nitrosodiphenylamine were detected in the leachate. These concentrations are just above the CRQL, thereby, implying some question as to their actual occurrence.

3.2.2 Private Well Sampling Results

On November 30 and December 1, 1988, U.S. Environmental Protection Agency Region III personnel and U.S. Environmental Protection Agency contractor personnel from CDM Federal Programs, Inc. sampled private water wells in the vicinity of the FPRQ site near Danville, Virginia. Westinghouse personnel were present during the sampling effort.

Ten aqueous samples were taken from the private water sources for analysis by U.S. Environmental Protection Agency and contractor laboratories. In addition, one (1) field blank and one (1) field duplicate pair were prepared for analysis. Samples were analyzed for TAL and TCL constituents. The identification of samples and sample sites is presented in the RI Report.

No compounds, target or non-target, were detected in the volatile, semi-volatile, pesticide, or PCB analyses for any of the samples. Only for two wells were any inorganics reported above drinking water standards. The Stone well had 466 ug per liter iron, above the Secondary Drinking Water criterion for iron of 300 ug per liter. The Juanita Wadell well had 333 ug per liter iron and 65.1 ug per liter manganese, above the Secondary Drinking Water criteria for iron and manganese of 300 ug per liter and 50 ug per liter, respectively.

Previous data from private wells in the area, which were summarized in the Task 3 Technical Memorandum (Westinghouse 1988b), suggest elevated concentrations of a few inorganics at some wells. These results confirm that the landfill has not affected any private wells in the area.

3.2.3 RI Phase II Sampling Results

Sampling activities conducted during Phase II of the FPRQ RI consisted of multimedia sampling for chemical analysis of soils in the quarry and vicinity, surface water and sediments, and ground water. In addition, surface water and sediment samples were obtained from surface water drainages for biological toxicity testing of both water and sediment elutriate. The results of the analyses of these samples are provided in the RI Report as well as in the appropriate individual technical memoranda.

3.3 INDICATOR CHEMICAL SELECTION

It is not realistic or cost effective to evaluate the potential risk of each of the chemicals that have been associated with the site, as many of these chemicals are found at relatively low concentration and/or are not ubiquitous at the site. In accord with U.S. Environmental Protection Agency guidance (1986), Westinghouse selected compounds of particular concern to serve as indicator chemicals representing constituents of "highest risk", thus permitting a public health evaluation that focuses on the chemicals of greatest potential concern at the site. Appropriate tables illustrating the indicator selection process are provided as Appendix A. In addition to the twelve chemicals selected by this process, manganese and bis (2-ethylhexyl) phthalate were evaluated, based on concentration and potential toxicity.

Analytical results from samples taken during the RI sampling events were evaluated and the chemicals present at FPRQ above the detection limit were identified. Due to the number of chemicals identified as being present above the detection limit, indicator chemicals were selected on the basis of both maximum and representative concentrations and route-specific toxicity data.

The first step involved listing all chemicals detected at the site, along with their concentration ranges and representative concentrations, in source areas, leachate, ground water, surface water, sediments, and soil. The ranges listed as "<x" in the tables in Appendix A indicate that the lower range was below the detection limit. The representative concentrations were calculated based on the mean of only those samples where the chemical was detected (i.e., above the detection limit). Also included are the chemicals' environmental mobility as reflected by their organic carbon partition coefficient values (K_{oc}) taken from tables provided in U.S. Environmental Protection Agency, 1986. This coefficient is a measure of the tendency for organics to be absorbed by soil and sediment and is calculated as:

$$Koc = \frac{\text{mg chemical adsorbed/kg organic carbon}}{\text{mg chemical dissolved/liter of solution}}$$

As the definition indicates, inorganics do not have a K_{oc} value. The comments columns in tables A-1 through A-6 indicate the frequency of detection in the three media: ground water, surface water, and soil. Only the inorganic concentrations in soil are in units of mg per kg. All other concentrations are in micrograms (ug), either ug/l for ground and surface water concentrations or ug/kg for soil concentrations.

Second, each compound was classified as a potential carcinogen or noncarcinogen, or both. Each compound was also tabulated according to whether or not it is an inorganic or organic compound.

For noncarcinogens, the severity of effect rating value was identified from appropriate tables in U.S. Environmental Protection Agency (1986) for the oral route of exposure and/or for the inhalation route. This unitless value, ranging

from 1 (low) to 10 (high), is an indication of the chemical's severity of effect. A rating of 1 indicates a biochemical change with no pathologic changes or change in organ weights. A rating of 5 indicates reversible cellular changes, and a value of 10 indicates death or pronounced life-shortening. A full description of these values is given in Exhibit B-1 of U.S. Environmental Protection Agency (1986).

For carcinogens, a qualitative weight of evidence rating was identified based on U.S. Environmental Protection Agency categories for potential carcinogens. These ratings qualify the level of evidence that supports a carcinogen classification as follows.

- Group A - human carcinogen based on sufficient evidence from epidemiologic studies.
- Group B1 - probable human carcinogen based on limited evidence in human epidemiologic studies.
- Group B2 - probable human carcinogen, but is based on sufficient evidence of carcinogenicity in animals.
- Group C - possible human carcinogen.
- Group D - not classified with regard to carcinogenicity.
- Group E - no evidence of carcinogenicity in humans.

Following identification of severity and/or weight-of-evidence, the appropriate toxicity constants, again obtained from tables in U.S. Environmental Protection Agency (1986), are listed (w_T, s_T, and a_T for water, soil, and air media, respectively). These constants, which represent the ingestion route of exposure for water and soil, and the inhalation route of exposure for air, are in units of $(mg/l)^{-1}$ for water, $(mg/kg)^{-1}$ for soil, and $(mg/m^3)^{-1}$ for air.

Chemicals are still listed even if no toxicity constants were available, as indicated with a "--" in the tables in Appendix A.

Carcinogens (both inorganic and organic chemicals) are tabulated and their indicator scores (IS) calculated. IS values are based on concentration values multiplied by toxicity constants for each media: ground water, surface water, soil, and air. The concentration values utilized are both the maximum values obtained and the representative concentrations calculated in each media (as indicated in the values given in the first step), and the two concentrations, maximum and representative, are kept separate. At the FPRQ site, since there has been no air sampling, no values were determined for the air media. The resulting Concentration times the Toxicity (CT) value is unitless. Since the chemical concentrations in ground water, surface water, and soil for organics were calculated in terms of microgram units, these values must be converted to milligram units for the CT product to be unitless. Then, the higher of the ground water or surface water CT value is summed with the soil CT value to give the IS (Indicator Score) value for both the maximum and representative concentrations. This summation assumes that all drinking water is obtained from the source with the higher CT value. Then each chemical with an IS value is ranked from high to low. Those chemicals that are not present in a media are indicated with a CT value of zero. For those without the appropriate toxicity constant but with chemical present in the media, the CT product is indicated with a "--" in the tables in Appendix A, and are not included in the ranking process. This same procedure is repeated for the noncarcinogens.

Finally the carcinogens and noncarcinogens are combined and ranked according to their IS scores. Several chemicals were present in one or more of

the various media and had high toxicity values, but were not included in the final indicator chemical list for the following reasons.

1) Beryllium's toxicity (both as a carcinogen and as a noncarcinogen) is via the inhalation route of exposure and not due to the ingestion route. Beryllium's air concentrations would be minimal if detectable at all at FPRQ and therefore beryllium was not included as an indicator chemical.

2) While cadmium is not ranked as a carcinogen it is included as a noncarcinogen indicator chemical.

3) Chromium had no samples above the detection limit in the surface water and was found at low concentrations in the soil. The ground water only had one sample above 32 ug per liter (141 ug/l).

4) Aluminum, calcium, cobalt, magnesium, potassium, sodium and sulfate are naturally occurring chemicals with normally high background concentrations and were not included for this reason.

5) Acetone and methylene chloride were detectable in all media but at fairly low concentrations. The ranges and representative concentrations were:

		Acetone	Methylene Chloride
Ground water:	Range:	<3-16 ug/l	3 ug/l
	Representative Conc:	6.6 ug/l	3 ug/l
Surface Water:	Range:	<7-18 ug/l	3 ug/l
	Representative Conc:	12 ug/l	3 ug/l
Soil:	Range:	<4-42 ug/kg	3-24 ug/kg
	Representative Conc:	13 ug/kg	8.2 ug/kg

Therefore these solvents were not included as indicator chemicals.

6) Benzo(a)anthracene, Benzo(b)fluoranthene, Benzo(k)fluoranthene, chrysene, pyrene, 1,2-Dichloroethene, 2,6-Dinitrotoluene, 2-Butanone, chloroform, ethylbenzene, 2-Hexanone, 4-Methyl-2-pentanone, Naphthalene, phenanthrene, phenol and toluene were not included as indicator chemicals as there were no detectable samples in either the ground water or the surface water and only 1 or 2 barely detectable samples each in the soil.

7) Xylene was only detectable in 2 samples in the soil, only 1 barely detectable sample in the ground water, and was not detectable in the surface water. It would therefore not be a good candidate for an indicator chemical.

8) Chlorobenzene was not detectable in the ground water or the soil and only 1 sample at 3 ug per liter was found in the surface water. It also would not be a good candidate as an indicator chemical.

9) N-Nitrosodiphenylamine was tentatively identified only in the soil at concentrations ranging from 1200 to 9100 ug per kg, with a representative concentration of 7975 ug per kg. Based on the low carcinogenic potency (q_1^*) of 4.92 E-3 mg per kg per day, the risk corresponding to estimated lifetime cancer risk of 10^{-6} would require cleanup only when the soil concentration reached 800 to 1000 ppm. The concentrations at FPRQ are orders of magnitude below this threshold risk level at 1 to 9 ppm, even if indeed N-Nitrosodiphenylamine were actually present. Therefore, based on this preliminary assessment for N-Nitrosodiphenylamine, it would not be a good candidate as an indicator chemical, with other organics with higher carcinogenic potencies also identified at FPRQ.

The Chemical Indicator Selection Process narrowed the field to the following twelve chemicals:

1) Antimony
2) Arsenic
3) Barium
4) Benzene
5) Cadmium
6) Copper
7) Lead
8) Mercury
9) Nickel
10) Selenium
11) Vanadium
12) Zinc

In addition, manganese and bis (2 ethylhexyl) phthalate were added to the final indicator chemical list.

The chemical characteristics, toxicology, and environmental fate and behavior of these fourteen indicator chemicals are discussed briefly in Section 4 of this technical memorandum and described in more detail in Appendix B. Section 4 also provides a summary of dose-response information for these indicator chemicals.

SECTION 4

TOXICOLOGICAL CHARACTERISTICS

A brief review of the toxicological characteristics, which form the basis for the U.S. Environmental Protection Agency risk assessment actions, are included below for each of the twelve indicator chemicals selected for the FPRQ site. For further toxicological information, see the toxicological profiles of each of the twelve indicator chemicals in Appendix B. A table listing the regulatory risk assessment data and a table delineating environmental fate and behavior for each of the twelve indicator chemicals are included as Appendix C.

4.1 ANTIMONY

Antimony is classified as a noncarcinogen and is listed with a severity rating of 10 by the oral route of exposure (U.S. Environmental Protection Agency 1986). This rating is given for compounds which produce death or pronounced life shortening effects as well as teratogenic effects without signs of maternal toxicity. Antimony also has a severity rating of 8 by the inhalation route of exposure. This is indicative of compounds producing necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction. According to the U.S. Environmental Protection Agency (1980), multimedia environmental antimony exposures are essentially negligible by comparison to those occupational exposures at which clinical health effects have been observed. The best characterized human health effect associated with antimony exposure is myocardial damage (IRIS, 1989). The only data supporting this health effect are inhalation exposure studies. These studies suggest a NOEL of 0.5 mg per cubic

meter (Brieger et al, 1954), which can be extrapolated to an oral reference dose of 0.003 mg per kg body weight per day. However, there are no adequate data on oral exposure to antimony to permit estimation of no effect levels associated with heart damage (IRIS, 1989). The U.S. Environmental Protection Agency (1986 and IRIS, 1989) has published an oral reference dose of 4E-4 mg per kg per day for chronic ingestion of antimony, based on a rat chronic oral bioassay that measured longevity, blood glucose, and cholesterol as the critical effects.

4.2 ARSENIC

Arsenic is classified as both a noncarcinogen and carcinogen by the U.S. Environmental Protection Agency. It is listed as a Group A Human Carcinogen with sufficient evidence from epidemiologic studies to support a causal connection between exposure and cancer (IRIS, 1989). This listing is based on observations of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations. Based on the increased prevalence of skin cancer (Tseng, 1977), a unit carcinogenic risk from oral exposure of 5E-5 per ug per liter (IRIS, 1989) was proposed. However, due to its essential nutrient value, the MCLG was only set at 0.05 mg per liter, the same as the MCL (National Academy of Science, 1983). However, the status of arsenic as an essential nutrient is currently the subject of scientific controversy. Arsenic is also listed with a noncarcinogen severity rating of 9 (U.S. Environmental Protection Agency, 1986). This listing is indicative of pronounced pathologic changes with severe organ or reproductive dysfunction or teratogenic effects with maternal toxicity.

4.3 BARIUM

Barium is classified as a noncarcinogen with a severity rating of 10 (U.S. Environmental Protection Agency, 1986). It has not been evaluated by the U.S. Environmental Protection Agency for evidence of human carcinogenic potential. The oral reference dose of 5E-2 mg per kg body weight per day is based on a NOEL of 10 ppm (0.51 mg/kg/day) and a LOAEL of 100 ppm (5.1 mg/kg/day)(IRIS, 1989). Systolic blood pressure measurements were monitored as the critical effect in the studies supporting this value. An MCLG of 1.5 mg per liter is proposed for barium, based on a LOAEL of 5.1 mg per kg per day and consumption of 2 liters of water per day, as well as an indication that 83 percent is the source contribution from drinking water (i.e., 0.7 mg/day) (IRIS, 1989).

4.4 BENZENE

Benzene is classified as a Group A human carcinogen, based on several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, as well as increased incidence of neoplasia in rats and mice exposed by inhalation and gavage. The U.S. Environmental Protection Agency lists a drinking water unit risk of 8.3E-7 per ug per liter with an oral slope factor of 2.9E-2 per mg per kg per day (IRIS, 1989). These data were derived from human exposure data (Rinsky et al., 1981 and Wong et al., 1983). The drinking water unit risk was calculated on the assumption that an adult consumes two liters of water per day. The risk level of 1 in 10,000 is associated with a drinking water concentration of 1E+2 ug per liter and the 1 in 1,000,000 risk level is associated with a concentration of 1E+0 ug per liter (IRIS, 1989). The One-day Health Advisory (HA) for a child is not available but the Ten-day Health

Advisory of 0.234 mg per liter (with a NOAEL of 2.35 mg/kg/day) is recommended as the One-day HA (IRIS, 1989). This recommendation assumes a 1 liter per day consumption for a 10-kg child. There are no longer-term health advisories for either a child or an adult because of benzene's potent carcinogenicity. An MCLG of 0 mg per liter was set as a final value in 1985, based on the carcinogenic effects of myelocytic anemia, thrombocytopenia, and acute myelogenous and monocytic leukemia. An MCL of 5 ug per liter was set as a final value in 1987. Also, the inhalation slope factor was set at 2.9E-2 per mg per kg body weight per day with an inhalation unit risk of 8.3E-6 per ug per cubic meter. The air concentrations associated with a risk level of 1 in 10,000 is 1E+1 ug per cubic meter and the risk level of 1 in 1,000,000 is associated with an air concentration of 1E-1 ug per cubic meter (IRIS, 1989).

4.5 BIS (2-ETHYLHEXYL) PHTHALATE

The U.S. Environmental Protection Agency has listed bis (2-ethylhexyl) phthalate (DEHP) as a probable human carcinogen, classified as weight-of-evidence Group B2. A number of studies have been conducted to investigate the acute toxic effects of DEHP. When administered by the oral, intraperitoneal, intravenous, and inhalation routes, DEHP has a low order of acute toxicity. The target organs for DEHP appear to be the liver and testes. At relatively high dose levels, DEHP has been found to induce morphological and biochemical changes in the liver of exposed rodents. Similar effects have been reported for a number of chemicals which induce hepatic xenobiotic metabolizing capabilities. The testicular effects of DEHP are characterized by a decrease in relative organ weight and damage to the seminiferous tubules. Similar effects have been reported in animals treated with a major metabolite of DEHP. Studies in rats and mice

suggest that DEHP is developmentally toxic. MCLs have not been established for bis (2-ethylhexyl) phthalate under the SDWA. The only standard set by the SWCB is for protection of aquatic life (3.0 ug/l). Water quality criteria have been established under the CWA both for drinking water and consuming aquatic organisms (15 mg/l) and from fish consumption alone (50 mg/l).

4.6 CADMIUM

Cadmium is listed as a Group B1 probable human carcinogen by inhalation. This classification is based on limited epidemiologic studies (i.e., a two-fold excess risk of lung cancer in cadmium smelter workers) as well as sufficient evidence of carcinogenicity in rats and mice. A statistically significant positive association with prostate cancer was also observed. The quantitative estimate of carcinogenic risk is 6E-2 ug per cubic meter for 1 in 10,000 incremental cancer incidence and 6E-4 ug per cubic meter for 1 in 1,000,000 incremental cancer incidence (IRIS, 1989). Studies of human ingestion are inadequate at present to assess carcinogenicity of cadmium by ingestion. The U.S. Environmental Protection Agency intends to add cadmium to the list of hazardous air pollutants and establish emission standards under Section 112(b)(1)(A) of the Clean Air Act (50 FR 42000 (16 October 1985)). Cadmium is also listed with a noncarcinogenic severity rating of 10 (U.S. Environmental Protection Agency, 1986). Cadmium has an oral reference dose of 5E-4 mg per kg body weight per day (water) based on the critical effect of significant proteinuria. It also has an oral reference dose of 1E-3 mg per kg per day (food) based on several human studies involving chronic exposures (IRIS, 1989). The NOAELs in water and food are 0.005 and 0.01 mg per kg per day, respectively. These levels are based on the maximum human renal cortex concentration of 200 ug

aspects of children's neurobehavioral development) at very low blood levels such that there may be no threshold concentration for non-carcinogenic effects of lead toxicity. Lead is classified as a B2 probable human carcinogen on the basis of sufficient animal evidence. However human evidence is inadequate. The U.S. Environmental Protection Agency Carcinogen Assessment Group has not recommended a numerical estimate of lead's carcinogenic risk potential by either the oral or inhalation route of exposure. This lack of recommendation is due to the many uncertainties (e.g., age, health, nutritional state, body burden, and exposure duration) which may influence absorption, release, and excretion of lead (IRIS, 1989). An MCLG of 0.02 mg per liter has been proposed in 1985 (50 FR 46936 Part IV (13 November 1985)) based on the neurological effects of lead in infants and the adverse effects associated with blood lead levels of 15 ug per deciliter (i.e., the U.S. Environmental Protection Agency utilized a conversion factor of 6.25 to convert from blood lead levels to drinking water, along with an uncertainty factor of 5 (IRIS, 1989)). An MCL has been issued as an interim level in 1980 at 0.05 mg per liter (45 FR 57332 (27 August 1980))

4.9 MANGANESE

In humans, chronic exposure to manganese causes degenerative changes in the central nervous system in the form of Parkinson-like disease. Modifications to liver functions can also occur. Acute exposure causes manganese pneumonitis. In humans, manganese dusts and compounds have relatively low oral and dermal toxicity, but they can cause a variety of toxic effects after inhalation exposure. Acute exposure to very high concentrations can cause manganese pneumonitis, increased susceptibility to respiratory disease, and pathologic changes including epithelial necrosis and mononuclear proliferation. Chronic

exposure is more common, but generally only occurs among persons occupationally exposed. Degenerative changes in the central nervous system are the major toxic effects. Individuals with an iron deficiency may be more susceptible to chronic poisoning.

4.10 MERCURY

Inorganic mercury is classified as a noncarcinogen with a severity rating of 7 by the oral route (i.e., indicative of necrosis, atrophy, hypertrophy, or metaplasia with a detectable decrement of organ functions, as well as any neuropathy with a measurable change in behavioral, sensory, or physiologic activity) and a rating of 8 by the inhalation route of exposure (i.e., indicative of necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction as well as any neuropathy with gross changes in behavior, sensory or motor performance and decrease in reproductive capacity and evidence of fetotoxicity)(U.S. Environmental Protection Agency 1986). Mercury is also listed by the U.S. Environmental Protection Agency with a carcinogen classification of D, not classifiable as to human carcinogenicity due to no human data available and animal and supporting data inadequate (IRIS, 1989). There are no quantitative estimates of carcinogenic risk from either the oral or inhalation routes of exposure (IRIS, 1989). The MCL has been established at 0.002 mg per liter and the proposed MCLG is 0.002 mg per liter (see also U.S. Environmental Protection Agency, 1987).

4.11 NICKEL

Nickel (soluble salts) is listed as a noncarcinogen with a severity rating of 10 for both the oral and inhalation routes of exposure (U.S. Environmental

Protection Agency 1986). Numerous cases of dermatitis have been reported (Clayton and Clayton, 1981-82). Signs of exposure include nausea, vomiting, diarrhea, and central nervous system depression (Weiss, 1980) as well as coughing, shortness of breath, chest pain, fever and weakness upon inhalation (Rumack, 1975 to Present). The Reference Dose (RfD), established by the U.S. Environmental Protection Agency, is based on the assumption that thresholds exist for certain toxic effects for nickel. These effects include cellular necrosis, but may not be appropriate for other effects such as carcinogenicity (IRIS, 1989). The oral RfD has been established at 2E-2 mg per kg body weight per day, based on a NOAEL of 5 mg per kg body weight per day (assuming 1 ppm = 0.05 mg kg body weight per day for rat food consumption) and a LOAEL of 50 mg per kg body weight per day. The critical effect monitored was decreased body and organ weights (Ambrose et al., 1976). Female rats exhibited significantly higher heart-to-body weight ratios and lower liver-to-body weight ratios than controls (Ambrose et al., 1976, see also U.S. Environmental Protection Agency, 1983). Nickel (soluble salts) has not been evaluated as a class by the U.S. Environmental Protection Agency for potential human carcinogenicity, however it is listed in the Superfund Public Health Evaluation Manual (U.S Environmental Protection Agency, 1986) with an EPA Weight-of-Evidence Category of A for both the oral and inhalation routes of exposure.

4.12 SELENIUM

Selenium is listed as a noncarcinogen with a severity rating of 10 for both the oral and inhalation routes of exposure (U.S Environmental Protection Agency, 1986). The U.S. Environmental Protection Agency has recommended an RfD for selenium in drinking water of 0.21 mg per day (or 0.003 mg/kg/day), based on an

LOAEL of 3.2 mg per day (Yang et al., 1983) and an uncertainty factor of 15 (IRIS, 1989). An uncertainty factor of 15 rather than 10 was utilized since selenium appears to be more efficiently absorbed in water than in food and the study was conducted in the diet. The National Academy of Science has determined an adequate and safe range for selenium intake of 50 to 200 ug per day for an adult man (National Academy of Science, 1980). The effects of selenium deficiency are potentially as serious as those of selenium toxicity (IRIS 1989). Selenosis has been reported due to high selenium intake ranging from 3.2 to 6.7 mg per day (average 5 mg/day). No selenosis occurred when the average intake was 0.75 mg per day (range was 0.24 to 1.51 mg/day) (Yang et al., 1983). Inorganic selenium compounds may cause dermatitis (Sax, 1984). Signs of exposure include garlic odor of breath, as well as pallor, nervousness, depression, and digestive disturbances (Sax, 1984; see also U.S. Environmental Protection Agency, 1985b).

4.13 VANADIUM

Vanadium is listed as a noncarcinogen with a severity rating of 1 for both the oral and inhalation routes of exposure (U.S. Environmental Protection Agency, 1986). This is indicative of enzyme induction or other biochemical change with no pathologic changes and no change in organ weights. An oral RfD has been recommended by the U.S. Environmental Protection Agency, based on the assumptions that thresholds exist for certain toxic effects, but may not exist for other toxic effects, such as carcinogenicity. The oral RfD is 9E-3 mg per kg per day (or 0.62 mg/day for a 70 kg adult) and the criteria used to evaluate vanadium toxicity were growth rate, survival, and hair cysteine content (Stockinger et al., 1953). The NOAEL for vanadium was 0.89 mg per kg per day and there was no LOAEL reported. The only significant change reported was a decrease in the

amount of cysteine in the hair, while other studies have reported a significant decrease in erythrocyte and hemoglobin levels (Mountain et al., 1953). Inhalation studies are inadequate at present to determine a minimum effective dose (IRIS, 1989). Respiratory symptoms of exposure to high concentrations usually subside within 7 to 14 days (IRIS, 1989). The probable oral lethal dose of vanadium pentoxide is approximately 5 to 50 mg per kg (Gosselin et al., 1984). Vanadium generally aggravates chronic respiratory diseases and can cause death by pulmonary edema. It is also known to cause irritation and redness in eyes and skin. Ingestion causes irritation of mouth and stomach, vomiting, abdominal spasms, and a green discoloration of the tongue (IRIS, 1989).

4.14 ZINC

Zinc is listed as a noncarcinogen with a severity rating of 8 for both the oral and inhalation routes of exposure (U.S. Environmental Protection Agency, 1986). Symptoms of zinc poisoning include lassitude, slower tendon reflexes, bloody enteritis, diarrhea, lowered leukocyte count, and central nervous system depression (Venugopal, 1978). There is no evidence that zinc is carcinogenic. A secondary Safe Drinking Water Act standard for zinc of 5 mg per liter has been set and adopted by the Surface Water Control Board.

SECTION 5

TRANSPORT PATHWAYS AND POTENTIAL EXPOSURE POINTS

For any particular site, there may be a variety of potential exposure routes, both simple and multipathway. The simple pathways are of primary significance at the FPRQ site. Such simple exposure routes for humans include consumption of ground water, bathing in ground water, consumption of surface water, bathing or play in surface water, ingestion of soil, dermal exposure to soil, and inhalation of fugitive dust emissions. Of these, ingestion pathways are the most important, based on site constituents and contaminant distribution. Multipathway exposure routes are pathways such as human ingestion of fish from the Dan River or Lawless Creek, human ingestion of milk from cows that have grazed along Lawless Creek, and human ingestion of game from the site. These multipathway exposure routes are significantly less important at the FPRQ site than direct pathways because the site contaminants, which are primarily metals, do not bioaccumulate to the extent that many organics do, nor are they relatively mobile in the environment. Furthermore, site sampling data indicate minimal off-site migration of contaminants in any environmental media.

This section describes the pathways by which materials could potentially be transported from the FPRQ site. In addition, potential exposure points for human and biotic exposure to site contaminants are identified. Finally, exposure point scenarios for which quantification of exposures is appropriate are identified. As appropriate, quantitative estimates of exposure for these exposure points are provided in Section 6.0 for humans and in Section 8.0 for biota.

5.1 TRANSPORT PATHWAYS

A preliminary set of four exposure routes, or transport pathways, was identified in the technical Memoranda for Subtask 2.2: Sensitive Receptor Survey (Westinghouse 1988a) and Subtask 4.7: Transport Pathway Identification (Westinghouse 1989c). These pathways include ground water, surface water, soil, and air. Transportation and migration of compounds through these pathways are complexly interrelated. Complex pathways include:

- soil to air or surface water, or direct contact and
- ground water to surface water or vice versa.

A brief discussion of these four transport pathways, or migration pathways, follows.

5.1.1 Ground Water

Ground water is a potential contaminant migration pathway at the site. However, data collected during the Phase II RI indicate that the potential extent of off-site ground water impacts is limited. Since the site is located near a ground water divide, and recharging and through-flowing ground water likely discharges to Lawless Creek and the northern and southern drainages, the ground water that could potentially be affected by the site is limited to a narrow area downgradient from the site to Lawless Creek. Private wells known to be located in the vicinity of the FPRQ site are upgradient of the quarry. There are no known private wells downgradient of the site.

Contaminant migration via ground water occurs principally in the dissolved state. In general, contaminants found in the quarry and source areas are primarily metals, which have a very low solubility and are readily adsorbed onto

the soil matrix. Dilution and adsorption and transport via sediment are important factors affecting migration and fate of ground water contaminants. The concentrations of contaminants at off-site locations are also likely to be affected by sorption and biodegradation.

5.1.2 Surface Water

The surface water migration pathways at the site include the northern and southern drainages. Discharge from the north pond and from leachate seeps enters the northern drainage. Most of the quarry is internally drained and therefore does not contribute to runoff from the site. In response to precipitation events, runoff from about one-half of the site is discharged to both the northern and southern drainages. These drainages also receive some ground water discharge downgradient of the site. Both drainages enter Lawless Creek about 1,400 feet to the west of the site. Lawless Creek is a tributary of Fall Creek.

Surface water may carry dissolved and entrained material from the site. The concentration of soluble contaminants that may leave the site would be expected to diminish as they become diluted by surface water run-off from the remaining portions of the drainage basin. In addition, contaminant concentrations would be further limited by sorption, oxidation, volatilization, and biodegradation along the transport pathway.

The potential for surface water dilution, particularly as the north and south drainages flow into Lawless Creek, is significant. Estimates of surface water dilution at various portions of the surface drainage from the site have been discussed briefly in Section 9.0 of this risk assessment, as well as in further detail in Section 4.0 of the RI Report.

5.1.3 Soils

There is minimal potential for soil transport from the site. Soil could migrate from the site if carried off by individuals or on the tires of vehicles entering and leaving the site. However, access to the site is now limited, following installation of the chain link fence along the front of the site. The remainder of the site has been posted with no trespassing signs.

The potential does exist for erosion of sediment, containing adsorbed contaminants, from the site into surface water drainage in both the north and south drainages. Samples taken from both of these drainages during Rounds 1 and 2 sampling events of the RI Phase II indicated limited movement of site contaminants close to the quarry in these drainages, but no movement of contamination in sediments beyond the immediate outflow of the quarry drainage into the north and south drainages.

5.1.4 Air

Potential exposure via the air transport pathway is minimal. Contaminants can migrate from the site via the air route as chemicals volatilize or as contaminated soil particles are entrained in wind. The prevailing wind direction in the vicinity of the site is from the southwest (Ruffner and Blair, 1987 and County of Pittsylvania, 1986). Negligible amounts of contaminants carried by a southwest wind would move away from the community of Beaver Park to forested and agricultural land northeast of the site, where particulates would be deposited or otherwise filtered out by vegetation.

The contaminants identified at the FPRQ site have extremely low vapor pressures. The indicator chemicals consist of 11 inorganic chemicals and 1

organic compound, benzene. The Permissible Exposure Limit (PEL) for inhalation exposure to benzene is 10 ppm. Its vapor pressure at 20° C is 74.6 mm Hg. Benzene was found in the quarry leachate at a maximum concentration of 13 ppb. Regardless of temperature considerations, it is unrealistic to imagine a scenario where benzene concentrations would exceed the PEL with these representative concentrations. The other 11 indicator chemicals have vapor pressures that are nil at 20° C. The potential for vaporization is not dependent on realistic ambient temperatures at the site. Furthermore, the risk assessment has under reasonable worst case assumptions that potential fugitive dust emissions would not contain sufficient levels of adsorbed metals to pose a risk, even to individuals living a lifetime in the quarry, based on a residential future use scenario.

5.2 POTENTIAL EXPOSURE POINTS

Potential exposure points have been discussed in the Technical Memorandum for Subtask 2.2: Sensitive Receptor Survey (Westinghouse 1988a). Target populations may be exposed to contaminants by the migration pathways discussed in the Subtask 4.7 Technical Memorandum: Transport Pathway Identification (Westinghouse 1989c), and summarized briefly in Section 5.1 of this risk assessment. The potential level of exposure experienced by the receptor population will be a function of the concentration of potential contaminants at the exposure point and the duration of exposure. Specific potential exposure points hypothesized for the FPRQ site are discussed below.

Potential human exposure to contaminants could be by five exposure routes: direct exposure to source material or exposure to ground water, surface water,

soil, and air. Qualitative discussions of the potential for human exposure via each of these routes from the FPRQ site are presented below; quantitative exposure estimates are provided, as appropriate, in Section 6.0.

5.2.1 Source Areas

Presently, there is minimal exposure to source contamination. There are no ground water wells within the confines of the site. Much of the landfill material is covered with a cap. Human contact to exposed source areas is presently limited by the presence of a fence surrounding portions of the site. There are presently no known activities that occur at the site that would result in direct contact, with the exception of the ongoing RI/FS.

However, under the no-action alternative for the site, it is not possible to rule out future uses of the quarry. Although highly unlikely, it is appropriate under the no-action alternative to consider as a scenario the possibility of residential development of the site, with drinking water wells in the leachate and with direct contact of children and adults to contaminant sources in the quarry and vicinity, such as drums, seeps, quarry pond water, carbon black, the old disposal area, and the waste pile.

5.2.2 Ground Water

There is no exposure to contaminants in ground water from the FPRQ site at present, nor is there reason to believe that such exposure is likely to occur in the future since the most likely areas for future development in the vicinity of the site are not located in areas down gradient from the quarry. Known wells in the community of Beaver Park are upgradient from the site. Withdrawal of water from these wells is not likely to reverse gradients. Historical data presented

in the Task 3 Technical Memorandum: Evaluation of Existing Data (Westinghouse 1988b) indicate no landfill induced

impacts to the private wells in the Beaver Park community. Additional water samples analyzed by the U.S. Environmental Protection Agency from 10 local domestic water wells in Beaver Park provide further confirmation of the absence of human exposure via this route. In addition, hydrogeological investigations at the site also confirm the absence of contaminant migration from the quarry via the ground water pathway.

However, under the no action alternative for the site, it is necessary to assume that it is possible for residential development of down gradient areas, with consequent installation of ground water wells, to occur. Therefore, human exposure via ingestion of down gradient ground water is evaluated in this risk assessment.

5.2.3 Surface Water and Sediments

Potential exposure via the surface water pathway is limited at present. Migration of contaminants from the quarry via the surface water pathway extends primarily to the north and south drainage with little, if any, contamination of the wetland or of Lawless Creek. Surface water down gradient of the site is not used for potable water. Fish species observed in Lawless and Fall Creeks are not likely to be used as a food source by the local human population. Therefore, this exposure route is a limited one due to lack of exposure as well as a result of dilution, sorption, volatilization, and biodegradation of contaminants along this potential transport pathway.

However, under the no-action alternative, it is assumed that humans could be exposed to surface water through consumption of water from Lawless Creek and through wading in local creeks and drainages from the quarry.

5.2.4 Soil

Exposure to contaminated soil at the site by direct contact is likely to be minimal. Installation of a security fence has limited human access to the site and most of the waste material is covered.

Given a no-action alternative for the site, potential, or reasonable, worst case scenarios include the possibility that the property is used for residential development at some time in the future. In addition, it is impossible to completely rule out the potential exposure for children or adults who gain unauthorized access to the site. To that end, exposure scenarios are developed that consider the possibility that children ingest incidental amounts of soil during play in soil as well as scenarios that consider incidental ingestion of soil by adult gardeners.

5.2.5 Air

Human exposure to quarry contaminants via fugitive dust emissions will depend on the soil contaminant level, amount of contaminant adsorbed to the soil, the size of the dust particles, and how far into the respiratory tract these particles penetrate (which is a function of particle size). Inhalation of volatilized compounds is unlikely since levels of volatiles in samples obtained from the site are very low and are not found in surface soils. This exposure route is therefore considered to be minimal, particularly since there are only small areas with low concentrations of a few volatile materials at the site and surrounding vegetation and minimal activity make it unlikely that significant fugitive dust emissions would be generated.

However, under the no-action alternative, it is impossible to rule out the potential for humans to be exposed to particulate emissions if the site is developed in the future for uses such as residential development.

5.3 EXPOSURE SCENARIOS

This subsection of the risk assessment identifies exposure scenarios that have been developed for the FPRQ site for the no-action alternative. The scenarios considered include potential exposure to on-site source material and leachate in the quarry, ground water, surface water and sediments, soils in the quarry and in the vicinity of the quarry, and particulate air emissions. Specific assumptions and specific exposure estimates derived for these scenarios are provided in Section 6.0

5.3.1 Source Areas and Leachate

The no-action alternative assumes that the FPRQ quarry could be developed for residential uses at some time in the future. Given such development, there is the potential for wells to intersect the leachate in the quarry and for subsequent human ingestion of leachate, as well as for individual residents to come into direct contact with source area contaminants during play or gardening.

Four scenarios have been developed that serve to represent the maximum exposure potential for individuals, given the unlikely event of residential development within the quarry.

1. Direct human ingestion of leachate.
2. Direct contact with, and subsequent incidental ingestion of, source material by children during play.

3. Direct contact with, and subsequent incidental ingestion of, source material by residential gardeners.

4. Incidental ingestion of surface water from the north and south ponds by children while playing.

Other possible exposures, such as incidental ingestion of test pit soils within the quarry area have not been considered, since it is assumed that the evaluation of potential ingestion of leachate poses a reasonable worst case exposure scenario.

5.3.2 Ground Water

Although there is no current use of down gradient ground water, there is the potential for the future development of areas immediately down gradient of the FPRQ site for residential, use along with installation of ground water wells, under the no action alternative. This potential is evaluated.

1. Human ingestion of down gradient ground water

Other potential exposure scenarios related to ground water, such as dermal contact or inhalation of volatiles while showering, have not been considered since the direct ingestion scenario represents maximum exposure.

5.3.3 Surface Water and Sediment

Despite the existence of a site fence at the present time, there still is a potential for children to play in the north and south drainages, as well as in Lawless Creek. In addition, while it is unlikely for children to swim in these drainages due to their lack of depth and surface area, it is possible for

children to ingest incidental amounts of water and/or sediments while playing or wading.

There is little use, if any, of surface water for potable supplies in the vicinity of the site. Never-the-less, there does exist the possibility that people could use Lawless Creek downstream of the FPRQ site for potable water in the future.

The following five scenarios have been developed for exposure to surface water and sediments from the FPRQ site.

1. Incidental ingestion of water from the north drainage by children during play.
2. Incidental ingestion of water from the south drainage by children during play.
3. Incidental ingestion of sediments from the north drainage by children during play.
4. Incidental ingestion of sediments from the south drainage by children during play.
5. Use of Lawless Creek as a source of potable water by nearby residents.

Other potential exposure scenarios, such as incidental ingestion of water from Lawless Creek, ingestion of fish from Lawless Creek, ingestion of cattle grazed along Lawless Creek, or consumption of vegetables grown using irrigation water from either the drainages or from Lawless Creek have not been considered. The three scenarios outlined above are assumed to represent reasonable worst case exposure scenarios for surface water and sediments.

5.3.4 Soil

Under the no-action alternative, there is the potential for exposure to soils among children or adults who play or work at the site, either in the quarry itself, or in the soils along the drainages near the site. Work in the quarry or in nearby areas could result in exposure during construction of houses, or during gardening activities by adults. Six scenarios have been developed to assess the potential risk associated with soil exposures.

1. Incidental ingestion of quarry soil by children during play.
2. Incidental ingestion of quarry soil by adults as a result of residential gardening.
3. Incidental ingestion of soil in northern drainage by children during play.
4. Incidental ingestion of soil in northern drainage by adults as a result of residential gardening.
5. Incidental ingestion of soil in southern drainage by children during play.
6. Incidental ingestion of soil in southern drainage by adults as a result of residential gardening.

Other potential exposure scenarios for soil, such as dermal exposures, have not been considered, since the contaminants of concern are metals and are not likely to be dermally absorbed and since the soils in the vicinity of the upper northern and southern drainages are expected to have the highest concentrations of contaminants of any soils outside of the quarry. Similar exposures to source areas have already been considered (Section 5.3.1).

5.3.5 Air

Under the no-action alternative, a scenario has been developed to estimate the potential lifetime exposure for humans exposed to particulate emissions if the site is developed in the future for uses such as residential development.

1. Lifetime exposure to inhalation of particulate matter

Additional air pathway scenarios, such as exposure of individuals in the community of Beaver Park to particulate material from the site, were not considered since the scenario that is developed represents the reasonable worst case events for the site.

SECTION 6

EXPOSURE ASSESSMENT

This subsection of the risk assessment provides assumptions and exposure estimates derived for the specific exposure scenarios identified in Subsection 5.3 above.

Development of these exposure scenarios are based on a series of general assumptions as well as on specific assumptions for the different scenarios. As appropriate, these assumptions are derived from guidance provided in U.S. Environmental Protection Agency documentation (1986, 1989a, 1989b and 1989c) and include such assumptions as:

- adults weigh 70 kg, adolescents weigh 36 kg, and children weigh 20 kg;
- lifetime drinking water exposures are based on individuals living an entire lifetime of 70 years, with 10 years childhood exposure, 5 years adolescent exposure, and 55 years adult exposure;
- lifetime exposures are based on individuals living an entire lifetime of 70 years at the site;
- for average concentrations of any given indicator chemical, below detectable results were assumed to be one half of the reported detection limits if at least one sample in the scenario had detectable concentrations of that indicator chemical;
- if all samples in the scenario had below detectable concentrations of any given indicator chemical, that chemical was assumed not to be present in the sample;
- base-level, or "average" case exposure calculations are based on arithmetic mean concentrations of indicator chemicals, while "reasonable worst" case exposure calculations are based on the lesser of the upper 95 percent confidence interval of the mean or the maximum observed value;
- bioavailability of metals in ingested soil and sediments is assumed to be 50 percent for the "average" case calculations;

- o - bioavailability of metals in ingested soil and sediments is assumed to be 100 percent for the "reasonable worst" case calculations; and

- o - the health criteria for comparison of exposures, which were obtained either from U.S. Environmental Protection Agency (1986 and 1990a and 1990b) or from IRIS (1989), are as follows:

Indicator Chemical	ORAL EXPOSURES (mg/kg/day)		INHALATION EXPOSURES $(mg/kg/day)^{-1}$	
	Reference Dose	Cancer Potency Factor	Reference Dose	Cancer Potency Factor
Antimony	4e-4	NA	4e-4	NA
Barium	5e-2	NA	1.4e-4	NA
Cadmium	5e-4	NA	5e-4	6.1e0
Copper	3.7e-2	NA	1e-2	NA
Lead	1.4e-3	NA	4.3e-4	NA
Manganese	5e-1	NA	5e-1	NA
Mercury	2e-3	NA	5.1e-5	NA
Nickel	2e-2	NA	2e-2	1.19e0
Selenium	3e-3	NA	1e-3	NA
Vanadium	9e-3	NA	9e-3	NA
Zinc	2e-1	NA	1e-2	NA
Arsenic	NA	1.75e	NA	5e+1
Benzene	NA	2.9e-2	NA	2.9e-2
Bis (2eh) phthalate	2e-2	1.4e-2	2e-2	NA

Reference Doses are provided for non-carcinogenic effects, cancer potency factors are provided for carcinogens.

These assumptions lend a degree of conservatism to the exposure estimates. For instance, it is unlikely that residents the FPRQ site would be developed for residential use, unlikely that individuals would live there for 70 years, and unlikely that such individuals would be exposed to the maximum concentrations of contaminants in any particular environmental media.

Actual exposure estimates for each of the exposure scenarios are detailed in Tables 6.1 through 6.17. Specific assumptions and calculation methods for

each of these exposure scenarios are provided with the appropriate table. Sections 4, 7, and Appendices B,C, and D provide additional information on toxicology and health-based criteria and standards for the fourteen indicator chemicals for the FPRQ site.

For each of Tables 6.1 through 6.17, the first page provides the appropriate health criterion (from Section 4 and the list of assumptions for Section 6), average (arithmetic mean) and maximum (lesser of the upper 95 percent confidence interval of the mean or the maximum observed value) concentrations, average and reasonable worst case exposure dose estimates, specific assumptions, and appropriate hazard indices and lifetime cancer risks. The second page of each table specifies the formulae used for calculation of exposure dose, provides a diagrammatic map indicating the locations from which the samples for the specific scenario were taken, and lists the specific samples used as they are identified in the RI Report.

6.1 SOURCE AREAS AND LEACHATE IN QUARRY

Exposure scenarios related to source areas and leachate within the quarry are based on the assumption that the FPRQ site, at some time in the future, is developed for residential purposes with no removal of waste material. These scenarios assume that waste material is not covered or otherwise sequestered from exposure to the public and that domestic potable water is obtained from the site, with a suite of constituents equivalent to aqueous samples taken from a test pit (FP-702B) and quarry seep (FP-404). The assumption of future residential use of the site is made in this suite of scenarios, in accord with the U.S. Environmental Protection Agency (1986) guidelines as a "most conservative choice"

since current land use in the vicinity of the site is residential, with use of ground water for drinking.

6.1.1 Human Ingestion of Leachate

This scenario is based on a lifetime consumption of leachate from the quarry site. An average case assumes ingestion by an adult of two liters of water (leachate) daily. As a reasonable worst case, it is assumed that a child (sensitive receptor) drinks one liter of water (leachate) daily. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria, such as acceptable intakes based on U.S. Environmental Protection Agency reference dose values (RfD) or Health Effects Assessment values (HEA). For carcinogens, exposure estimates assume a lifetime exposure, with 10 years of childhood exposure, 5 years of adolescent exposure, and 55 years of adult exposure averaged over 70 years. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for human consumption of leachate from on-site sources are provided in Table 6-1. The ambient concentrations of indicator chemicals were derived from analyses of the following samples:

Test Pit 702B - Leachate (Aqueous sample)

Sample 404 - Quarry Seep

6.1.2 Incidental Ingestion of Source Material by Children

This scenario is based on a lifetime consumption of source material from the quarry site. An average case assumes ingestion by a child of 200 mg of soil

during play per day (U.S. Environmental Protection Agency, 1989a), with the child playing two days per week. As a reasonable worst case, it is assumed that a child with pica (tendency to eat soil) ingests 400 mg of source material during play per day (U.S. Environmental Protection Agency, 1989a), four days per week. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a 70-year lifetime exposure, with 15 years of childhood (ingestion of 200 or 400 mg of soil daily, 20 kg body weight) and 10 to 30 years of adult exposure (60 to 100 mg per day, 70 kg weight), averaged over 70 years, with play or other contact (gardening) in the source material occurring two to four days per week, 26 to 40 weeks per year when weather conditions are most favorable for such outdoor activity. These assumptions, therefore, incorporate adult exposures as part of the total lifetime exposure.

Specific assumptions and exposure estimates for incidental human ingestion of source material from on-site sources are provided in Table 6-2. The ambient concentrations of indicator chemicals were derived from analyses of the following samples:

Samples 410 through 412 - Phase I Source Area Samples (Waste Pile, Black Drum, Grey Drum)

Samples 110 through 113 - Phase II - Waste Pile

Samples 117 through 120 - Phase II - Carbon Black

6.1.3 Incidental Ingestion of Source Material by Adults

This scenario is based on consumption of source material from the quarry site during gardening activities by adults. An average case assumes ingestion of 60 mg of soil by an adult during gardening activity (U.S. Environmental

Protection Agency, 1989a), with the individual gardening two days per week during the six months of spring, summer, and fall. As a reasonable worst case, it is assumed that an individual gardens four days per week during a 40-week season. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a 10 to 30 years of exposure. Lifetime exposure calculations incorporate 15 years of childhood exposure.

Specific assumptions and exposure estimates for human consumption of source material from on-site sources as a result of gardening are provided in Table 6-3. The ambient concentrations of indicator chemicals were derived from analyses of the following samples:

Samples 410 through 412 - Phase I Source Area Samples (Waste Pile, Black Drum, Grey Drum)

Samples 110 through 113 - Phase II - Waste Pile

Samples 117 through 120 - Phase II - Carbon Black

6.1.4 Incidental Ingestion of Water from North and South Ponds by Children

This scenario is based on incidental consumption of surface water from the quarry site (north and south ponds) by children during wading. The U.S. Environmental Protection Agency (1989a) suggests an incidental ingestion rate of 50 ml water per hour during swimming. It is assumed here that children playing and wading ingest less incidental water than individuals would during swimming activity. Therefore, an average case assumes incidental ingestion, by a child, of 25 ml water per play day, with two days of play per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child plays two hours per play day and ingests 50 ml of water per day, with four days of play per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate

chronic exposure health-based criteria. For carcinogens, exposure estimates assume only 15 years of childhood exposure averaged over 70 years. This scenario assumes that adults will not be exposed to the surface water in the quarry. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for incidental human consumption of water from on-site sources are provided in Table 6-4. The ambient concentrations of indicator chemicals were derived from the following sample analyses:

Sample 401 - South Pond

Sample 402 and 403 - North Pond

6.2 GROUND WATER

The exposure scenario related to ground water. as with the previous scenarios discussed above, assumes that the area of the FPRQ site, at some time in the future, is developed for residential purposes. This scenario assumes that residents will use local ground water for domestic use. One scenario has been derived.

6.2.1 Human Ingestion of Downgradient Ground Water

This scenario is based on a lifetime consumption of ground water in the vicinity of the FPRQ site by residents. An average case assumes ingestion by a 70 kg adult of two liters of water daily. As a reasonable worst case, it is assumed that a 20 kg child (sensitive receptor) drinks one liter of water daily. For non-carcinogens, exposure is compared to appropriate chronic exposure health-

based criteria. For carcinogens, exposure estimates assume a lifetime exposure, with 10 years of childhood exposure, 5 years of adolescent exposure, and 55 years of adult exposure, averaged over 70 years. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for human consumption of ground water are provided in Table 6-5. The ambient concentrations of indicator chemicals were derived from all of the non-background, non-QA samples of ground water from the Phase II Round 1 ground water sampling activity, dissolved analyses only, including the following samples.

FP-003A
FP-003B
FP-004
FP-005A
FP-005B
FP-006A
FP-006B
FP-007A
FP-007B
FP-008A
FP-008B
FP-009

6.3 SURFACE WATER AND SEDIMENT

Given a no-action alternative for the FPRQ site, there is the potential of exposure of residents in the vicinity of the site to contaminants that may exist in either the northern and southern drainages or in Lawless Creek. Scenarios related to this type of exposure, while less representative of worst case conditions than are those scenarios developed for direct exposure to source material in the quarry, are important to an understanding of the residual risk associated with remedial actions that would essentially isolate the present waste material at the FPRQ site. The following five scenarios were developed.

6.3.1 Incidental Ingestion of Water from the Northern Drainage by Children

This scenario is based on incidental consumption of surface water from the northern drainage by children during wading or other play activity. The U.S. Environmental Protection Agency (1989a) suggests an incidental ingestion rate of 50 ml water per hour during swimming. It is assumed here that children playing and wading ingest less incidental water than individuals would during swimming activity. Therefore, an average case assumes incidental ingestion, by a child, of 25 ml water per play day, with two days of play per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child ingests 50 ml of water per play day, with four days of play per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume 15 years of childhood exposure averaged over 70 years. It is assumed that adults will have no chronic exposure to surface water in the northern drainage. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for incidental human consumption of water from the northern drainage are provided in Table 6-6. The ambient concentrations of indicator chemicals were derived from analyses of the following samples.

FP-309 (Rounds 1 and 2)
FP-315 (Round 2)
FP-310 (Round 2)
FP 311 (Rounds 1 and 2)

6.3.2 Incidental Ingestion of Water from the Southern Drainage by Children

This scenario is based on incidental consumption of surface water from the southern drainage by children during wading or other play activity. The U.S. Environmental Protection Agency (1989a) suggests an incidental ingestion rate of

50 ml water per hour during swimming. It is assumed here that children playing and wading ingest less incidental water than individuals would during swimming activity. Therefore, an average case assumes incidental ingestion, by a child, of 25 ml water per play day, with two days of play per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child ingests 50 ml of water per play day, with four days of play per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume 15 years of childhood exposure averaged over 70 years. It is assumed that adults will have no chronic exposure to surface water in the southern drainage. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for incidental human consumption of water from the southern drainage are provided in Table 6-7. The ambient concentrations of indicator chemicals were derived from analyses of the following samples.

FP-306 (Rounds 1 and 2)
FP-307 (Round 2)
FP-308 (Rounds 1 and 2)
FP 319 (Round 1)

6.3.3 Incidental Ingestion of Northern Drainage Sediments by Children

This scenario is based on incidental consumption of sediments from the northern drainage by children during play. An average case assumes ingestion by a child of 200 mg of sediment during play (U.S. Environmental Protection Agency, 1989a), with the child two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child with pica ingests 400 mg of sediment during each play day (U.S. Environmental Protection Agency, 1990), with the child

playing four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 15 years of exposure, averaged over 70 years. It is assumed that adults would not be exposed to sediments in this drainage to any appreciable degree.

Specific assumptions and exposure estimates for incidental human ingestion of sediments from the northern drainage is are provided in Table 6-8. The ambient concentrations of indicator chemicals were derived from analyses of the following samples.

FP-209 (Rounds 1 and 2)
FP-215 (Round 2)
FP-210 (Round 2)
FP-211 (Rounds 1 and 2)

6.3.4 Incidental Ingestion of Southern Drainage Sediments by Children

This scenario is based on incidental consumption of sediments from the southern drainage by children during play. An average case assumes ingestion by a child of 200 mg of sediment during play (U.S. Environmental Protection Agency, 1989a), with the child playing two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child with pica ingests 400 mg of sediment during each play day (U.S. Environmental Protection Agency, 1990) with the child playing four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 15 years of exposure, averaged over 70 years. It is assumed that adults would not be exposed to sediments in this drainage to any appreciable degree.

Specific assumptions and exposure estimates for incidental human ingestion of sediments from the southern drainage is are provided in Table 6-9. The

ambient concentrations of indicator chemicals were derived from analyses of the following samples.

FP-206 (Rounds 1 and 2)
FP-207 (Round 2)
FP-208 (Rounds 1 and 2)
FP-219 (Round 1)

6.3.5 Use of Lawless Creek as a Potable Water Supply

This scenario is based on a lifetime consumption of water from Lawless Creek and assumes that at some time in the future, there is residential development of the area immediately downstream of the site along Lawless Creek and that residents use the creek water for potable water.

It is recognized that an associated scenario would be the ingestion of fish from Lawless Creek or the consumption of cattle grazed along the creek. However, since the contaminants identified at the site do not bioaccumulate to any appreciable extent, direct ingestion of contaminants in drinking water is considered the most conservative exposure scenario for exposure to contaminants in Lawless Creek.

An average case assumes ingestion by an adult of two liters of water daily. As a reasonable worst case, it is assumed that a child (sensitive receptor) drinks one liter of water daily. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a lifetime exposure, with 15 years of childhood exposure (1 liter per day, 20 kg), 5 years of adolescent exposure (2 liters per day, 36 kg), and 55 years of adult exposure (2 liters per day, 70 kg) averaged over 70 years. Carcinogenic risk is a function of exposure times cancer potency.

Specific assumptions and exposure estimates for human consumption of water from Lawless Creek are provided in Table 6-10. The ambient concentrations of indicator chemicals were derived from analyses of the following samples.

FP-313 (Rounds 1 and 2)
FP-314 (Rounds 1 and 2)

6.4 SOIL

Given a no-action alternative for the FPRQ site, there is the potential for exposure of residents in the vicinity of the site to contaminants that may exist in soil in the quarry or in the vicinity of the quarry. The worst case conservative assumption of exposure to source material in the quarry itself has been discussed in Subsection 6.1. In this section, potential exposures to quarry soils and to soils near the quarry (in the northern and southern drainage areas adjacent to the quarry) are considered. Scenarios related to this type of exposure, while less representative of worst case conditions than are those scenarios developed for direct exposure to site source contamination, are important to an understanding of the residual risk associated with remedial actions that would essentially isolate the present contaminated soils at the FPRQ site.

6.4.1 Incidental Ingestion of Quarry Soil by Children

This scenario is based on incidental ingestion of soil in the quarry by children during play. An average case assumes ingestion by a child of 200 mg of soil during play (U.S. Environmental Protection Agency, 1989a), with the child playing in the quarry two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child with pica ingests 400 mg of soil during each play day (U.S. Environmental Protection Agency, 1990), with the child

playing four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 15 years of exposure, averaged over 70 years. In addition, lifetime exposure to carcinogens assumes that the exposed child grows up and gardens in soils on the site, ingesting an additional 60 to 100 mg of soil per gardening day as an adult.

Specific assumptions and exposure estimates for human consumption of soil from on-site sources are provided in Table 6-11. The ambient concentrations of indicator chemicals were derived from analyses of the following surface soil samples.

FP-103
FP-104
FP-105
FP-106
FP-107
FP-108
FP-109

6.4.2 Incidental Ingestion of Quarry Soil by Adults

This scenario is based on a lifetime consumption of soil from the quarry site during gardening activities by adults. An average case assumes ingestion by an adult of 60 mg of soil during gardening activity (U.S. Environmental Protection Agency, 1989a), with the individual gardening two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that an individual ingests 100 mg of soil per gardening day and gardens four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a lifetime exposure. Lifetime exposure calculations incorporate 15 years of childhood exposure.

Specific assumptions and exposure estimates for human consumption of source material from on-site sources as a result of gardening are provided in Table 6-12. The ambient concentrations of indicator chemicals were derived from analyses of the following surface soil samples.

FP-103
FP-104
FP-105
FP-106
FP-107
FP-108
FP-109

6.4.3 Incidental Ingestion of Northern Drainage Soil by Children During Play

This scenario is based on incidental ingestion of soil from the northern drainage adjacent to the quarry by children during play. An average case assumes ingestion by a child of 200 mg of soil during play (U.S. Environmental Protection Agency, 1989a), with the child playing two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child with pica ingests 400 mg of soil during each play day (U.S. Environmental Protection Agency, 1990) four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 15 years of exposure, averaged over 70 years. In addition, lifetime exposure to carcinogens assumes that the exposed child grows up and gardens in soils on the site, ingesting an additional 100 mg of soil per gardening day as an adult.

Specific assumptions and exposure estimates for human consumption of soil from the northern drainage are provided in Table 6-13. The ambient concentrations of indicator chemicals were derived from the following surface soil samples taken from the area of the northern drainage.

FP-121 Grid
FP-122 Grid
FP-123 Grid
FP-124 Grid

6.4.4 Incidental Ingestion of Northern Drainage Soil by Adults

This scenario is based on a lifetime ingestion of soil from the northern drainage adjacent to the quarry during gardening activities by adults. An average case assumes ingestion by an adult of 60 mg of soil per day during gardening activity (U.S. Environmental Protection Agency, 1989a), with the individual gardening two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that an individual ingests 100 mg of soil and gardens four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a lifetime exposure. Lifetime exposure calculations incorporate 15 years of childhood exposure.

Specific assumptions and exposure estimates for human consumption of soil from the northern drainage as a result of gardening are provided in Table 6-14. The ambient concentrations of indicator chemicals were derived from the following surface soil samples taken from the area of the northern drainage.

FP-121 Grid
FP-122 Grid
FP-123 Grid
FP-124 Grid

6.4.5 Incidental Ingestion of Southern Drainage Soil (Disturbed Area) by Children During Play

This scenario is based on incidental ingestion of soil from the southern drainage adjacent to the quarry by children during play. An average case assumes ingestion by a child of 200 mg of soil during each play day (U.S. Environmental

Protection Agency, 1989a), with the child playing two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that a child with pica ingests 400 mg of soil during each play day (U.S. Environmental Protection Agency, 1990), with the child playing four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 15 years of exposure, averaged over 70 years. In addition, lifetime exposure to carcinogens assumes that the exposed child grows up and gardens in soils on the site, ingesting an additional 60 to 100 mg of soil per gardening day as an adult.

Specific assumptions and exposure estimates for human consumption of soil from the southern drainage are provided in Table 6-15. The ambient concentrations of indicator chemicals were derived from the following surface soil samples taken from the area of the southern drainage.

FP-114
FP-115
FP-116
FP-125

6.4.6 Incidental Ingestion of Southern Drainage Soil (Disturbed Area) by Adults

This scenario is based on a lifetime ingestion of soil from the southern drainage adjacent to the quarry during gardening activities by adults. An average case assumes ingestion by an adult of 60 mg of soil during gardening activity (U.S. Environmental Protection Agency, 1989a), with the individual gardening two days per week for 26 weeks per year. As a reasonable worst case, it is assumed that an individual ingests 100 mg of soil and gardens four days per week for 40 weeks per year. For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure

estimates assume a lifetime exposure. Lifetime exposure calculations incorporate 15 years of childhood exposure.

Specific assumptions and exposure estimates for human consumption of soil from the southern drainage as a result of gardening are provided in Table 6-16. The ambient concentrations of indicator chemicals were derived from the following surface soil samples taken from the area of the southern drainage.

FP-114
FP-115
FP-116
FP-125

6.5 AIR

Given a no-action alternative for the FPRQ site, there is the potential for exposure of residents in the vicinity of the site to inhalation of airborne particulate material that might contain entrained or adsorbed material from the soil at or in the vicinity of the FPRQ site. The worst case conservative assumption of exposure to source material in the quarry by ingestion has been

discussed in Subsection 6.1. In this section, potential exposure to contaminants adsorbed onto fugitive dust emissions from quarry soil is considered. This scenario assesses the lifetime inhalation exposure of humans to quarry soil, assuming future residential development of the FPRQ site along with development of a roadway in the quarry. As a conservative assumption, it is assumed that this roadway is dirt and no dust suppression measures are employed.

6.5.1 Inhalation of Particulate Matter

This scenario assumes inhalation of particulate fugitive emissions from surface soil in the quarry by humans. The scenario assumes generation of dust on an unpaved roadway through the quarry by traffic. The average case assumes adult exposure, while the reasonable worst case includes exposure assumptions related to children for chronic exposure estimates.

For non-carcinogens, exposure is compared to appropriate chronic exposure health-based criteria. For carcinogens, exposure estimates assume a total of 70 years of exposure.

Specific assumptions and exposure estimates for human inhalation of soil from on-site sources are provided in Table 6-17. The ambient concentrations of indicator chemicals were derived from analyses of the following surface soil samples.

FP-103
FP-104
FP-105
FP-106
FP-107
FP-108
FP-109

Derivation of exposure estimates for potential inhalation exposure to site contaminants from the FPRQ site involves several steps, including estimation of generation rates for particulate fugitive emissions, estimation of subsequent ambient air concentrations of individual contaminants given soil concentrations and ambient climatic data, and estimates of exposure to each indicator chemical.

Potential emissions of fugitive dust from the site are estimated based on guidance from U.S.EPA (1988c). For dust generation from unpaved roads, the following equation is used:

$$E=k(1.7)x(s/12)x(S/48)x(W/2.7)^{0.7x}(w/4)^{0.5x}((365-P)/365)kg/VKT$$

where

E = emission factor in kg dust per VKT (vehicle kilometers traveled);

k = non-dimensional particle size multiplier;

s = percent soil silt content;

S = mean vehicle speed in km per hour;

W = mean vehicle weight in metric tons;

w = mean number of wheels per vehicle; and

P = number of days with at least 0.254 mm (0.01 inch) of precipitation per year.

Estimates of fugitive dust emissions were calculated for the FPRQ site under both average and reasonable worst case assumptions based on the following input assumptions:

Input Parameter	Average Case	Worst Case	Reasonable Comment
k	0.36	0.36	particles ≥ 10um (U.S. Environmental Protection Agency 1988c)
s	15 %	20 %	residential dust suppression
S	30 km/hr	50 km/hr	slow traffic, residential area
W	3 mtons	15 mtons	primarily light trucks and cars
w	4 wheels	6 wheels	primarily light trucks and cars
P	120 days	120 days	(U.S. Environmental Protection Agency 1988c)

Dust generation estimates of 0.345 kg per VKT (average case) and 2.9 kg per VKT (reasonable worst case) were calculated given the above assumptions. Further, assuming:

- an average of 25 daily round trips along a 100 meter unpaved roadway in the quarry for an average case and 100 daily round trips along a 100 meter unpaved roadway for a reasonable worst case and
- a 16-hour day for activity,

average fugitive dust generation rates of 3.004×10^{-9} grams per second under an average case scenario and 1.007×10^{-6} grams per second under the worst case scenario are calculated based on the following equation:

$$\text{Dust Generation(g/sec)} = \frac{\text{Emission Rate(g/VKT)} \times \text{daily travel distance(km)}}{16\text{(hours/day)} \times 3600\text{(seconds/hour)}}$$

The above values for dust generation were then used to generate annual ambient contaminant concentrations in the vicinity of the FPRQ site on the basis of site air quality dispersion modeling using the U.S. Environmental Protection Agency Industrial Source Complex Short Term (ISCST) model. A screening level ambient air quality dispersion model was run using one year (1986) of meteorological data from the Raleigh-Durham International Airport in North Carolina. This analysis was based on a hypothetical unpaved road running for approximately 100 meters in an east-west direction in the approximate mid-point of the quarry on-site soil sampling grids. The roadway was represented by twenty volume sources, each with a 1 gram per second emission rate. The procedures outlined in the ISCST manual were followed to represent a line source as multiple volume sources. Meteorological defaults in the ISCST model were used to determine worst case downwind concentrations at receptor locations placed 10 meters from the line source at 20-meter intervals along the line.

The output from the ISCST model, based on a 1 gram per second unit emission (generation rate) was corrected by the appropriate dust generation rate, as calculated above, to derive a site-specific ambient fugitive dust concentration. The annual average ISCST-modeled fugitive dust concentration at the receptor of maximum concentration (9.124 mg per cubic meter) was used to derive an average case ambient concentration, while the maximum 24-hour concentration (44.187 mg per cubic meter) was used for the reasonable worst case scenario. Since the 100 meter modeled line source was represented by twenty volume sources, each generating 1 gram per second, and the site specific fugitive emission rates calculated above are a unit emissions based on travel along a 100 meter roadway, site specific ambient concentrations were derived as follows:

$$\text{Ambient Conc}(ug/M^3) = \frac{\text{Dust Generation}(g/sec) \times \text{ISCST}(mg/M^3) \times 1000(ug/mg)}{20(g/sec)}$$

to obtain the following values.

	Dust Generation (g/sec)	ISCST (mg/M^3)	Ambient Concentration (ug/M^3)
Average Case	3.004×10^{-9}	9.124	1.37×10^{-6}
Worst Case	1.007×10^{-6}	44.187	2.22×10^{-3}

The ambient dust concentrations calculated and presented in the above table were then multiplied by contaminant-specific soil concentrations in Table 6-17 to derive specific exposure estimates for each indicator chemical at the FPRQ site. Additional specific assumptions and exposure estimates for human inhalation of soil from on-site sources as a result of residential use of the FPRQ site are provided in Table 6-17.

SECTION 7

HUMAN HEALTH BASED STANDARDS AND CRITERIA

This section describes the procedures for identifying chemical-specific and location-specific Applicable or Relevant and Appropriate Requirements (ARARs) for the FPRQ and presents the human health-based standards and criteria against which exposure estimates are compared. This section also discusses issues of interpretation and analysis involving the various potential requirements and provides guidance as to when certain requirements will be ARARs. In general, removal actions will require attaining ARARs to the greatest extent practicable. However, ARARs are not necessarily to be used to assess the health risks. Comparison of FPRQ data to ARARs can be used to assess the attainment or non-attainment of institutional requirements. The procedures utilized in the identification of these ARARs were obtained from the draft guidance manual on CERCLA compliance with other laws (U.S. Environmental Protection Agency, 1988b).

7.1 IDENTIFICATION AND ANALYSIS OF CHEMICAL-SPECIFIC ARARS

Tables presenting the chemicals identified on site and specific media-based ARARs are provided in Appendix D. Chemical-specific ARARs are usually health- or risk-based numerical values. They serve to establish the acceptable concentration of a chemical that may be found in, or be discharged to, the ambient environment. Where a chemical has more than one ARAR, then compliance should be with the most stringent (U.S. Environmental Protection Agency, 1988b).

7.1.1 Determination of Applicability

Applicable requirements are those standards, environmental protection requirements, or other federal and state limitations that specifically address a chemical, location, remedial action, or other circumstance at a CERCLA site. RCRA requirements for the treatment, storage, or disposal of hazardous waste would apply to FPRQ if FPRQ contains RCRA listed or characteristic hazardous wastes that were treated or disposed of after the effective date of the RCRA regulations under consideration as ARARs. For example, RCRA Maximum Contaminant Limits (MCLs) are applicable requirements for ground water protection at RCRA regulated units. These units must have received RCRA hazardous waste after July 6, 1982. This is not the case at FPRQ. If it cannot be determined that a substance falls under the RCRA regulations, then the RCRA regulations are not applicable; however, they may be relevant and appropriate. For a standard or other federal or state limitation to be applicable, it must satisfy all of the jurisdictional prerequisites of a requirement.

Safe Drinking Water Act Maximum Contaminant Levels (SDWA MCLs) are enforceable standards for public drinking water systems. The SDWA MCLs apply to community water systems, which are public water systems having at least 15 service connections or serving an average of at least 25 year-round residents. In the case of FPRQ, the individual wells in the local community are upgradient from the site and are not public sources of drinking water. Therefore the SDWA standards are not applicable here.

Based on an analysis of all of the pertinent regulations, standards and other federal and state limitations, none are applicable as chemical-specific ARARs at the FPRQ site.

7.1.2 Determination of Relevance and Appropriateness

The U.S. Environmental Protection Agency has determined that MCLs will be relevant and appropriate for ground water or surface water that currently is or may in the future be used directly for drinking. Therefore, the MCLs must be met in the surface water or ground water itself. While the wells near this site are presently upgradient of the site, there is the potential that ground water supply wells could be installed downgradient in the future. Therefore, the MCls would be relevant and appropriate for the ground water.

Surface water runoff from the site could potentially be used as a drinking supply; therefore, the MCLs could be considered relevant and appropriate requirements for the surface water. While the Primary SDWA standards are considered enforceable, the secondary standards are not. However, the secondary standards are still considered relevant and appropriate, as their goals are sufficiently similar to the goals of cleanup of the FPRQ site.

The MCL Goals (MCLGs) are non-enforceable health goals that should be considered in special cases, such as at FPRQ where there are multiple contaminants. Therefore, the MCLGs are relevant and appropriate requirements for the surface water.

The Federal Water Quality Criteria are non-enforceable guidelines used by the states to set surface water quality standards. These federal standards are considered relevant and appropriate requirements by the U.S. Environmental Protection Agency for the FPRQ surface water.

The Virginia Water Quality Standards for Surface Water and for Aquatic Life would also be considered relevant and appropriate requirements for the surface

water at the site. The specific goals and objectives for the site are to protect existing and attainable use or uses of the receiving waters.

7.2 COMPARISON OF MOST STRINGENT ARARS WITH CONTAMINANT CONCENTRATIONS

The fourteen indicator chemicals identified at FPRQ are listed in Section 3.0 of this risk assessment. These fourteen chemicals' most stringent ARARs are compared in this subsection with existing concentrations, both average and maximum, in ground water and surface water.

7.2.1 Ground Water and Surface Water ARARs

The most stringent ARARs for the fourteen indicator chemicals for ground water and surface water are indicated below. In addition, for cadmium, copper, lead, and nickel, ARARS are also calculated based on hardness in accord with the formulae noted on Table 3 of Appendix D. The calculations assume an average hardness of 39.92 mg per liter.

Indicator Chemical	ARAR (ug/l)		
	Ground Water	Surface Water	Surface Water Hardness-Based
Antimony	--	150	--
Arsenic	50	0.0022	--
Barium	1000	1000	--
Benzene	5	0.66	--
Bis (2-EH) phthalate	--	3	--
Cadmium	0.4	1.1	550
Copper	1000	12	5390
Lead	50	3.2	1010
Manganese	50	50	--
Mercury	0.05	0.012	--
Nickel	--	134	47560
Selenium	10	10	--
Vanadium	--	--	--
Zinc	50	47	--

These ARARs consist of the most stringent values from the Federal Safe Drinking Water Act/RCRA values, Clean Water Act's Water Quality Criteria, and Virginia Water Quality Standards.

7.2.2 Maximum and Average Indicator Chemical Ground Water and Surface Water Concentrations

The maximum and average concentrations of the fourteen indicator chemicals in the surface water and ground water are provided below. The average concentrations are the arithmetic means of sample concentrations. The maximum concentrations are the lesser of the upper 95 percent confidence interval or the maximum observed value.

Indicator Chemical	Ground Water (Concentrations in ug/l)		Surface Water (Concentrations in ug/l)	
	Average	Maximum	Average	Maximum
Antimony	1.117	1.317	1.717	2.59
Arsenic	BDL	BDL	17.16	30.56
Barium	120.9	139.4	1646	2786
Benzene	BDL	BDL	BDL	BDL
Bis (2-EH) phthalate	7.5	9.306	BDL	BDL
Cadmium	BDL	BDL	23.22	41.11
Copper	4.867	6.73	14.21	19.85
Lead	BDL	BDL	3.932	6.36
Manganese	595.6	1245	901.7	1312
Mercury	BDL	BDL	BDL	BDL
Nickel	BDL	BDL	20.07	25.11
Selenium	BDL	BDL	BDL	BDL
Vanadium	BDL	BDL	BDL	BDL
Zinc	140.0	223.3	15546	29619

7.3 HUMAN HEALTH BASED CRITERIA

Human health based standards and criteria, as established by the U.S. Environmental Protection Agency, where available, are presented for the fourteen indicator chemicals in Appendix D. This appendix includes such standards as reference doses, health advisories, and cancer potencies where relevant.

7.4 CONCLUSION

The identification of "applicable or relevant and appropriate requirements" allows a comparison to exposure point concentrations for the chemicals identified at the First Piedmont site. This comparison serves as the basis for the baseline public health evaluation.

Generally, the policy of the U.S. Environmental Protection Agency is to attain contaminant level ARARs to ensure protection at all points of potential exposure. At each potential point of exposure, a reasonable maximum exposure scenario must be examined and the cleanup goals set accordingly. Where cleanup goals are not practicable or cost effective, exposure controls may be utilized in combination with treatment/engineered controls.

SECTION 8

RISK SUMMARY

This section provides a brief discussion of the nature of risk, a discussion of conservatism in the risk assessment process as it applies to the FPRQ site risk assessment, and a summary of risk for the FPRQ site based on the exposure scenarios and calculations provided in Section 6 and Tables 6.1 through 6.17.

8.1 NATURE OF RISK ASSESSMENT

The risk assessment process involves evaluation of a series of hypothetical assumptions to derive some estimate of the potential adverse effects on human health, given the assumptions made. These assumptions are conservative in nature in order to provide a margin of safety. The conservative nature of the specific assumptions made in this risk assessment for the FPRQ site are discussed in the next subsection.

For risk to exist, there must also be exposure to toxic or otherwise harmful conditions. Without such exposure, there is no risk. Currently, there is no exposure to the FPRQ site by residents, therefore, the risks estimated in this risk assessment are potential risks, based on the assumption that the site is developed or otherwise used in the future such that there will be some degree of exposure.

In terms of carcinogenic risk, the risks estimated in Tables 6-1 through 6-17 provide an estimate of the potential incremental risk, posed by the site conditions under the various scenario assumptions, of an individual contracting

cancer. A risk of 1×10^{-6} represents an incremental increase in risk of cancer occurrence of one in one million. Given that the general population has a background risk of approximately 300,000 cancers in one million, an incremental risk of 1×10^{-6} means that instead of 300,000 cancers, there will be 300,001 cancers.

8.2 CONSERVATIVE FACTORS IN THE FPRQ RISK ASSESSMENT

The risk assessment for the FPRQ site is based on chain of conservative assumptions that, taken together, provide a significant margin of safety in estimating potential risk from the site. Examples of such conservatism include the following.

- Exposure scenarios assume that the site will be developed for residential purposes at some time in the future.
- Exposure scenarios assume that children play, or adults garden, in source areas or in known contaminated areas of the site.
- Exposure scenarios assume that children play, or adults garden, daily or every other day for nine months of the year, regardless of severe weather or other potential activities.
- Exposure scenarios assume that adults weigh 70 kg, adolescents weigh 36 kg, and children weigh 20 kg.
- Exposure scenarios assume that all individuals live for 70 years.
- Average concentrations are derived based on the arithmetic mean of sample concentrations (for each constituent) in which detectable levels were reported, samples in which below detectable levels were reported were included in the average as one-half of the reported detection level.
- Concentrations used to assess risk due to exposure to soil do not account for the presence of uncontaminated soil over the contaminated material. That is, the risk assessment assumes exposure to covered soil.
- Throughout the scenarios is the implied assumption that individuals will be maximally exposed to contaminants; that children and adults are exposed to source material or to quarry soil; that children play

in the northern and/or southern drainages every day that they play outside; and that exposure is always to contaminated environmental media.

8.3 RISK SUMMARY FOR THE FPRQ RISK ASSESSMENT

Section 6 and Tables 6-1 through 6-17 provide a detailed discussion and estimation of the risk of adverse effects to human health given one or more of a variety of scenarios under a no-action remedial alterative for the site. These scenarios are to be interpreted with the caveat that very conservative assumptions went into the risk estimates, as described in subsection 8.2 above. Based on the exposure assumptions and calculations presented in the exposure estimation tables (Tables 6.1 through 6.17), potential risks to human health are restricted to a limited set of constituents at the FPRQ site. Furthermore, those limited exposures for which potential risks are identified are limited to areas within the confines of the quarry itself or to surface soils adjacent to the quarry. In addition, scenarios for which potential risks to human health are identified are all potential future use scenarios that make the conservative assumptions that the site undergoes no remediation and is developed for residential use, that exposure is to areas of maximum likely contamination (i.e. leachate, quarry soils, source areas, etc.), and that exposures are high (i.e. exposure every play or gardening day, 70 year lifetime exposure for drinking water, etc.). It should be emphasized that not only is it highly unlikely that the FPRQ site would be developed for residential use in the foreseeable future, the remaining assumptions in these scenarios are also unlikely in their conservatism.

Under the assumptions inherent in the "average case" calculations for the various scenarios, the following areas of potential concern are identified.

- o - Quarry Leachate - exposure to antimony, barium, lead, arsenic, and benzene as a consequence of future use of the quarry leachate as a source of potable water.

- o - Source Material - exposure to lead as a consequence of soil ingestion by children playing in source material.

- o - Northern Drainage Sediments - exposure to arsenic as a consequence of incidental ingestion of soil by children playing in sediments.

- o - Quarry Soil - exposure to lead as a consequence of soil ingestion by children playing in quarry soil.

- o - Northern Drainage Soil - exposure to arsenic as a result of ingestion of soil by children playing and adults gardening in Northern Drainage soil.

Given the extremely conservative assumptions inherent in the "reasonable worst case" calculations for the various scenarios, the following additional areas of concern were identified.

- o - Source Material - exposure to arsenic as a result of ingestion by children playing and adults gardening in source material and exposure to lead as a consequence of adults ingesting soil while gardening in source material.

- o - North and South Ponds - exposure to arsenic as a consequence of incidental ingestion of water by children playing in water.

- o - Northern Drainage Sediments - exposure to barium and lead as a consequence of incidental ingestion of soil by children playing in sediments.

- o - Quarry Soils - exposure to lead and arsenic by adults as a consequence of ingestion of soil while gardening in quarry soils and exposure to arsenic by children as a result of playing in quarry soils.

- o - Northern Drainage Soils - exposure to lead by children as a consequence of incidental ingestion of soil while playing in Northern Drainage soils.

- o - Southern Drainage Soils (Disturbed Area) - exposure to arsenic as a result of ingestion by children playing and adults gardening in Southern Drainage soils.

The following are summaries of the risks associated with each suite of exposure scenarios, given the conservatism inherent in the analyses.

AREAS OF POTENTIAL CONCERN

Area or Medium	Constituent	Hazard Index or Lifetime Cancer Risk[1] Average Exposure	Reasonable Worst Case
Quarry Leachate	Antimony	3.39	8.25
	Barium	11.66	34.70
	Lead	193.88	675.00
	Arsenic	6.46e-3	1.16e-2
	Benzene	1.07e-5	1.27e-5
Source Material	Lead	1.58	41.83
	Arsenic	--[2]	2.87e-5
North and South Ponds	Arsenic	--	2.38e-5
Northern Drainage Sediments	Barium	--	1.05
	Lead	--	3.10
	Arsenic	1.12e-5	3.07e-4
Quarry Soil	Lead	1.39	39.14
	Arsenic	--	3.17e-5
Northern Drainage Soil	Lead	--	10.90
	Arsenic	5.76e-6	9.62e-5
Southern Drainage Soil	Arsenic	--	7.51e-6

[1] Values are for children where they are greater than for adults.
[2] Values of Hazard Index less than 1, or Cancer Risk less than or equal to 1e-6.

8.3.1 Source Areas and Leachate in Quarry

This suite of exposure scenarios assumes that the FPRQ site, at some time in the future, is developed for residential purposes with no removal of source material. Tables 6-1 through 6-4 provide estimates of risk from exposure to

source material and leachate as a result of direct ingestion of leachate, ingestion of source material in the quarry by playing children and gardening adults, and incidental ingestion of water in the North and South Ponds.

Exposure estimates provided in Table 6-1 indicate that human ingestion of leachate from the FPRQ site could pose a significant risk to human health in terms of both chronic exposure to the non-carcinogens (particularly antimony, barium, and lead) and to the carcinogens, arsenic and, to a lesser extent, benzene. However, to be at the levels of risk estimated here, one would have to drink leachate from the site as one's sole source of potable water.

Exposure estimates provided in Tables 6-2 and 6-3 indicate that incidental ingestion of source material from the FPRQ site could pose significant risk to human health in terms of chronic exposure to the non-carcinogen lead among children. Under reasonable worst case assumptions, a maximally exposed adult could be at risk as a result of exposure to lead. Also under reasonable worst case assumptions, lifetime exposure of both adults and children to the carcinogen arsenic could result in risk on the order of 3 in 100,000 incremental lifetime cancers, given a lifetime of gardening and playing in source material.

Exposure estimates provided in Table 6-4 indicate that incidental ingestion by children of surface water from the North and South Ponds in the quarry itself would pose no significant risk to human health. Under reasonable worst case assumptions, incremental lifetime cancer risk could be on the order of 2 in 100,000.

In summary, there is a potential risk to human health due to exposure to leachate and to source material if the quarry itself is, at some time in the future, developed for residential use; that no provisions are made to remove

segregate, or treat source material; and the assumptions detailed above apply. The pica child and the maximally exposed adult could be exposed to an increased incremental risk of cancer due to exposure to source material and to North and South Pond Water.

8.3.2 Down Gradient Ground Water

This scenario is based on a lifetime consumption of ground water in the vicinity of the FPRQ site by residents. Exposure estimates provided in Table 6-5 indicate that incidental ingestion by residents of ground water down gradient of the quarry would pose no significant risk to human health in terms of chronic exposure to the non-carcinogens or in terms of lifetime exposure to carcinogens.

8.3.3 Surface Water and Sediment

This suite of exposure scenarios assumes that the vicinity of the FPRQ site, at some time in the future, is developed for residential purposes, with subsequent exposure of children to surface water and sediments in the northern and southern drainages and/or use of Lawless Creek as a source of potable water by residents.

Tables 6-6 and 6-7 provide estimates of risk from exposure to northern and southern drainage water as a result of incidental ingestion by children during play. The exposure estimates provided in these two tables indicate that such ingestion would pose no risk to human health in terms of either chronic exposure to the non-carcinogens or lifetime exposure to carcinogens.

Tables 6-8 and 6-9 provide estimates of risk from exposure to northern and southern sediments as a result of incidental ingestion by children during play.

The exposure estimates provided in these two tables indicate that such ingestion would pose no risk to human health in terms of chronic exposure to the non-carcinogens. In terms of exposure to the carcinogen arsenic, children playing in the northern drainage sediments could have an increased lifetime cncer risk of 1 in 100,000. Under the reasonable worst case assumptions, a child with pica could be at risk, primarily as a result of barium and lead concentrations in the northern drainage sediments.

Table 6-10 provides estimates of risk from exposure to Lawless Creek water. This scenario is based on a lifetime consumption of water from Lawless Creek by residents. Exposure estimates provided in Table 6-10 indicate that incidental ingestion by residents of water from Lawless Creek would pose no significant risk to human health in terms of chronic exposure to the non-carcinogens or in terms of lifetime exposure to carcinogens. As indicated in Table 6-10, under reasonable worst case assumptions, use of Lawless Creek water as a potable water supply could pose a slight risk to human health as a result of the combination of cadmium and lead. However, Lawless Creek is not classified as a public water supply.

In summary, there is no risk to human health that would be expected to result from exposure to surface water. A child playing in the northern drainage sediments could be at risk due to ingestion of arsenic. There is a potential risk for the pica child that could result from exposure to sediments in the upper northern drainage, particularly with respect to the presence of barium and lead, if the assumptions detailed above obtain, if no means are taken to remove or segregate drainage sediments, and if children play in and eat those sediments.

8.3.4 Soil

This suite of exposure scenarios also assumes that the vicinity of the FPRQ site, at some time in the future, is developed for residential purposes with subsequent incidental exposure of adults and children to soils both in and adjacent to the quarry. Tables 6-11 through 6-16 provide estimates of risk from exposure to soils as a result of incidental ingestion by children during play and by adults during gardening.

Table 6-11 provides estimates of risk from exposure by incidental ingestion of quarry soils by children. The exposure estimates provided in Table 6-11 indicate that such ingestion could pose risk to children in terms of chronic exposure to the non-carcinogen lead. Under the reasonable worst case assumptions, the pica child could be at risk due to levels of lead in the quarry soil and could have an increased lifetime cancer risk of 3 in 10,000 due to arsenic.

Table 6-12 provides estimates of risk from exposure by incidental ingestion of quarry soils by adults. The exposure estimates provided in Table 6-12 indicate that such ingestion would pose no risk to adults. Under the reasonable worst case assumptions, a maximally exposed adult could have an incremental cancer risk of 3 in 100,000 and could be at risk due to exposure to lead.

Tables 6-13 and 6-14 provide estimates of risk from exposure by incidental ingestion of soils in the north drainage area by children and adults. The exposure estimates provided in these two tables indicate that such ingestion would pose no risk to children or adults in terms of chronic exposure to the non-carcinogens. Lifetime exposure to arsenic by both children and adults could result in a marginal increase in cancer by a factor of approximately 6 in

1,000,000 in both children and adults. Given the reasonable worst case assumptions, the pica child would be at risk due to exposure to lead, and both the pica child and the maximally exposed adult would be at risk due to exposure to arsenic, with an incremental cancer risk of approximately 1 in 10,000.

Potential exposures of children and adults are estimated in Tables 6-15 and 6-16 for the disturbed area of soil in the southern drainage. These exposure estimates indicate that the soils in this area pose no risk to either children or adults as a result of exposures to non-carcinogens or to carcinogens. However, under reasonable maximum exposure assumptions, the pica child and the maximally exposed adult could have a lifetime incremental cancer risk of approximately 8 in 1,000,000 as a result of incidental ingestion of arsenic.

In summary, exposure to soils in the quarry could result in potential risks to human health in terms of exposure of children to lead. Exposure to soils in the upper northern drainage could result in risks due to the presence of arsenic. Under the reasonable worst case assumptions, the pica child and the maximally exposed adult could be at risk due to exposure to lead and arsenic in quarry soils and to arsenic in the adjacent drainage soils. The maximally exposed child could be at risk due to exposure to lead in adjacent northern drainage soils.

8.3.5 Air

This scenario is based on a lifetime exposure to contaminated dust by a hypothetical person located approximately 10 meters from an unpaved road running through the quarry. Exposure estimates provided in Table 6-17 indicate that inhalation by residents of dust contaminated by material from the site would pose no risk to human health in terms of chronic exposure to the non-carcinogens or

in terms of lifetime exposure to carcinogens.

SECTION 9

ENVIRONMENTAL EFFECTS ASSESSMENT

Section 9.0 is an evaluation of the potential adverse effects to the environment posed by the FPRQ site, assuming the no-action remedial alternative. Potential environmental effects are qualitatively discussed for three sectors: physical, biological, and human. The previous section of this technical memorandum addressed potential impacts (health effects) to humans. The discussion in this section is presented in terms of potential environmental impacts. However, few such impacts, if any, are anticipated.

9.1 PHYSICAL ENVIRONMENT

The FPRQ site, given the no-action alternative, would result in little to no adverse environmental effects on most components of the physical environment. The ground and surface water components have the highest potential for localized adverse effects.

9.1.1 Physiography/Topography

The immediate area of the site is approximately four acres. The FPRQ has been a capped landfill since the early 1970's. The site topography was altered by earlier quarrying operations and further modified by subsequent land fill operations. However, there are no operations currently under way at the site, except those related to RI/FS investigations, that would alter or have any other effect on site physiography or topography.

Given the no-action alternative for the site, no alteration or change to site physiography or topography would occur. However, if construction-based remedial actions were proposed for the site, alterations to the physiography and topography would be likely to occur in as much as the areal extent of the site is disturbed by such construction activities.

9.1.2 Climate

The site is too small to have any significant effect, if at all, on local climatological factors. The two ponds on site are not sufficiently large to affect either local temperature or precipitation.

9.1.3 Geology

The area and volume of the site is relatively small. Considering the past use of the property for quarrying operations and the relatively small size of the FPRQ site landfill itself, it is unlikely that the site would have any adverse environmental effects on local or regional geology.

9.1.4 Soils

The area and volume of the site is relatively small. Considering the past use of the property for quarrying operations and the relatively small size of the FPRQ site landfill itself, it is unlikely that the site would have any adverse environmental effects on local or regional soils. Within the quarry, some soils have increased levels of metals or other contaminants, however, the no-action alternative would not result in significant future degradation of off-

site soils since off-site migration of contaminants is negligible to non-existent.

9.1.5 Ground Water

The extent of ground water impacts at the site are limited. Ground water sampling data indicate minimal level of metals in ground water in the immediate vicinity of the site. Ground water use in the vicinity of the FPRQ has not been impaired by contaminants at the site, since available data indicate that domestic water wells in the area are all located hydraulically upgradient of the quarry. This observation has been verified by analytical results from water well sampling by the U.S. Environmental Protection Agency as well as ground water analyses conducted as part of the RI activities.

Ground water occurrence, flow, and hydraulic properties are not expected to be adversely affected by current conditions at the FPRQ site nor by continuation of these conditions under the no-action alternative.

9.1.6 Surface Water

Surface water impacts at the site are minimal, based on available data obtained during the RI activities. The source area sampling data indicate that surface water within the site has some elevated concentrations of a few constituents.

Surface water occurrence and flow would not be adversely affected by current conditions at the FPRQ site given a continuation of the no-action alternative.

9.1.7 Air

Air quality is not likely to be adversely affected by volatiles, as only very low concentrations have been detected at the site. There is a possibility that some contaminants may be entrained with fugitive dust in as much as dust is generated from quarry soils. However, as indicated in subsection 6.5, it is highly unlikely that site contaminants would be carried from the site. Due to the low topographic setting and the dense surrounding vegetation, actual transport of fugitive dust emissions from the site would be even less than estimated in the risk assessment.

9.2 BIOTIC ENVIRONMENT

The FPRQ site, given the no-action alternative, would result in little to no adverse environmental effects on the biological environment. Habitat areas influenced by surface water or animals that might come into contact with contaminated surface water or soils within the site itself would have the highest potential to be adversely affected.

9.2.1 Aquatic and Wetland Habitat

No adverse effects on aquatic or wetland habitat were observed in the vicinity of the FPRQ site. There is a potential for contamination of surface water in the immediate vicinity of the site, particularly in the regions of the northern and southern drainages near the site. However, due to adsorption and dilution of contaminants, there is no evidence of adverse effects by the time water from these drainages reaches Lawless Creek.

It is possible that contaminated water, such as the two ponds in the quarry itself, may exert an adverse environmental effect on pond biota and on biota along the pond banks. Lack of observations of adverse effects are not sufficient to rule out the possibility of subtle effects on aquatic and emergent biota as a result of surface water contamination from the quarry site. It should be noted, however, that the quarry ponds are an integral part of the site, and exist only as a result of the landfill. Similarly, the areas of hydrophytic vegetation along the northern drainage are attributed to the change in the hydrologic system caused by the site.

As part of the RI activities, samples of surface water and sediments were collected along the north drainage and Lawless Creek for aquatic toxicity testing. The samples collected underwent the following tests:

- Four, 7-day chronic toxicity tests using Ceriodaphnia and fathead minnows were conducted on the surface water samples. Three of the tests (FP-802, FP-803, and FP-804) were undiluted samples and one (FP-801) contained a full set of serial dilutions.
- Four, 7-day chronic toxicity tests using Ceriodaphnia were conducted on the sediment elutriate. All of these tests were full serial dilutions.

Biological toxicity tests were performed by Biological Monitoring, Inc. (BMI), Blacksburg, Virginia. U.S. Environmental Protection Agency Test Method 1000.0 and 1002.0 were utilized to conduct the tests. Appendix A of the RI Report contains a more detailed description of the biological toxicity tests conducted on these samples. In addition, the Subtask 4.14 Technical Memorandum: Bioassessment Sampling and Analysis (Westinghouse 1989f) presents laboratory analysis data and report prepared by Biological Monitoring, Inc. of Blacksburg,

Virginia as Appendix A. Appendix B of that memorandum presents Field Sampling and Analysis forms showing the field parameters measured.

Survival of test Ceriodaphnia and fathead minnows in surface water test results indicate no effect. Survival test results did not differ from controls for any test samples analyzed. In the serial dilution test performed on the water sample from Station FP-801, closest to the landfill, reduced growth in minnows and reduced reproduction in Ceriodaphnia were reported at test concentrations of 60 and 100 percent. In addition, reduced reproduction was reported among Ceriodaphnia tested in the water sample from Station FP-802. No other effects on growth or reproduction were observed in any of the other tests.

Survival of test Ceriodaphnia and fathead minnows in sediment elutriate tests from all stations did not differ from controls. BMI reported that there was no statistically significant difference in reproduction for Ceriodaphnia or growth for minnows between any of the sediment elutriate tests and the controls. However, the Ceriodaphnia control in this test exhibited reproduction rates lower than normally seen at the laboratory. Reproduction in the elutriate tests were higher than the control, but within typical ranges of reproductive rates normally seen at the laboratory.

These test results indicate that the surface water in the northern drainage and in Lawless Creek were not acutely or subchronically toxic to the test organisms. However, surface water from the upper reaches of the northern drainage (FP-801) did result in significant decreases in growth among fathead minnows and reproduction among Ceriodaphnia at concentrations above 30 percent dilution. Undiluted water from the lower northern drainage (FP-802) resulted in decreased reproduction of Ceriodaphnia.

To evaluate the potential for adverse effects of the effluent leachate in the upper northern drainage at the site (FP-801) on Lawless Creek biota, the potential dilution of flow from the northern quarry into the northern drainage water and into Lawless Creek is considered. Calculations presented in subsection 4.7.2 of the RI Report indicate that the mean annual flow from the northern portion of the quarry contributes approximately 7.7 and 0.07 percent of the mean annual flow of the northern drainage basin and of Lawless Creek, respectively. Comparisons of effluent dilution into receiving waters are generally based on 7Q10 values, which represent the 7-day low flow during the lowest 10-year flow of the receiving stream. Such values are not available for the northern drainage basin or for Lawless Creek in the vicinity of the site. However, since flow from the quarry into the northern drainage is a direct function of ground water levels and surface runoff, it would be expected that dilution ratios of upper northern drainage flow in the northern drainage basin and into Lawless Creek would be relatively constant under a wide variety of flow regimes. The bioassay results also show that lower northern drainage and Lawless Creek water samples were not acutely or subchronically toxic to test organisms and did not show adverse effects on growth or reproduction, indicating lack of adverse environmental effect on the receiving stream (Lawless Creek).

Within the southern drainage, several constituents were reported in water samples at concentrationsin excess of ARARs. With respect to the Clean Water Act criteria for protection of aquatic life, the following table compares acute and chronic aquatic life criteria with surface water analytical data from the southern drainage.

Constituent	Southern Drainage Concentration (mg/l) Mean	Maximum[1]	CWA - Protection of Aquatic Life (mg/l) Acute	Chronic
Cadmium	0.0449	0.0813	0.0039	0.0011
Copper	0.0183	0.0252	0.018	0.012
Lead	0.00283	0.00474	0.080	0.0032
Nickel	0.023	0.0328	1.4	0.16
Zinc	49.1	88.7	0.13	0.11

[1] Maximum is the lesser of the upper 95% confidence level or maximum observed value.

Cadmium and zinc concentrations in the southern drainage surface water samples are appreciably higher than appropriate ARARs for the protection of aquatic life.

9.2.2 Woodland Habitat

The woodland habitat is not expected to be adversely affected by the FPRQ site under the no-action alternative. The woodland areas are separated from the site and from streams and other aquatic areas where there may be contaminated ground or surface water. It is possible, however, that some species of woodland

fauna may drink from ponds, seeps, streams, or springs that are potentially contaminated with constituents from the FPRQ site. However, since the surface water ponds in the quarry represent small areas, it is unlikely that any game species would utilize these waters to such an extent that significant toxic effects would result.

9.2.3 Edge Habitat

The edge habitat is not expected to be adversely affected by the FPRQ site under the no-action alternative. The edge areas are separated from the site and from streams and other aquatic areas where there may be contaminated ground or surface water.

9.2.4 Agricultural and Farm Habitat

Agricultural habitat and farm species are not expected to be adversely affected by the FPRQ site under the no-action alternative. Agricultural areas are separated from the site and from streams and other aquatic areas where there may be contaminated ground or surface water.

As noted in Section 2.0 of this risk assessment, approximately 100 head of cattle are grazed along Lawless Creek. These cattle would no be expected to be adversely affected by contaminants in Lawless Creek. As estimated in Section 6.3.3 of this risk assessment, lifetime human consumption of water from Lawless Creek presented no likelihood of adverse effects to human health. Because of the safety margins inherent in the analysis and the safety margins inherent in the health-based criteria, it is equally unlikely that water from Lawless Creek would pose a potential for adverse effect for livestock.

9.2.5 Threatened and/or Endangered Species

There are no identified threatened and/or endangered species in the vicinity of the FPRQ site. Therefore, there are no anticipated adverse environmental effects on such species as a result of the site.

9.3 HUMAN ENVIRONMENT

Potential adverse effects to the human environment from the FPRQ site under the no-action remedial alternative would be primarily those associated with potential adverse health effects. Such effects were addressed in the preceding sections of this risk assessment.

9.3.1 Land Use

At present, the FPRQ site is partially fenced with restricted access.

9.3.2 Human Populations

As described in previous sections of this risk assessment, it is unlikely that the FPRQ would pose significant adverse effects to human populations under the no action alternative. The primary risks to human health associated with the site are those involving direct contact with site soils and source material.

9.3.3 Historical and Archaeological Resources

There are no known historical and archaeological resources that would be adversely affected in the vicinity of the FPRQ site.

SECTION 10

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CHATHAM, VA

TABLE 1 — Climatological Data Summary For Chatham, VA

PERIOD: 1951-80
ELEVATION: 700 FT

CLIMATOLOGICAL SUMMARY

	TEMPERATURE (F)															PRECIPITATION TOTALS (INCHES)											
	MEANS			EXTREMES						MEAN NUMBER OF DAYS				DEGREE DAYS						SNOW			MEAN NUMBER OF DAYS		
										MAX		MIN															
	* DAILY MAXIMUM	* DAILY MINIMUM	* MONTHLY	RECORD HIGHEST	YEAR	DAY	RECORD LOWEST	YEAR	DAY	90 AND ABOVE	32 AND BELOW	32 AND BELOW	0 AND BELOW	* HEATING BASE 65	* COOLING BASE 65	* MEAN	* GREATEST MONTHLY	YEAR	GREATEST DAILY	YEAR	DAY	MEAN	MAXIMUM MONTHLY	YEAR	.10 OR MORE	.50 OR MORE	1.00 OR MORE
JAN	46.5	25.1	35.8	76+	75	30	-1	72	16	0	3	24	0	905	0	3.41	8.53	78	2.02	79	21	6.4	35.5	66	6	2	1
FEB	49.0	26.3	37.7	81+	77	27	-2	79	10	0	2	21	0	764	0	3.32	6.26	60	2.02	79	25	3.9	17.0	60	7	2	1
MAR	57.7	33.9	45.8	86+	68	22	7+	80	4	0	0	15	0	595	0	4.16	11.76	75	3.58	75	30	2.8	17.5	60	8	3	1
APR	69.0	42.6	55.8	92+	80	24	22+	77	9	0	0	5	0	284	8	3.54	7.71	78	3.79	78	27	.0	.0	0	6	2	1
MAY	77.3	51.5	64.4	95+	62	20	28+	77	10	1	0	1	0	111	92	3.89	10.73	71	4.44	71	13	.0	.0	0	7	3	1
JUN	83.7	59.0	71.4	100+	52	28	33+	77	8	6	0	0	0	17	209	3.60	8.19	72	3.15	72	21	.0	.0	0	6	2	1
JUL	87.6	63.6	75.6	104+	52	30	46+	79	8	11	0	0	0	0	329	3.94	7.90	65	2.78	64	13	.0	.0	0	6	3	1
AUG	86.5	62.7	74.6	101+	54	17	38+	76	31	10	0	0	0	0	298	3.80	8.69	67	6.47	64	31	.0	.0	0	6	2	1
SEP	80.8	55.6	68.3	104+	54	7	32	51	30	4	0	0	0	49	148	3.79	10.81	79	5.09	79	22	.0	.0	0	5	2	1
OCT	70.4	43.3	56.9	96+	51	6	20+	76	19	0	0	5	0	276	24	3.55	10.18	76	4.05	54	16	.0	.0	0	6	2	1
NOV	59.7	34.9	47.3	85+	74	2	10	56	25	0	0	14	0	531	0	3.09	7.49	57	3.46	62	10	.5	5.0	68	5	2	1
DEC	49.7	27.5	38.6	78+	71	17	-2	80	26	0	1	22	0	818	0	3.39	7.53	67	4.44	58	29	1.6	8.0	66	6	2	1
YEAR	68.2	43.8	56.0	104	SEP 54	7	-2	DEC 80	26	32	6	107	0	4350	1108	43.48	11.76	MAR 75	6.47	AUG 64	31	15.2	35.5	JAN 66	74	27	12

*FROM 1951-80 NORMALS

ESTIMATED VALUE BASED ON DATA FROM SURROUNDING STATIONS

+ ALSO ON EARLIER DATES.

DEGREE DAYS TO SELECTED BASE TEMPERATURES (F)

BASE	* HEATING DEGREE DAYS												
BELOW	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC	ANN
65	905	764	595	284	111	17	0	0	49	276	531	818	4350
60	750	624	446	159	43	0	0	0	14	165	381	663	3245
57	657	540	360	104	21	0	0	0	6	115	297	570	2670
55	595	489	305	74	13	0	0	0	0	86	242	508	2312
50	452	358	187	21	0	0	0	0	0	32	126	368	1544

BASE	COOLING DEGREE DAYS												
ABOVE	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC	ANN
55	0	0	20	98	304	492	639	608	399	145	11	0	2716
57	0	0	13	68	250	432	577	546	345	112	6	0	2349
60	0	0	6	33	180	346	484	453	263	69	0	0	1834
65	0	0	0	8	92	209	329	298	148	24	0	0	1108
70	0	0	0	0	33	106	181	151	69	8	0	0	548

DERIVED FROM THE 1951-80 MONTHLY NORMALS

PROBABILITY THAT THE MONTHLY PRECIPITATION WILL BE EQUAL TO OR LESS THAN THE INDICATED PRECIPITATION AMOUNT

PROBABILITY LEVELS	MONTHLY PRECIPITATION (INCHES)											
	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
.05	1.05	1.01	1.84	1.61	1.55	1.38	1.44	.83	.69	.57	.86	.76
.10	1.37	1.32	2.21	1.93	1.91	1.71	1.81	1.19	1.03	.88	1.15	1.08
.20	1.86	1.79	2.73	2.36	2.42	2.19	2.34	1.74	1.58	1.39	1.60	1.58
.30	2.27	2.20	3.15	2.71	2.84	2.59	2.79	2.25	2.10	1.88	1.99	2.02
.40	2.67	2.59	3.54	3.03	3.24	2.96	3.21	2.75	2.63	2.38	2.36	2.47
.50	3.09	3.00	3.94	3.36	3.64	3.35	3.65	3.29	3.19	2.93	2.76	2.95
.60	3.55	3.45	4.36	3.71	4.08	3.77	4.12	3.89	3.84	3.56	3.20	3.48
.70	4.09	3.98	4.84	4.11	4.58	4.25	4.67	4.62	4.62	4.33	3.72	4.12
.80	4.78	4.66	5.45	4.62	5.22	4.86	5.37	5.57	5.67	5.36	4.39	4.96
.90	5.87	5.73	6.38	5.38	6.20	5.81	6.45	7.09	7.34	7.03	5.45	6.30
.95	6.87	6.71	7.22	6.07	7.09	6.67	7.43	8.52	8.94	8.62	6.43	7.56

THESE VALUES WERE DETERMINED FROM THE INCOMPLETE GAMMA DISTRIBUTION.

TABLE 2

Summary of Average Monthly Climatic Data
Westinghouse Job No. 4112-88-907B

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Temp.[1] (°F)	35.8	37.7	45.8	55.8	64.4	71.4	75.6	74.6	68.3	56.9	47.3	38.6	----
Wind Speed[2](mph)	8.8	8.8	9.3	9.2	7.7	6.9	6.6	6.3	6.9	7.4	7.9	7.8	----
Wind Direction[2]	SW	SW	SW	SW	SW	SW	SW	N	N	N	SW	SW	----
Precip.[1](inches)	3.41	3.32	4.16	3.54	3.89	3.60	3.94	3.80	3.79	3.55	3.09	3.39	43.48
Pan Evaporation[3]	ND	ND	ND	4.04	4.36	4.69	4.83	4.58	3.37	2.66	ND	ND	52[3]
Potential Evapotranspiration[4]	0.16	0.26	0.99	2.35	3.96	5.31	6.23	5.68	3.81	2.08	0.88	0.27	32.09
Potential Moisture Deficit/Excess[5]	+3.25	+3.06	+3.17	+1.19	-0.07	-1.71	-2.29	-1.89	-0.12	+1.47	+2.21	3.12	+11.39

1. County of Pittsylvania Comprehensive Plan, 1986. Derived from the 1951-1980 monthly normals for Chatham, VA.

2. Ruffner and Bair, 1987. Derived from the 1951-1980 monthly normals for Lynchburg, VA.

3. National Climate Data Center, 1988. Average monthly total pan evaporation derived from the period 1967-1983 for Philpott, VA. Data not available for November through March. Total pan evaporation calculated to be 52 inches. Pan coefficient = 0.77. Lake evaporation = 0.77 pan evaporation.

4. State Climatology Board, 1988. Derived from the 1951-1980 monthly normals for Danville, VA, calculated using Thornthwaite method.

5. Potential moisture deficit excess = Precipitation minus potential evapotranspiration. + = excess, - = deficit.

TABLE 3

Pittsylvania County Population Data by Age Bracket and Gender[1]
Westinghouse Job No. 4112-88-907 B

Pre-School Population - Under 5 Years of Age			
1970	4,946	2,508 male	2,438 female
1980	4,423	2,252 male	2,171 female
% changed	-10.6 %	-10.2%	-11.0%
School Age Population - 5 to 14 Years of Age			
1970	12,811	6,464 male	6,347 female
1980	10,600	5,493 male	5,107 female
% changed	-17.3%	-15.0%	-19.5%
High School - Working Age 15 to 59 Years of Age			
1970	33,643	16,576 male	17,076 female
1980[2]	40,892	20,130 male	20,130 female
% changed	+21.5%	+21.4%	21.6%
Pre-Retirement Age - 60 to 64 Years of Age			
1970	2,330	1,123 male	1,207 female
1980	3,094	1,399 male	1,695 female
% changed	+32.8	+24.6	+40.4
Retired - Elderly - 65 Years of Age and Older			
1970	5,059	2,284 male	2,775 female
1980	7,138	3,145 male	3,993 female
% changed	+41.1%	+37.7 %	+43.9%
Total County Population			
1970	58,789		
1980	66,147		

1. From County of Pittsylvania Comprehensive Plan,1986.
2. Data as reported in source document.

TABLE 4

Summary of Population Data for Community of Beaver Park
Westinghouse Job No. 4112-88-907B

1972 VADOT Aerial Photograph	U.S.G.S. Map	Tax Records	Site Reconnaissance	RI/FS Work Plan	1988 Dewberry & Davis Aerial Photograph
328	269	242	257	260	266

TABLE 5
Target Constituents - First Piedmont Rock Quarry RI/FS

16 inorganics	18 inorganics
arsenic	aldrin
barium	benzene
beryllium	bis(2-chloroethyl) ether
cadmium	2-chloroethylvinyl ether
chromium	ethylbenzene
copper	hepachlor epoxide
iron	4-methyl-2-pentanone
lead	n-nitrosodiphenylamine
manganese	phenol
mercury	phenanthrene
nickel	chrysene
selenium	di-n-butyl phthalate
silver	pyrene
thallium	tetrachloroethene
vanadium	1,1,1-trichloroethane
zinc	bis(2-ethylhexyl)phthalate
	trichlorofluoromethane
	xylene (total)

(aexp611.cal)
04-Dec-90

TABLE 6-1
INGESTION OF LEACHATE

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	4.75e-2	6.6e-2	1.36e-3	3.3e-3	3.39	8.25	
BARIUM	5e-2	2.04e1	3.47e1	5.83e-1	1.735e0	11.66	34.70	
BIS (2 EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	7.5e-3	8.9e-3	2.14e-4	4.45e-4	.43	.89	
COPPER	3.7e-2	1.72e-2	2.95e-2	4.91e-4	1.48e-3	.01	.04	
LEAD	1.4e-4	9.5e-1	1.89e0	2.71e-2	9.45e-2	193.88	675.00	
MANGANESE	5e-1	8.08e-1	9.45e-1	2.31e-2	4.73e-2	.05	.09	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	3.1e-2	5.21e-2	8.86e-4	2.61e-3	.04	.13	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	2.08e0	3.82e0	5.94e-2	1.91e-1	.30	.96	
					TOTAL	209.76	720.06	

ASSUMPTIONS and NOTES

Ingested metals are 100% bioavailable; concentrations of leachate are dissolved concentrations.
Exposure averaged over a 1-year period.
Average = Daily ingestion by 70 kg adults of 2 liters of the arithmetic mean concentration of leachate in the quarry and seeping into the North Drainage.
Worst Case = Daily ingestion by 20 kg child of 1 liter of the lower of the 95% confidence limits of the mean or the highest observed concentration of leachate in the quarry or seeping into the North Drainage.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	1.1e-1	1.97e-1	3.69e-3	6.61e-3	6.46e-3	1.16e-2
BENZENE	A	2.9e-2	1.1e-2	1.3e-2	3.69e-4	4.36e-4	1.07e-5	1.27e-5
BIS (2 EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	7.5e-3	8.9e-3	2.52e-4	2.99e-4	0	0
NICKEL	NA	NA	3.1e-2	5.21e-2	1.04e-3	1.75e-3	0	0
						TOTAL	6.47e-3	1.16e-2

ASSUMPTIONS and NOTES

Assumptions for exposure to non-carcinogenic compounds apply.
NA = Non-carcinogenic by the oral route.
Life-time exposure assumes 10 years of childhood (20 kg, 1 liter/d), 5 years adolescence (36 kg, 2 liter/d), and 55 years of adulthood (70 kg, 2 liter/d) with exposure averaged over 70 years.

TABLE 6-1 (Continued)

HUMAN INGESTION OF LEACHATE

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I}{W}$$

where:

Ca = contaminant concentration in mg/l
I = ingestion rate in liters/day
W = weight in kg

EXPOSURE TO CARCINOGENS

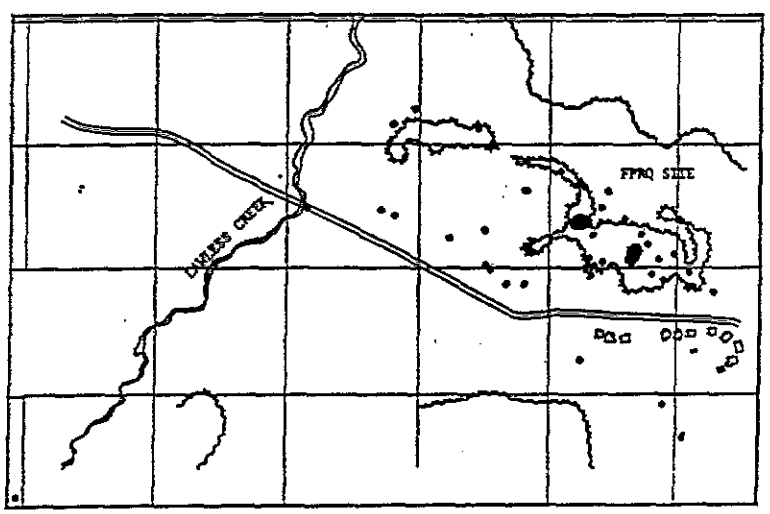
$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times Ca \times I(\alpha)}{W(\alpha)}}{LT}$$

where:

ED(α) = exposure duration
Ca = contaminant concentration in mg/l
I(α) = ingestion rate in liters/day for each ED
W(α) = weight in kg for each ED
LT = lifetime in years

SAMPLE LOCATIONS

FP-404 Leachate
FP-702B Test Pit



(aexp612.cal)
04-Dec-90

TABLE 6-2
INCIDENTAL INGESTION OF SOURCE MATERIAL - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	1.72e0	3.05e0	1.23e-6	2.67e-5	.00	.07	
BARIUM	5e-2	2.49e2	5.69e2	1.77e-4	4.99e-3	.00	.10	
BIS (2EH) PHTHALATE	2e-2	1.17e1	2.93e1	8.33e-6	2.57e-4	.00	.01	
CADMIUM	5e-4	2.25e0	3.66e0	1.60e-6	3.21e-5	.00	.06	
COPPER	3.7e-2	1.75e1	2.79e1	1.25e-5	2.45e-4	.00	.01	
LEAD	1.4e-4	3.11e2	6.68e2	2.22e-4	5.86e-3	1.58	41.83	
MANGANESE	5e-1	1.84e2	2.44e2	1.31e-4	2.14e-3	.00	.00	
MERCURY	2e-3	9.8e-2	1.7e-1	6.98e-8	1.49e-6	.00	.00	
NICKEL	2e-2	4.98e0	6.96e0	3.55e-6	6.10e-5	.00	.00	
SELENIUM	3e-3	3.4e-1	6.01e-1	2.42e-7	5.27e-6	.00	.00	
VANADIUM	9e-3	6.22e0	9.19e0	4.43e-6	8.06e-5	.00	.01	
ZINC	2e-1	1.1e3	2.6e3	7.84e-4	2.28e-2	.00	.11	
					TOTAL	1.60	42.21	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in source material at the site. Ingested concentrations are arithmetic mean of site source samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in source material at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site source samples.

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	4.45e0	7.64e0	7.18e-7	1.64e-5	1.26e-6	2.87e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	1.17e1	2.93e1	1.89e-6	6.29e-5	2.64e-8	8.81e-7
CADMIUM	NA	NA	2.25e0	3.66e0	3.63e-7	7.86e-6	0	0
NICKEL	NA	NA	4.98e0	6.96e0	8.04e-7	1.49e-5	0	0
						TOTAL	1.28e-6	2.96e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-2 (Continued)

INCIDENTAL INGESTION OF SOURCE MATERIAL - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

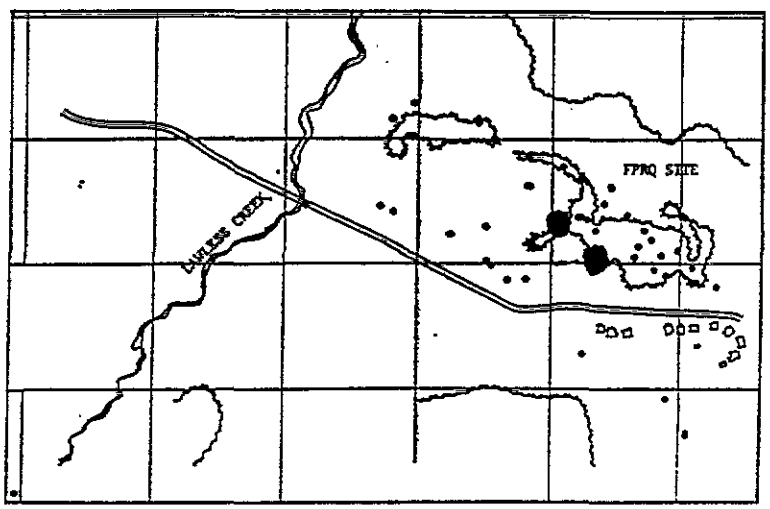
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\dfrac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/kg
- I(α) = soil ingestion rate in kg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS



FP-410 Waste Pile
FP-411 Black Drum
FP-412 Grey Drum
FP-110 Waste Pile
FP-111 Waste Pile
FP-112 Waste Pile
FP-113 Waste Pile
FP-117 Carbon Black
FP-118 Carbon Black
FP-119 Carbon Black
FP-120 Carbon Black

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04-Dec-90

TABLE 6-3
INCIDENTAL INGESTION OF SOURCE MATERIAL - ADULTS

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE DOSE (mg/kg/day) Average Case	EXPOSURE DOSE (mg/kg/day) Reasonable Worst Case	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	1.72e0	3.05e0	1.05e-7	1.91e-6	.00	.00	
BARIUM	5e-2	2.49e2	5.69e2	1.52e-5	3.56e-4	.00	.01	
BIS (2EH) PHTHALATE	2e-2	1.17e1	2.93e1	7.14e-7	1.83e-5	.00	.00	
CADMIUM	5e-4	2.25e0	3.66e0	1.37e-7	2.29e-6	.00	.00	
COPPER	3.7e-2	1.75e1	2.79e1	1.07e-6	1.75e-5	.00	.00	
LEAD	1.4e-4	3.11e2	6.68e2	1.90e-5	4.18e-4	.14	2.99	
MANGANESE	5e-1	1.84e2	2.44e2	1.12e-5	1.53e-4	.00	.00	
MERCURY	2e-3	9.8e-2	1.7e-1	5.98e-9	1.06e-7	.00	.00	
NICKEL	2e-2	4.98e0	6.96e0	3.04e-7	4.36e-6	.00	.00	
SELENIUM	3e-3	3.4e-1	6.01e-1	2.08e-8	3.76e-7	.00	.00	
VANADIUM	9e-3	6.22e0	9.19e0	3.80e-7	5.75e-6	.00	.00	
ZINC	2e-1	1.1e3	2.6e3	6.72e-5	1.63e-3	.00	.01	
					TOTAL	.14	3.02	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 70 kg adult of 60 mg of soil per activity day, adult gardens 2 days per week for 26 weeks per year in source material at the site. Ingested concentrations are arithmetic mean of site source samples.
Worst = Ingestion by a 70 kg adult of 100 mg of soil per activity day, adult gardens 4 days per week for 40 weeks per year in source material at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site source samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE DOSE (mg/kg/day) Average Case	EXPOSURE DOSE (mg/kg/day) Reasonable Worst Case	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	4.45e0	7.64e0	7.18e-7	1.64e-5	1.26e-6	2.87e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	1.17e1	2.93e1	1.89e-6	6.29e-5	2.64e-8	8.81e-7
CADMIUM	NA	NA	2.25e0	3.66e0	3.63e-7	7.86e-6	0	0
NICKEL	NA	NA	4.98e0	6.96e0	8.04e-7	1.49e-5	0	0
						TOTAL	1.28e-6	2.96e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-3 (Continued)

INCIDENTAL INGESTION OF SOURCE MATERIAL - ADULTS

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

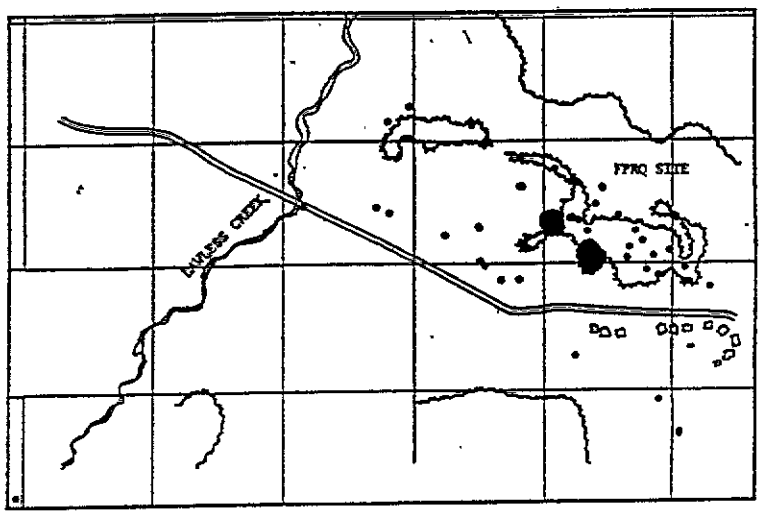
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/kg
- I(α) = soil ingestion rate in kg/day for each ED
- F = daily frequency
- W(α) = weight of human in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS



FP-410 Waste Pile
FP-411 Black Drum
FP-412 Grey Drum
FP-110 Waste Pile
FP-111 Waste Pile
FP-112 Waste Pile
FP-113 Waste Pile
FP-117 Carbon Black
FP-118 Carbon Black
FP-119 Carbon Black
FP-120 Carbon Black

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04-Dec-90

TABLE 6-4
INCIDENTAL INGESTION OF WATER FROM NORTH AND SOUTH PONDS - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	5.62e0	8.42e0	1.00e-3	9.23e-3	.02	.18	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	6.12e-3	8e-3	1.09e-6	8.77e-6	.00	.02	
COPPER	3.7e-2	BDL	BDL	0	0	.00	.00	
LEAD	1.4e-4	1.46e-2	2.11e-2	2.6e-6	2.31e-5	.02	.17	
MANGANESE	5e-1	1.09e0	1.21e0	1.94e-4	1.33e-3	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	BDL	BDL	0	0	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	1.51e-1	2.19e-1	2.69e-5	2.4e-4	.00	.00	
					TOTAL	.04	.37	

ASSUMPTIONS and NOTES

Ingested metals in water are 100% bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 25 ml of water per play day while wading in water, child plays 2 days per week for 26 weeks per year in ponds at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 50 ml of water per play day, child plays 4 days per week for 40 weeks per year in ponds at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	3.8e-2	5.8e-2	1.45e-6	1.36e-5	2.54e-6	2.38e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	6.12e-3	8e-3	2.34e-7	1.88e-6	0	0
NICKEL	NA	NA	BDL	BDL	0	0	0	0
						TOTAL	2.54e-6	2.38e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg), exposures averaged over 70 years. Adults ingest no pond water.
Average = Assumes child ingests 25 ml per play day for 15 years. Play occurs 2 days per week for 26 weeks per year.
Worst = Assumes child ingests 50 ml per play day for 15 years, play occurs 4 days per week, 40 weeks per year.

AR302047

TABLE 6-4 (Continued)

INCIDENTAL INGESTION OF WATER FROM NORTH AND SOUTH PONDS - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F}{W}$$

where:

Ca = contaminant concentration in mg/l
I = ingestion rate in liters/day
F = daily frequency
W = weight of child in kg

EXPOSURE TO CARCINOGENS

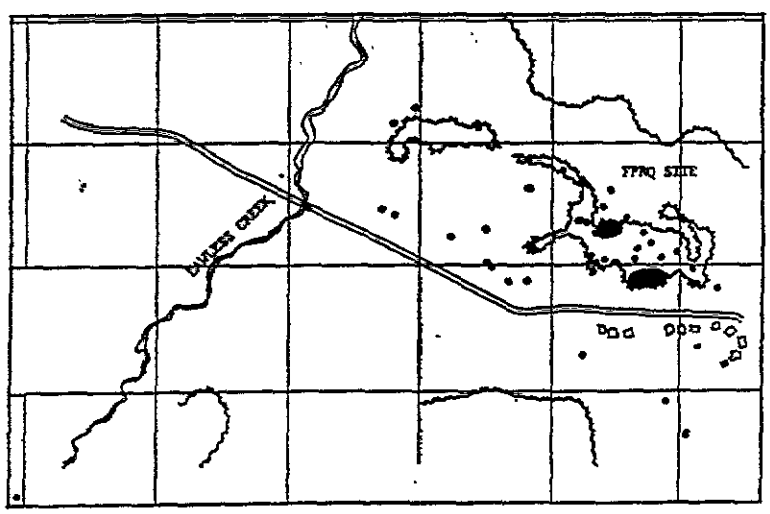
$$\text{Exposure Dose} = \frac{ED \times P \times Ca \times I \times F}{W \times LT}$$

where:

ED = exposure duration
P = proportion of year exposed
Ca = contaminant concentration in mg/l
I = ingestion rate in liters/day
F = daily frequency
W = weight of child in kg
LT = lifetime in years

SAMPLE LOCATIONS

FP-401 South Pond
FP-402 North Pond
FP-403 North Pond(D)



(aexp62.cal)
04-Dec-90

TABLE 6-5
INGESTION OF GROUNDWATER

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	1.12e-3	1.32e-3	3.2e-5	6.6e-5	.08	.17	
BARIUM	5e-2	1.21e-1	1.39e-1	3.46e-3	6.95e-3	.07	.14	
BIS (2 EH) PHTHALATE	2e-2	7.5e-3	8.97e-3	2.14e-4	4.49e-4	.01	.02	
CADMIUM	5e-4	BDL	BDL	0	0	.00	.00	
COPPER	3.7e-2	4.87e-3	6.73e-3	1.39e-4	3.37e-4	.00	.01	
LEAD	1.4e-4	BDL	BDL	0	0	.00	.00	
MANGANESE	5e-1	5.96e-1	1.25e0	1.70e-2	6.25e-2	.03	.13	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	BDL	BDL	0	0	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	1.4e-1	2.23e-1	4e-3	1.12e-2	.02	.06	
					TOTAL	.22	.52	

ASSUMPTIONS and NOTES

Ingested metals are 100% bioavailable; concentrations of material are dissolved concentrations.
Exposure averaged over a 1-year period.
Average = Daily ingestion by 70 kg adults of 2 liters of the arithmetic mean concentration of material in the downgradient groundwater.
Worst Case = Daily ingestion by 20 kg child of 1 liter of the lower of the 95% confidence limits of the mean or the highest observed concentration of material in the downgradient groundwater.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	BDL	BDL	0	0	0	0
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2 EH) PHTHALATE	B2	1.4e-2	7.5e-3	8.97e-3	2.52e-4	3.01e-4	3.52e-6	4.21e-6
CADMIUM	NA	NA	BDL	BDL	0	0	0	0
NICKEL	NA	NA	BDL	BDL	0	0	0	0
						TOTAL	3.52e-6	4.21e-6

ASSUMPTIONS and NOTES

Assumptions for exposure to non-carcinogenic compounds apply.
NA = Non-carcinogenic by the oral route.
Life-time exposure assumes 10 years of childhood (20 kg, 1 liter/d), 5 years adolescence (36 kg, 2 liter/d), and 55 years of adulthood (70 kg, 2 liter/d) with exposure averaged over 70 years.

TABLE 6-5 (Continued)

INGESTION OF DOWN GRADIENT GROUND WATER

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I}{W}$$

where:

Ca = contaminant concentration in mg/l
I = ingestion rate in liters/day
W = weight in kg

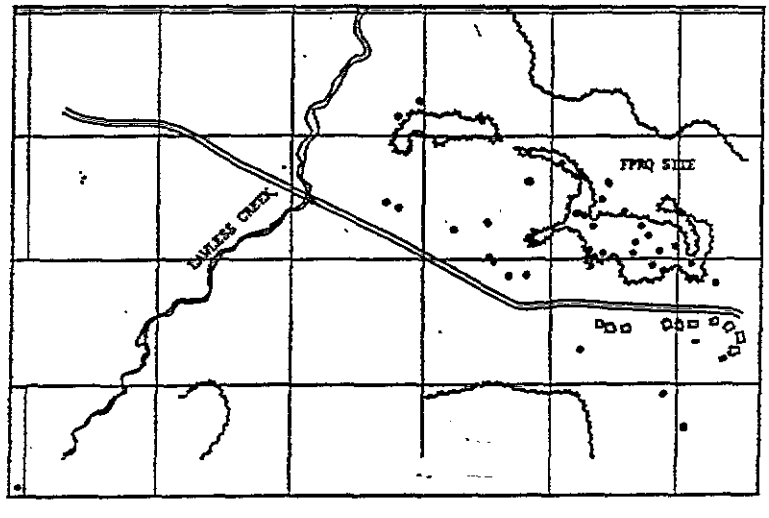
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times Ca \times I(\alpha)}{W(\alpha)}}{LT}$$

where:

ED(α) = exposure duration
Ca = contaminant concentration in mg/l
I(α) = ingestion rate in liters/day for each ED
W(α) = weight in kg for each ED
LT = lifetime in years

SAMPLE LOCATIONS



FP-003A
FP-003B
FP-004
FP-005A
FP-005B
FP-006A
FP-006B
FP-007A
FP-007B
FP-008A
FP-008B
FP-009A
(Dup.FP-003A)

(aexp631.cal)
04-Dec-90

TABLE 6-6
INCIDENTAL INGESTION OF NORTH DRAINAGE WATER - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	1.72e-3	2.59e-3	3.06e-7	2.84e-6	.00	.01	
BARIUM	5e-2	2.38e0	4.3e0	4.24e-4	4.71e-3	.01	.09	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	BDL	BDL	0	0	.00	.00	
COPPER	3.7e-2	BDL	BDL	0	0	.00	.00	
LEAD	1.4e-4	1.78e-3	2.8e-3	3.17e-7	3.07e-6	.00	.02	
MANGANESE	5e-1	7.65e-1	1.35e0	1.36e-4	1.48e-3	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.72e-2	2.16e-2	3.06e-6	2.37e-5	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	2.5e-2	3.52e-2	4.45e-6	3.86e-5	.00	.00	
					TOTAL	.01	.13	

ASSUMPTIONS and NOTES

Ingested metals in water are 100% bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 25 ml of water per play day while wading in water, child plays 2 days per week for 26 weeks per year in water at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 50 ml of water per play day, child plays 4 days per week for 40 weeks per year in water at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	CONCENTRATION Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	6.87e-3	1.18e-2	2.62e-7	2.77e-6	4.59e-7	4.85e-6
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	BDL	BDL	0	0	0	0
NICKEL	NA	NA	1.72e-2	2.16e-2	6.56e-7	5.07e-6	0	0
						TOTAL	4.59e-7	4.85e-6

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg), exposures averaged over 70 years. Adults ingest no water.
Average = Assumes child ingests 25 ml per play day for 15 years. Play occurs 2 days per week for 26 weeks per year.
Worst = Assumes child ingests 50 ml per play day for 15 years, play occurs 4 days per week, 40 weeks per year.

TABLE 6-6 (Continued)

INCIDENTAL INGESTION OF WATER FROM NORTH DRAINAGE - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F}{W}$$

where:

- Ca = contaminant concentration in mg/l
- I = ingestion rate in liters/day
- F = daily frequency
- W = weight of child in kg

EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{ED \times P \times Ca \times I \times F}{W \times LT}$$

where:

- ED = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/l
- I = ingestion rate in liters/day
- F = daily frequency
- W = weight of child in kg
- LT = lifetime in years

SAMPLE LOCATIONS

FP-309(1) Upper N.
FP-309(2) Upper N.
FP-315(2) Upper N.
Dup.FP-309
FP-310(2) Mid. N.
FP-311(1) Lower N.
FP-311(2) Lower N.

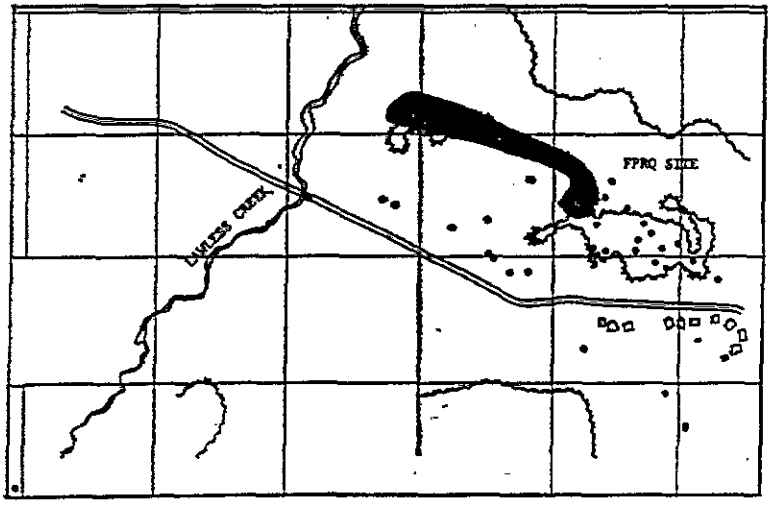


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TABLE 6-7
INCIDENTAL INGESTION OF SOUTH DRAINAGE WATER - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	2.16e-2	3.5e-2	3.85e-6	3.84e-5	.00	.00	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	4.49e-2	8.13e-2	8.00e-6	8.91e-5	.02	.18	
COPPER	3.7e-2	1.83e-2	2.62e-2	3.26e-6	2.87e-5	.00	.00	
LEAD	1.4e-4	2.83e-3	4.74e-3	5.04e-7	5.19e-6	.00	.04	
MANGANESE	5e-1	1.51e0	2.67e0	2.69e-4	2.93e-3	.00	.01	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	2.3e-2	3.28e-2	4.10e-6	3.59e-5	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	4.91e1	8.87e1	8.74e-3	9.72e-2	.04	.49	
					TOTAL	.06	.71	

ASSUMPTIONS and NOTES

Ingested metals in water are 100% bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 25 ml of water per play day while wading in water, child plays 2 days per week for 26 weeks per year in water at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 50 ml of water per play day, child plays 4 days per week for 40 weeks per year in water at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	BDL	BDL	0	0	0	0
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	4.49e-2	8.13e-2	1.71e-6	1.91e-5	0	0
NICKEL	NA	NA	2.3e-2	3.28e-2	8.78e-7	7.70e-6	0	0
						TOTAL	0	0

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg), exposures averaged over 70 years. Adults ingest no water.
Average = Assumes child ingests 25 ml per play day for 15 years. Play occurs 2 days per week for 26 weeks per year.
Worst = Assumes child ingests 50 ml per play day for 15 years, play occurs 4 days per week, 40 weeks per year.

TABLE 6-7 (Continued)

INCIDENTAL INGESTION OF WATER FROM SOUTH DRAINAGE - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F}{W}$$

where:

- Ca = contaminant concentration in mg/l
- I = ingestion rate in liters/day
- F = daily frequency
- W = weight of child in kg

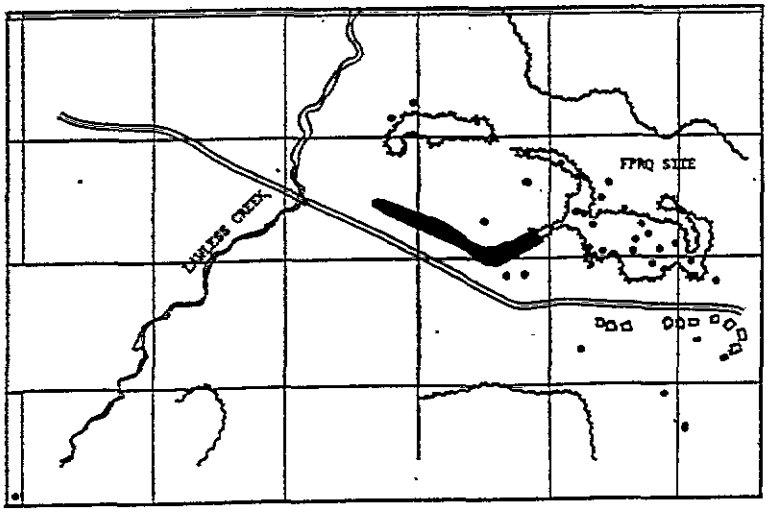
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{ED \times P \times Ca \times I \times F}{W \times LT}$$

where:

- ED = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/l
- I = ingestion rate in liters/day
- F = daily frequency
- W = weight of child in kg
- LT = lifetime in years

SAMPLE LOCATIONS



FP-306(1) Upper S.
FP-306(2) Upper S.
FP-307(2) Mid. S.
FP-308(1) Lower S.
FP-308(2) Lower S.
FP-319(1) Upper S.
Dup. FP-306

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TABLE 6-8
INCIDENTAL INGESTION OF NORTH DRAINAGE SEDIMENTS - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	1.01e0	1.85e0	7.19e-7	1.62e-5	.00	.04	
BARIUM	5e-2	3.06e3	5.99e3	2.18e-3	5.25e-2	.04	1.05	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	1.01e1	2e1	7.19e-6	1.75e-4	.01	.35	
COPPER	3.7e-2	7.92e0	1.01e1	5.64e-6	8.85e-5	.00	.00	
LEAD	1.4e-4	3.22e1	4.95e1	2.29e-5	4.34e-4	.16	3.10	
MANGANESE	5e-1	3.74e2	5.92e2	2.66e-4	5.19e-3	.00	.01	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.84e1	3.64e1	1.31e-5	3.19e-4	.00	.02	
SELENIUM	3e-3	6.89e-1	9.99e-1	4.91e-7	8.76e-6	.00	.00	
VANADIUM	9e-3	1.03e1	1.35e1	7.34e-6	1.18e-4	.00	.01	
ZINC	2e-1	3.05e2	5.08e2	2.17e-4	4.45e-3	.00	.02	
					TOTAL	.23	4.61	

ASSUMPTIONS and NOTES

Ingested metals in sediment are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in sediments at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in sediments at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	4.2e1	9.35e1	6.41e-6	1.76e-4	1.12e-5	3.07e-4
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	1.01e1	2e1	1.54e-6	3.76e-5	0	0
NICKEL	NA	NA	1.84e1	3.64e1	2.81e-6	6.84e-5	0	0
						TOTAL	1.12e-5	3.07e-4

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg), no adult exposure to sediments.
Average = Assumes child ingests 200 mg per play day, 2 days per week for 26 weeks per year.
Worst = Assumes child ingests 400 mg per play day, 4 days per week for 40 weeks per year.

TABLE 6-8 (Continued)

INCIDENTAL INGESTION OF NORTH DRAINAGE SEDIMENTS - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

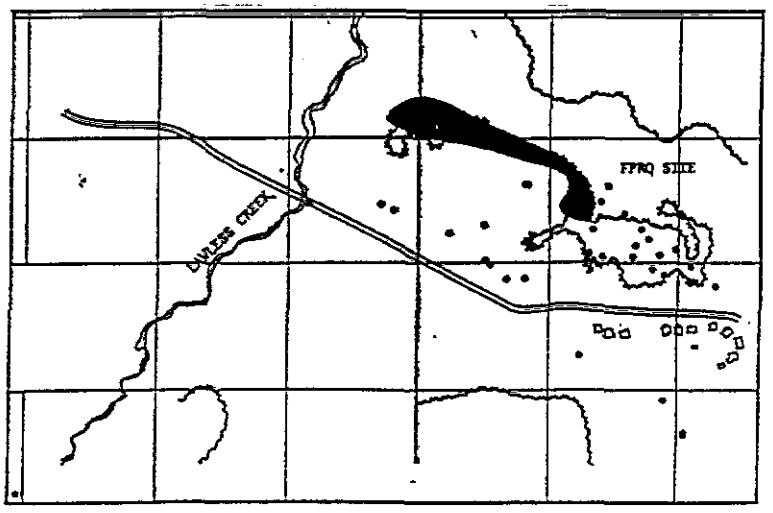
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in kg/kg
- I(α) = soil ingestion rate in mg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS



FP-209(1) Upper N.
FP-209(2) Upper N.
FP-215(2) Upper N.
Dup.FP-209
FP-210(2) Mid. N.
FP-211(1) Lower N.
FP-211(2) Lower N.

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TABLE 6-9
INCIDENTAL INGESTION OF SOUTH DRAINAGE SEDIMENTS - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	1.06e1	1.41e1	7.55e-6	1.24e-4	.00	.00	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	BDL	BDL	0	0	.00	.00	
COPPER	3.7e-2	1.21e1	2.35e1	8.62e-6	2.06e-4	.00	.01	
LEAD	1.4e-4	1.17e1	1.36e1	8.33e-6	1.19e-4	.06	.85	
MANGANESE	5e-1	2e2	3.21e2	1.42e-4	2.81e-3	.00	.01	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	BDL	BDL	0	0	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	3.48e0	5.69e0	2.48e-6	4.99e-5	.00	.01	
ZINC	2e-1	3e2	4.6e2	2.14e-4	4.03e-3	.00	.02	
					TOTAL	.06	.89	

ASSUMPTIONS and NOTES

Ingested metals in sediment are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in sediments. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in sediments. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of sediment samples.

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EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	6.3e-1	7.54e-1	9.62e-8	1.42e-6	1.68e-7	2.48e-6
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	BDL	BDL	0	0	0	0
NICKEL	NA	NA	BDL	BDL	0	0	0	0
						TOTAL	1.68e-7	2.48e-6

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg), no adult exposure to sediments.
Average = Assumes child ingests 200 mg per play day, 2 days per week for 26 weeks per year.
Worst = Assumes child ingests 400 mg per play day, 4 days per week, 40 weeks per year.

TABLE 6-9 (Continued)

INCIDENTAL INGESTION OF SOUTH DRAINAGE SEDIMENTS - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

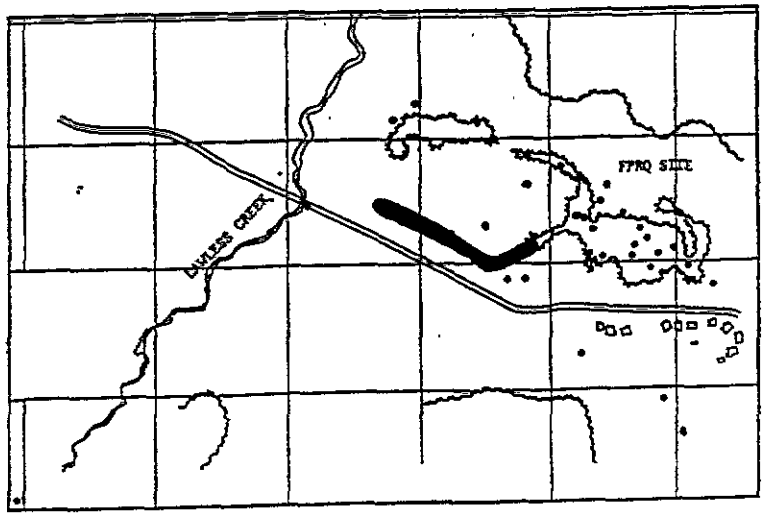
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\dfrac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in kg/kg
- I(α) = soil ingestion rate in mg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS



FP-206(1) Upper S.
FP-206(2) Upper S.
FP-207(2) Mid. S.
FP-208(1) Lower S.
FP-208(2) Lower S.
FP-219(1) Upper S.
Dup. FP-206

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TABLE 6-10
INGESTION OF RESIDENTIAL POTABLE WATER FROM LAWLESS CREEK

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	7.23e-3	1.63e-2	2.07e-4	8.15e-4	.00	.02	
BIS (2 EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	3.59e-3	6.11e-3	1.03e-4	3.06e-4	.21	.61	
COPPER	3.7e-2	8.1e-3	1.53e-2	2.31e-4	7.65e-4	.01	.02	
LEAD	1.4e-4	7.88e-4	1.28e-3	2.25e-5	6.4e-5	.16	.46	
MANGANESE	5e-1	5.52e-2	7.9e-2	1.58e-3	3.95e-3	.00	.01	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	BDL	BDL	0	0	.00	.00	
SELENIUM	3e-3	BDL	BDL	0	0	.00	.00	
VANADIUM	9e-3	BDL	BDL	0	0	.00	.00	
ZINC	2e-1	2.45e-2	2.8e-2	7e-4	1.4e-3	.00	.01	
					TOTAL	.38	1.12	

ASSUMPTIONS and NOTES

Ingested metals are 100% bioavailable; concentrations are dissolved concentrations.
Exposure averaged over a 1-year period.
Average = Daily ingestion by 70 kg adults of 2 liters of the arithmetic mean concentration of material in Lawless Creek.
Worst Case = Daily ingestion by 20 kg child of 1 liter of the lower of the 95% confidence limits of the mean or the highest observed concentration of contaminants in Lawless Creek.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/l)	Maximum (mg/l)	EXPOSURE DOSE Average Case (mg/kg/day)	Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	Reasonable Worst Case
ARSENIC	A	1.75e0	BDL	BDL	0	0	0	0
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2 EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	3.59e-3	6.11e-3	1.20e-4	2.05e-4	0	0
NICKEL	NA	NA	BDL	BDL	0	0	0	0
						TOTAL	0	0

ASSUMPTIONS and NOTES

Assumptions for exposure to non-carcinogenic compounds apply.
NA = Non-carcinogenic by the oral route.
Life-time exposure assumes 10 years of childhood (20 kg, 1 liter/d), 5 years adolescence (36 kg, 2 liter/d), and 55 years of adulthood (70 kg, 2 liter/d) with exposure averaged over 70 years.

TABLE 6-10 (Continued)

INGESTION OF POTABLE WATER FROM LAWLESS CREEK

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I}{W}$$

where:

Ca = contaminant concentration in mg/l
I = ingestion rate in liters/day
W = weight in kg

EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times Ca \times I(\alpha)}{W(\alpha)}}{LT}$$

where:

ED(α) = exposure duration
Ca = contaminant concentration in mg/l
I(α) = ingestion rate in liters/day for each ED
W(α) = weight in kg for each ED
LT = lifetime in years

SAMPLE LOCATIONS

FP-313(1) S. Road
FP-313(2) S. Road
FP-314(1) Downstream
FP-314(2) Downstream

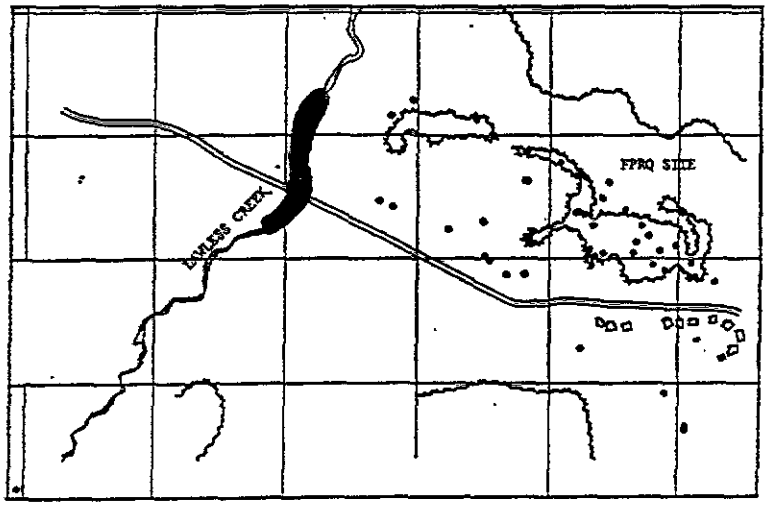

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TABLE 6-11
INCIDENTAL INGESTION OF QUARRY SOIL - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	5.16e-1	1.01e0	3.68e-7	8.85e-6	.00	.02	
BARIUM	5e-2	1.92e2	3.86e2	1.37e-4	3.38e-3	.00	.07	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	1.28e0	1.71e0	9.12e-7	1.50e-5	.00	.03	
COPPER	3.7e-2	8.14e0	9.75e0	5.80e-6	8.55e-5	.00	.00	
LEAD	1.4e-4	2.73e2	6.25e2	1.94e-4	5.48e-3	1.39	39.14	
MANGANESE	5e-1	1.28e2	1.68e2	9.12e-5	1.47e-3	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.21e1	1.67e1	8.62e-6	1.46e-4	.00	.01	
SELENIUM	3e-3	1.69e-1	2.23e-1	1.20e-7	1.96e-6	.00	.00	
VANADIUM	9e-3	2.02e1	2.49e1	1.44e-5	2.18e-4	.00	.02	
ZINC	2e-1	1.04e2	1.7e2	7.41e-5	1.49e-3	.00	.01	
					TOTAL	1.40	39.30	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in quarry soil. Ingested concentrations are arithmetic mean of quarry soil samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in quarry soil. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	5.19e0	8.44e0	8.37e-7	1.81e-5	1.47e-6	3.17e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	1.28e0	1.71e0	2.07e-7	3.67e-6	0	0
NICKEL	NA	NA	1.21e1	1.67e1	1.95e-6	3.59e-5	0	0
						TOTAL	1.47e-6	3.17e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-11 (Continued)

INCIDENTAL INGESTION OF QUARRY SOIL - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/kg
- I(α) = soil ingestion rate in kg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- A = bioavailability (fraction)
- LT = lifetime in years

SAMPLE LOCATIONS

FP-103 Grid
FP-104 Grid
FP-105 Grid
FP-106 Grid
FP-107 Grid
FP-108 Grid
FP-109 Grid

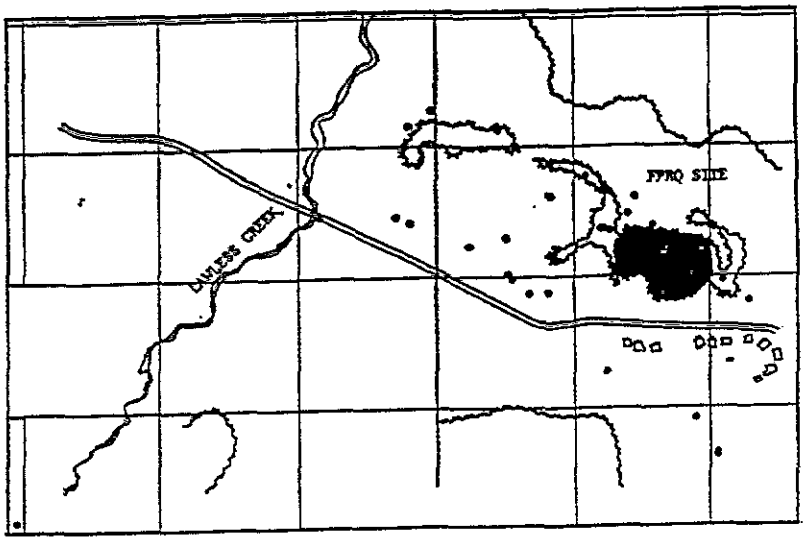

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TABLE 6-12
INCIDENTAL INGESTION OF QUARRY SOIL - ADULTS

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	5.16e-1	1.01e0	3.15e-8	6.32e-7	.00	.00	
BARIUM	5e-2	1.92e2	3.86e2	1.17e-5	2.42e-4	.00	.00	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	1.28e0	1.71e0	7.82e-8	1.07e-6	.00	.00	
COPPER	3.7e-2	8.14e0	9.75e0	4.97e-7	6.11e-6	.00	.00	
LEAD	1.4e-4	2.73e2	6.25e2	1.67e-5	3.91e-4	.12	2.80	
MANGANESE	5e-1	1.28e2	1.68e2	7.82e-6	1.05e-4	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.21e1	1.67e1	7.39e-7	1.05e-5	.00	.00	
SELENIUM	3e-3	1.69e-1	2.23e-1	1.03e-8	1.40e-7	.00	.00	
VANADIUM	9e-3	2.02e1	2.49e1	1.23e-6	1.56e-5	.00	.00	
ZINC	2e-1	1.04e2	1.7e2	6.35e-6	1.06e-4	.00	.00	
					TOTAL	.12	2.81	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 70 kg adult of 60 mg of soil per activity day, adult gardens 2 days per week for 26 weeks per year in quarry soil. Ingested concentrations are arithmetic mean of quarry soil samples.
Worst = Ingestion by a 70 kg adult of 100 mg of soil per activity day, adult gardens 4 days per week for 40 weeks per year in quarry soil. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	5.19e0	8.44e0	8.37e-7	1.81e-5	1.47e-6	3.17e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	1.28e0	1.71e0	2.07e-7	3.67e-6	0	0
NICKEL	NA	NA	1.21e1	1.67e1	1.95e-6	3.59e-5	0	0
						TOTAL	1.47e-6	3.17e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-12 (Continued)

INCIDENTAL INGESTION OF QUARRY SOIL - ADULTS

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

Ca	=	contaminant concentration in mg/kg
I	=	soil ingestion rate in kg/day
F	=	daily frequency
W	=	weight in kg
A	=	bioavailability (fraction)

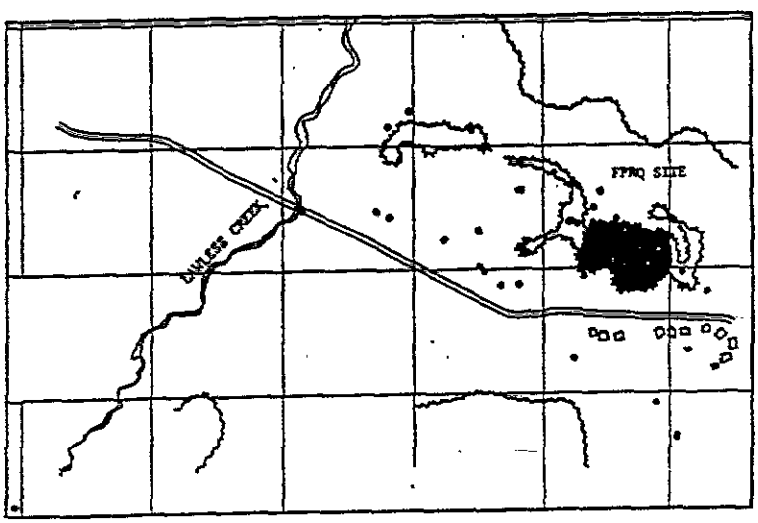
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\dfrac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

ED(α)	=	exposure duration
P	=	proportion of year exposed
Ca	=	contaminant concentration in mg/kg
I(α)	=	soil ingestion rate in kg/day for each ED
F	=	daily frequency
W(α)	=	weight in kg for each ED
A	=	bioavailability (fraction)
LT	=	lifetime in years

SAMPLE LOCATIONS



FP-103 Grid
FP-104 Grid
FP-105 Grid
FP-106 Grid
FP-107 Grid
FP-108 Grid
FP-109 Grid

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TABLE 6-13
INCIDENTAL INGESTION OF NORTH DRAINAGE SOIL - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	7.11e-1	8.54e-1	5.06e-7	7.49e-6	.00	.02	
BARIUM	5e-2	1.48e3	1.82e3	1.05e-3	1.60e-2	.02	.32	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	4.71e0	6.88e0	3.36e-6	6.03e-5	.01	.12	
COPPER	3.7e-2	1.16e1	9.25e0	8.26e-6	8.11e-5	.00	.00	
LEAD	1.4e-4	1.41e2	1.74e2	1.00e-4	1.53e-3	.72	10.90	
MANGANESE	5e-1	1.542e3	1.66e3	1.10e-3	1.46e-2	.00	.03	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	6.89e0	6.9e0	4.91e-6	6.05e-5	.00	.00	
SELENIUM	3e-3	2.68e-1	2.55e-1	1.91e-7	2.24e-6	.00	.00	
VANADIUM	9e-3	1.15e1	1.12e1	8.19e-6	9.82e-5	.00	.01	
ZINC	2e-1	5.78e2	6.18e2	4.12e-4	5.42e-3	.00	.03	
					TOTAL	.75	11.43	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in soil at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in soil at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	2.04e1	2.56e1	3.29e-6	5.50e-5	5.76e-6	9.62e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	4.71e0	6.88e0	7.60e-7	1.48e-5	0	0
NICKEL	NA	NA	6.89e0	6.9e0	1.11e-6	1.48e-5	0	0
						TOTAL	5.76e-6	9.62e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-13 (Continued)

INCIDENTAL INGESTION OF NORTH DRAINAGE SOIL - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/kg
- I(α) = soil ingestion rate in kg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS

FP-121 Grid
FP-122 Grid
FP-123 Grid
FP-124 Grid

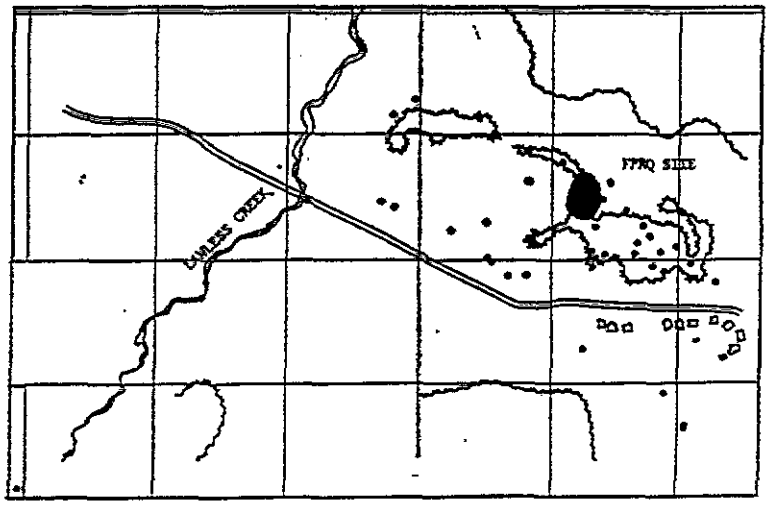

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TABLE 6-14
INCIDENTAL INGESTION OF NORTH DRAINAGE SOIL - ADULTS

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	7.11e-1	8.54e-1	4.34e-8	5.35e-7	.00	.00	
BARIUM	5e-2	1.48e3	1.82e3	9.04e-5	1.14e-3	.00	.02	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	4.71e0	6.89e0	2.88e-7	4.31e-6	.00	.01	
COPPER	3.7e-2	1.16e1	9.25e0	7.08e-7	5.79e-6	.00	.00	
LEAD	1.4e-4	1.41e2	1.74e2	8.61e-6	1.09e-4	.06	.78	
MANGANESE	5e-1	1.542e3	1.66e3	9.41e-5	1.04e-3	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	6.89e0	6.9e0	4.21e-7	4.32e-6	.00	.00	
SELENIUM	3e-3	2.68e-1	2.55e-1	1.64e-8	1.60e-7	.00	.00	
VANADIUM	9e-3	1.5e0	1.12e1	9.16e-8	7.01e-6	.00	.00	
ZINC	2e-1	5.78e2	6.18e2	3.53e-5	3.87e-4	.00	.00	
					TOTAL	.06	.82	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 70 kg adult of 60 mg of soil per activity day, adult gardens 2 days per week for 26 weeks per year in soil at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 70 kg adult of 100 mg of soil per activity day, adult gardens 4 days per week for 40 weeks per year in soil at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	2.04e1	2.56e1	3.29e-6	5.50e-5	5.76e-6	9.62e-5
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	4.71e0	6.89e0	7.60e-7	1.48e-5	0	0
NICKEL	NA	NA	6.89e0	6.9e0	1.11e-6	1.43e-5	0	0
						TOTAL	5.76e-6	9.62e-5

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-14 (Continued)

INCIDENTAL INGESTION OF NORTH DRAINAGE SOIL - ADULTS

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

Ca	=	contaminant concentration in mg/kg
I	=	soil ingestion rate in kg/day
F	=	daily frequency
W	=	weight in kg
A	=	bioavailability (fraction)

EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\dfrac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

ED(α)	=	exposure duration
P	=	proportion of year exposed
Ca	=	contaminant concentration in mg/kg
I(α)	=	soil ingestion rate in kg/day for each ED
F	=	daily frequency
W(α)	=	weight in kg for each ED
LT	=	lifetime in years
A	=	bioavailability (fraction)

SAMPLE LOCATIONS

FP-121 Grid
FP-122 Grid
FP-123 Grid
FP-124 Grid

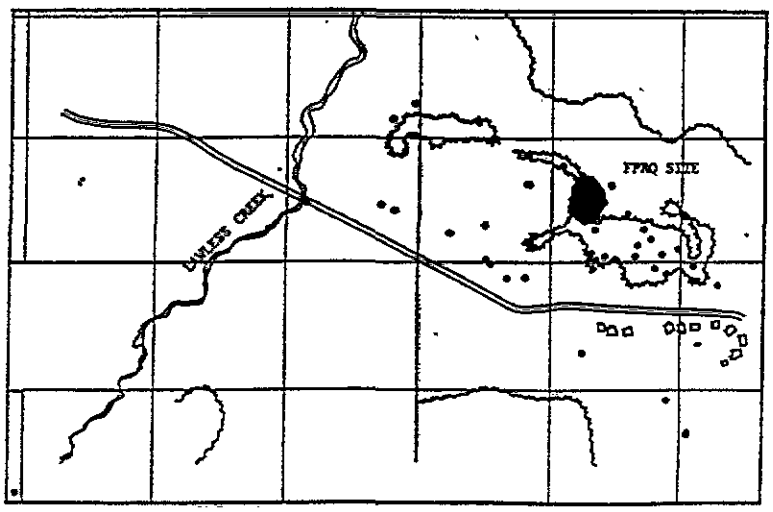


TABLE 6-15
INCIDENTAL INGESTION OF SOUTH DRAINAGE SOIL (DISTURBED AREA) - CHILDREN

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	5.34e1	8.72e1	3.80e-5	7.64e-4	.00	.02	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	1.18e0	1.7e0	8.41e-7	1.49e-5	.00	.03	
COPPER	3.7e-2	8.75e0	1.26e1	6.23e-6	1.10e-4	.00	.00	
LEAD	1.4e-4	9.4e0	1.07e1	6.70e-6	9.38e-5	.05	.67	
MANGANESE	5e-1	3.78e2	8.7e2	2.69e-4	7.63e-3	.00	.02	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.05e1	1.79e1	7.48e-6	1.57e-4	.00	.01	
SELENIUM	3e-3	1.63e-1	2.36e-1	1.16e-7	2.07e-6	.00	.00	
VANADIUM	9e-3	1.49e1	2.26e1	1.06e-5	1.98e-4	.00	.02	
ZINC	2e-1	2.8e1	3.47e1	1.99e-5	3.04e-4	.00	.00	
					TOTAL	.05	.77	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 20 kg child of 200 mg of soil per play day, child plays 2 days per week for 26 weeks per year in soil at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 20 kg child of 400 mg of soil per play day, child plays 4 days per week for 40 weeks per year in soil at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	1.65e0	2e0	2.66e-7	4.29e-6	4.66e-7	7.51e-6
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	1.18e0	1.7e0	1.90e-7	3.65e-6	0	0
NICKEL	NA	NA	1.05e1	1.79e1	1.69e-6	3.84e-5	0	0
						TOTAL	4.66e-7	7.51e-6

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-15 (Continued)

INCIDENTAL INGESTION OF SOUTH DRAINAGE (DISTURBED AREA) SOIL - CHILDREN

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

- Ca = contaminant concentration in mg/kg
- I = soil ingestion rate in kg/day
- F = daily frequency
- W = weight in kg
- A = bioavailability (fraction)

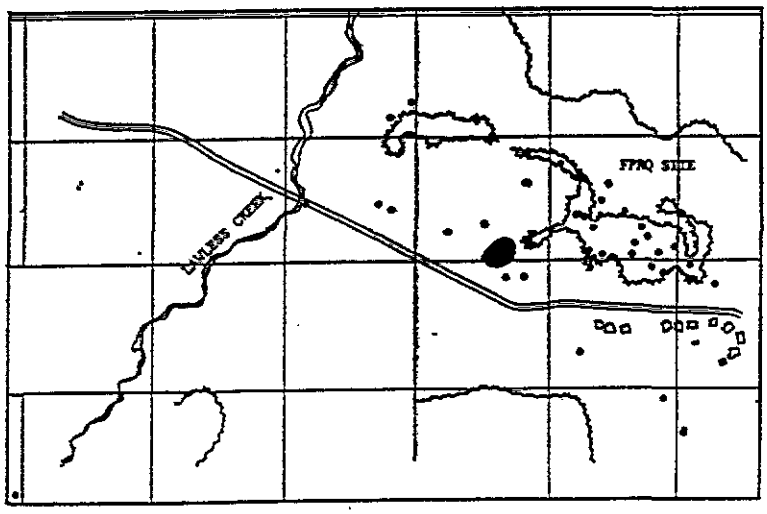
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{\frac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

- ED(α) = exposure duration
- P = proportion of year exposed
- Ca = contaminant concentration in mg/kg
- I(α) = soil ingestion rate in kg/day for each ED
- F = daily frequency
- W(α) = weight in kg for each ED
- LT = lifetime in years
- A = bioavailability (fraction)

SAMPLE LOCATIONS



FP-114 Disturbed Area
FP-115 Disturbed Area
FP-116 Disturbed Area
FP-125 Disturbed Area
Dup FP-115

(aexp646.cal)
04-Dec-90

TABLE 6-16
INCIDENTAL INGESTION OF SOUTH DRAINAGE SOIL (DISTURBED AREA) - ADULTS

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	HAZARD INDEX Average Case	HAZARD INDEX Reasonable Worst Case	END POINT
ANTIMONY	4e-4	BDL	BDL	0	0	.00	.00	
BARIUM	5e-2	5.34e1	8.72e1	3.26e-6	5.46e-5	.00	.00	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM	5e-4	1.18e0	1.7e0	7.20e-8	1.06e-6	.00	.00	
COPPER	3.7e-2	8.75e0	1.26e1	5.34e-7	7.89e-6	.00	.00	
LEAD	1.4e-4	9.4e0	1.07e1	5.74e-7	6.70e-6	.00	.05	
MANGANESE	5e-1	3.78e2	8.7e2	2.31e-5	5.45e-4	.00	.00	
MERCURY	2e-3	BDL	BDL	0	0	.00	.00	
NICKEL	2e-2	1.05e1	1.79e1	6.41e-7	1.12e-5	.00	.00	
SELENIUM	3e-3	1.63e-1	2.36e-1	9.95e-9	1.48e-7	.00	.00	
VANADIUM	9e-3	1.49e1	2.26e1	9.10e-7	1.42e-5	.00	.00	
ZINC	2e-1	2.8e1	3.47e1	1.71e-6	2.17e-5	.00	.00	
					TOTAL	.00	.05	

ASSUMPTIONS and NOTES

Ingested metals in soil matrix are 50 (average case) to 100% (worst case) bioavailable; exposures averaged over 1 year.
Average = Ingestion by a 70 kg adult of 60 mg of soil per activity day, adult gardens 2 days per week for 26 weeks per year in soil at the site. Ingested concentrations are arithmetic mean of site samples.
Worst = Ingestion by a 70 kg adult of 100 mg of soil per activity day, adult gardens 4 days per week for 40 weeks per year in soil at the site. Ingested concentrations are the lower of the 95% confidence limits of the mean or the highest observed concentration of site samples.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	CONCENTRATION Maximum (mg/kg)	EXPOSURE DOSE Average Case (mg/kg/day)	EXPOSURE DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME CANCER RISK Average Case	LIFETIME CANCER RISK Reasonable Worst Case
ARSENIC	A	1.75e0	1.65e0	2e0	2.66e-7	4.29e-6	4.66e-7	7.51e-6
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	1.4e-2	BDL	BDL	0	0	0	0
CADMIUM	NA	NA	1.18e0	1.7e0	1.90e-7	3.65e-6	0	0
NICKEL	NA	NA	1.05e1	1.79e1	1.69e-6	3.84e-5	0	0
						TOTAL	4.66e-7	7.51e-6

ASSUMPTIONS and NOTES

NA = Non-carcinogenic by the oral route. Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes 15 years of childhood (20 kg) and 10 years (average) to 30 years (worst case) of adulthood (70 kg) with exposure averaged over 70 years.
Average = Assumes child ingests 200 mg per play day for 15 years, adults ingest 60 mg per activity day for 10 years. Outdoor activity occurs 2 days per week for 26 weeks of the year.
Worst = Assumes child ingests 400 mg per play day for 15 years, adults ingest 100 mg per activity day for 30 years. Outdoor activity occurs 4 days a week for 40 weeks per year.

TABLE 6-16 (Continued)

INCIDENTAL INGESTION OF SOUTH DRAINAGE (DISTURBED AREA) SOIL - ADULTS

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times I \times F \times A}{W}$$

where:

Ca = contaminant concentration in mg/kg
I = soil ingestion rate in kg/day
F = daily frequency
W = weight in kg
A = bioavailability (fraction)

EXPOSURE TO CARCINOGENS

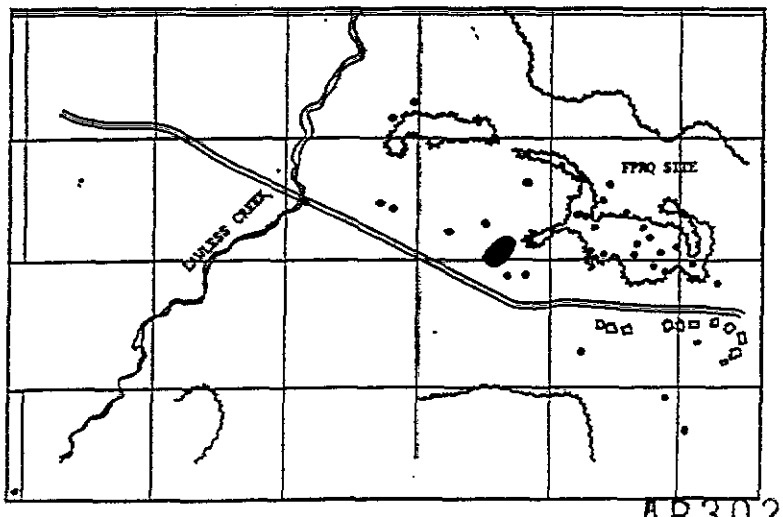
$$\text{Exposure Dose} = \frac{\dfrac{ED(\alpha) \times P \times Ca \times I(\alpha) \times F \times A}{W(\alpha)}}{LT}$$

where:

ED(α) = exposure duration
P = proportion of year exposed
Ca = contaminant concentration in mg/kg
I(α) = soil ingestion rate in kg/day for each ED
F = daily frequency
W(α) = weight in kg for each ED
LT = lifetime in years
A = bioavailability (fraction)

SAMPLE LOCATIONS

FP-114 Disturbed Area
FP-115 Disturbed Area
FP-116 Disturbed Area
FP-125 Disturbed Area
Dup FP-115



(aexp651.cal)
04-Dec-90

TABLE 6-17
INHALATION OF AIR-BORNE CONTAMINANTS IN QUARRY

EXPOSURE TO NON-CARCINOGENS

Non-Carcinogenic Contaminant of Concern	RfD or Other Standard (mg/kg/day)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	HAZARD Average Case	INDEX Reasonable Worst Case	END POINT
ANTIMONY*	4e-4	5.16e-1	1.01e0	2.3e-15	1.5e-11	.00	.00	
BARIUM	1.4e-4	1.92e2	3.86e2	8.4e-13	5.88e-9	.00	.00	
BIS (2EH) PHTHALATE	2e-2	BDL	BDL	0	0	.00	.00	
CADMIUM*	5e-4	1.28e0	1.71e0	5.6e-15	2.6e-11	.00	.00	
COPPER	1e-2	8.14e0	9.75e0	3.6e-14	1.5e-10	.00	.00	
LEAD	4.3e-4	2.73e2	6.25e2	1.2e-12	9.51e-9	.00	.00	
MANGANESE	5e-1	1.28e2	1.68e2	5.6e-13	2.56e-9	.00	.00	
MERCURY	5.1e-5	BDL	BDL	0	0	.00	.00	
NICKEL*	2e-2	1.21e1	1.67e1	5.3e-14	2.5e-10	.00	.00	
SELENIUM	1e-3	1.69e-1	2.23e-1	7.4e-16	3.4e-12	.00	.00	
VANADIUM*	9e-3	2.02e1	2.49e1	8.9e-14	3.8e-10	.00	.00	
ZINC	1e-2	1.04e2	1.7e2	4.6e-13	2.59e-9	.00	.00	
					TOTAL	.00	.00	

ASSUMPTIONS and NOTES

For constituents marked with "*", oral chronic health standards are used to account for swallowing particulates, since there are no inhalation standards for chronic inhalation.
Inhaled metals in soil matrix are 100% bioavailable.
Average = Inhalation by a 70 kg adult of 1.4 cubic meters of air per hour, containing 1.37x10E-6 ug per cubic meter of dust for 16 hours per day.
Worst = Inhalation by a 70 kg adult of 3.0 cubic meters of air per hour, containing 2.22x10-3 ug per cubic meter of dust for 16 hours per day.

* *

EXPOSURE TO CARCINOGENS

Carcinogenic Contaminant of Concern	Cancer Weight of Evidence	Cancer Potency Factor (mg/kg/d)	CONCENTRATION Average (mg/kg)	Maximum (mg/kg)	EXPOSURE Average Case (mg/kg/day)	DOSE Reasonable Worst Case (mg/kg/day)	LIFETIME Average Case	CANCER RISK Reasonable Worst Case
ARSENIC	A	5e1	5.19e0	8.44e0	1.1e-14	1.3e-10	5.7e-13	6.42e-9
BENZENE	A	2.9e-2	BDL	BDL	0	0	0	0
BIS (2EH) PHTHALATE	B2	NA	BDL	BDL	0	0	0	0
CADMIUM	A	6.1e0	1.28e0	1.71e0	2.8e-15	2.6e-11	1.7e-14	1.6e-10
NICKEL	A	1.19e0	1.21e1	1.67e1	2.7e-14	2.5e-10	3.2e-14	3.0e-10
						TOTAL	6.2e-13	6.89e-9

ASSUMPTIONS and NOTES

Assumptions for exposure to non-carcinogenic compounds apply.
Life-time exposure assumes a 70 kg person with exposure averaged over 70 years.
Average = Assumes lifetime inhalation for 204,400 hours (8 hours per day, 70 years).
Worst = Assumes lifetime inhalation for 408,800 hours (16 hours per day, 70 years).

TABLE 6-17 (Continued)

INHALATION OF AIR-BORNE CONTAMINANTS ON SITE

FORMULAE

EXPOSURE TO NON-CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times Cb \times I \times D \times CF}{W}$$

where:

Ca	=	contaminant concentration in mg/kg
Cb	=	dust concentration in mg/cubic meter
I	=	inhalation rate in cubic meters/day
D	=	daily duration in hours
CF	=	correction factor to convert to mg
W	=	weight in kg

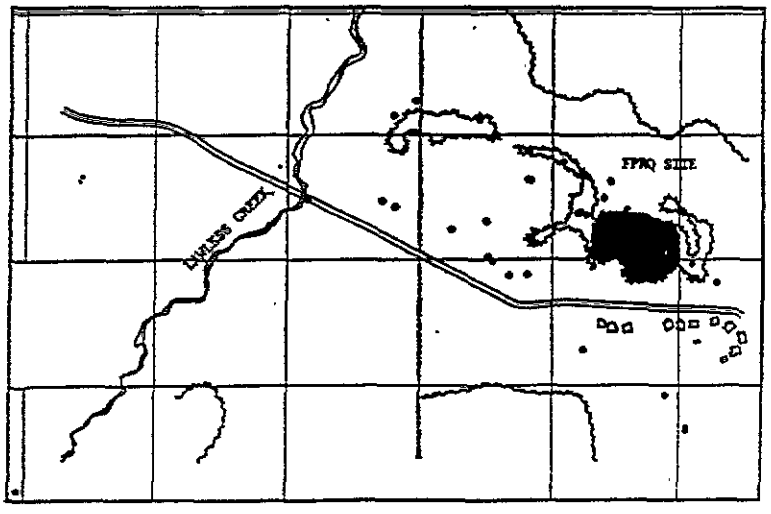
EXPOSURE TO CARCINOGENS

$$\text{Exposure Dose} = \frac{Ca \times Cb \times I \times ED \times CF}{W \times LT \times D}$$

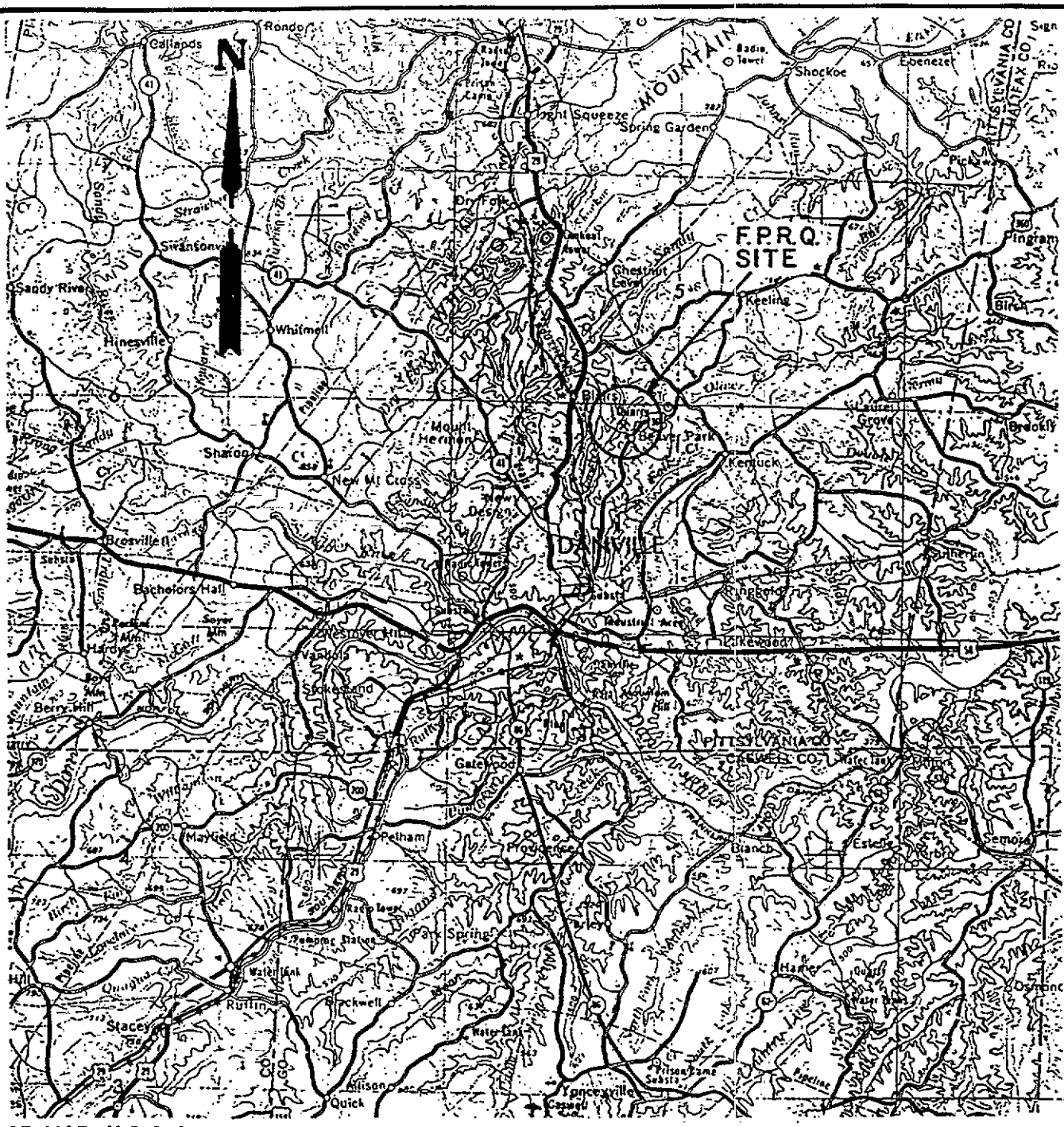
where:

Ca	=	contaminant concentration in mg/kg
Cb	=	dust concentration in mg/cubic meter
I	=	inhalation rate in cubic meters/day
ED	=	exposure duration in total lifetime hours
CF	=	correction factor to convert to mg
W	=	weight in kg
LT	=	lifetime in years
D	=	365 days per year

SAMPLE LOCATIONS



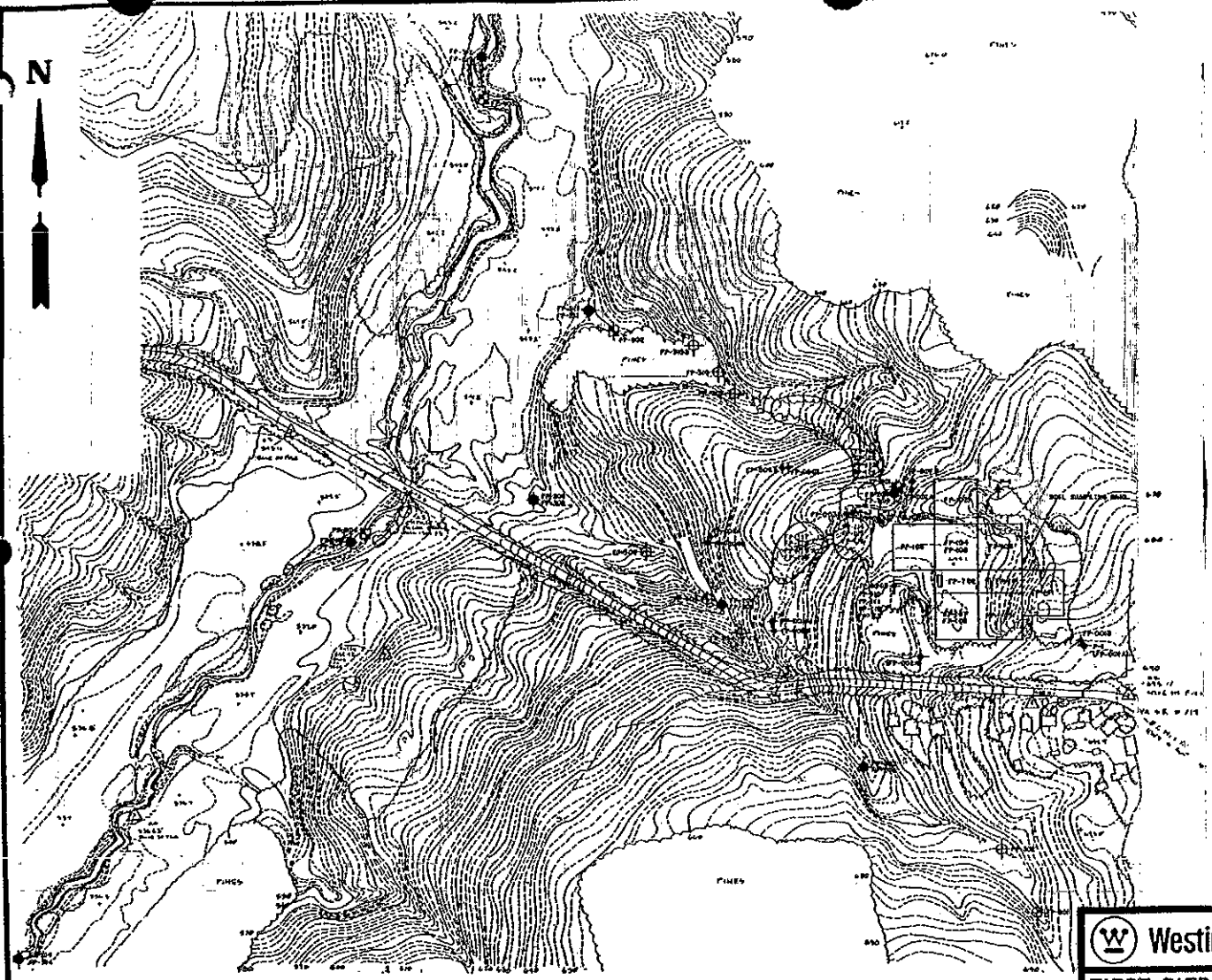
FP-103 Grid
FP-104 Grid
FP-105 Grid
FP-106 Grid
FP-107 Grid
FP-108 Grid
FP-109 Grid



BASE MAP: U.S.G.S.
GREENSBORO, N.C. 1°x2°

FPRQ VICINITY MAP

PROJECT FIRST PIEDMONT ROCK QUARRY DANVILLE, VA.	Westinghouse AR302075	SCALE: 1:250,000 JOB NO: 4112-88-907B FIG. NO: 1

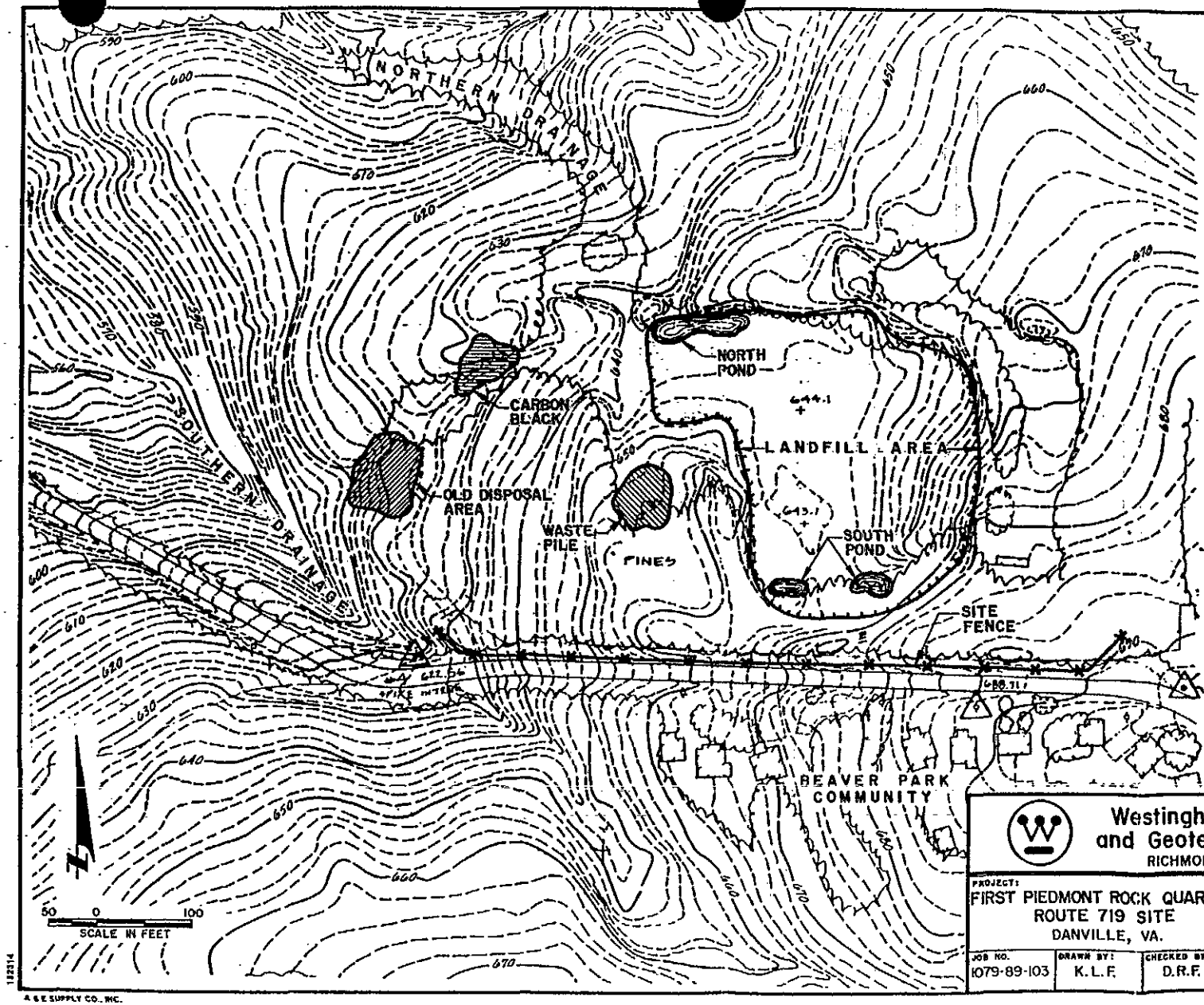


PHASE II RI MONITORING LOCATIONS

Westinghouse Environmental Services

FIRST PIEDMONT ROCK QUARRY ROUTE 719 SITE DANVILLE, VA.	DRWN. BY: MCJ	CHKD. BY:
	JOB NO.: 4112-88-90 7B	DATE: 9/89
	SCALE: 1"= 250'	FIGURE: 2

AR302076



NOTE:
TOPOGRAPHY PRODUCED FROM AERIAL PHOTOGRAPHY JAN. 24, 1989.
CONTOUR INTERVAL = 2 FT.

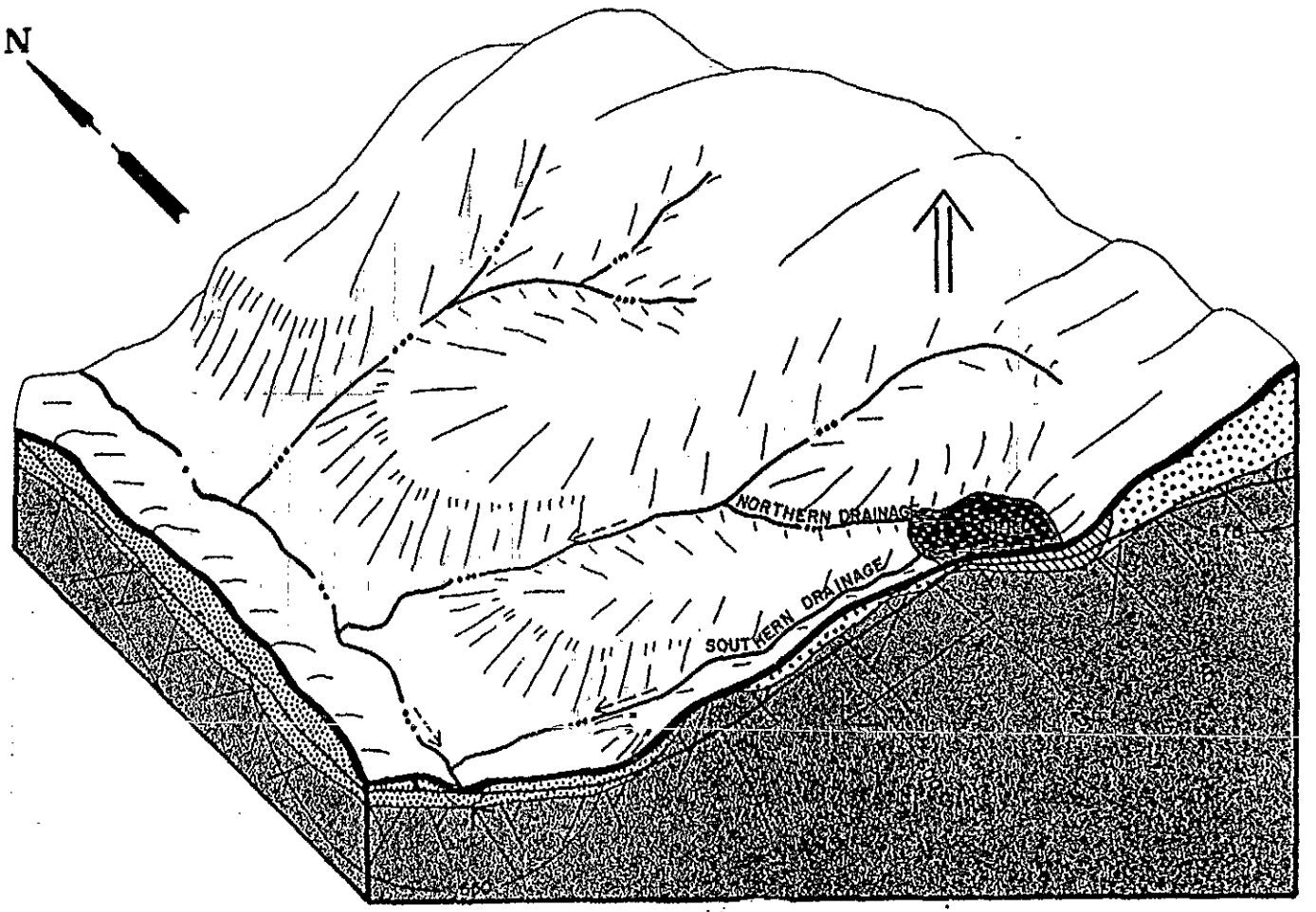
Westinghouse Environmental and Geotechnical Services, Inc.
RICHMOND, VIRGINIA

PROJECT:			TITLE:		
FIRST PIEDMONT ROCK QUARRY ROUTE 719 SITE DANVILLE, VA.			SITE LAYOUT AND TOPOGRAPHY		
JOB NO.	DRAWN BY:	CHECKED BY:	SCALE:	DATE:	FIGURE NO.
1079-89-103	K.L.F.	D.R.F.	1" = 100'	11/11/89	2-A

122314

A & E SUPPLY CO., INC.

AR302077



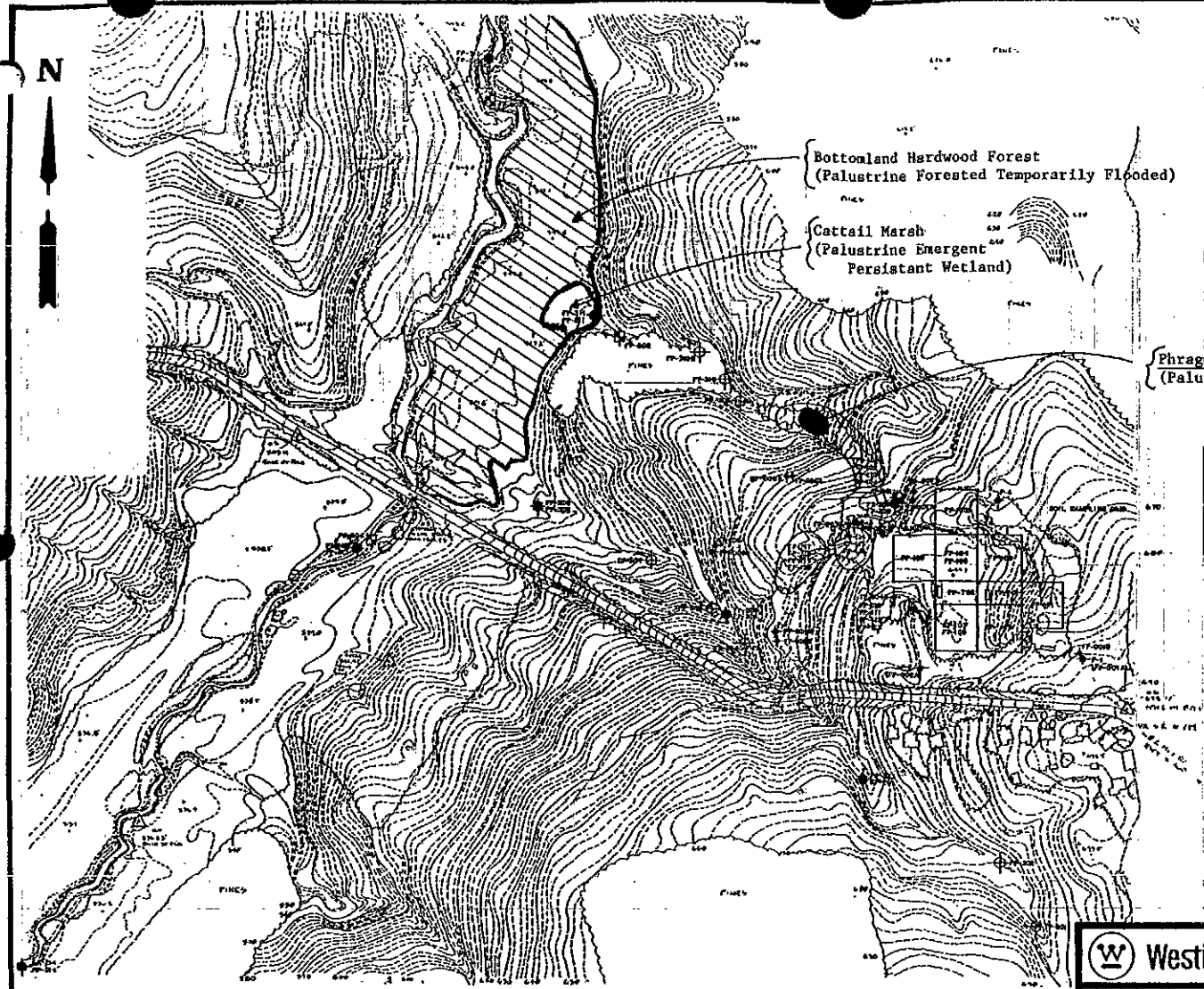
EXPLANATION:

- LANDFILL MATERIAL
- ALLUVIUM
- RESIDUUM SAPROLITE
- CRYSTALLINE BEDROCK
- FRACTURES
- WATER TABLE
- 575 POTENTIOMETRIC CONTOUR (Ft. MSL)
- GROUND WATER FLOW DIRECTION
- SURFACE WATER DRAINAGE
- DIRECTION OF SURFACE WATER FLOW
- PREVAILING WIND DIRECTION

CONCEPTUAL TRANSPORT PATHWAY MODEL

Westinghouse Environmental Services

FIRST PIEDMONT ROCK QUARRY DANVILLE, VA.	DRWN. BY: MCJ/SWC	CHKD. BY:
	JOB NO.: 4112-68-90 7B	DATE: 1/89
	SCALE: N.T.S.	FIGURE: 3



WETLANDS DELINEATION

Westinghouse Environmental Services

FIRST PIEDMONT ROCK QUARRY ROUTE 719 SITE DANVILLE, VA.	DRWN. BY: MCJ	CHKD. BY:
	JOB NO.: 4112-88-904 0	DATE: 9/89
	SCALE: 1" = 250'	FIGURE: 4

APPENDIX A

Indicator Chemical Selection Tables

TABLE A-1

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-970B

Ground Water

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
		µg/L		
Aluminum	--	<51.9-16,900	5,181	--
Antimony	--	<2-66	34.2	Only 2 above det. (66 & 22)
Arsenic	--	<2-197	197	All <2 except for 197 sample
Barium	--	<2.1-34,700	1292	Only 1 sample > 350
Beryllium	--	<1.8-22	22	Only 1 sample above detection
Cadmium	--	<4.7-10	7.3	Only 3 samples above detection
Calcium	--	594-55,000	22,074	--
Chromium	--	<5.4-141	41.6	Only 1 sample >32 (i.e.,141)
Cobalt	--	<26.2-35.9	31.9	Only 2 samples were detectable
Copper	--	<6.8-57.9	20.3	--
Iron	--	<7.9-50,900	5288	--
Lead	--	<1.3-1890	179	Only 1 sample >28.8
Magnesium	--	353-21,600	7253	--
Manganese	--	7.6-6350	681	--
Mercury	--	<0.2-0.57	0.57	Only 1 sample detectable
Nickel	--	<24.7-193	123	Only 2 detectable
Potassium	--	<790-13,100	3556	--
Selenium	--	<1.4	0	--
Silver	--	<9.3	0	--
Sodium	--	8,010-44,000	18,665	--
Thallium	--	<2	0	--
Vanadium	--	<18.3-50.1	50.1	Only 1 sample detectable
Zinc	--	<10.7-3,820	341	--
Cyanide	--	<10	0	--
Sulfate	--	3,400-30,600	13,700	--

TABLE A-2

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-907B

Surface Water

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
		µg/L		
Aluminum	--	<51.9-5310	1,464	Only 2 samples >357
Antimony	--	<2-2.6	2.6	Only 1 sample-detectable
Arsenic	--	<2-13.7	13.7	Only 1 sample-detectable
Barium	--	<2.1-5600	732	Only 1 sample >109
Beryllium	--	<1.8-6.4	6.35	Only 2 samples-detectable
Cadmium	--	<4.7-105	52.9	Only 4 samples-detectable
Calcium	--	2,160-23,600	10,190	--
Chromium	--	<5.4	0	None detectable
Cobalt	--	<26.2	0	None detectable
Copper	--	<6.8-27.3	25.9	Only 2 detectable
Iron	--	28.7-59,800	6,948	Only 1 sample >647
Lead	--	<1.3-5.8	3.0	--
Magnesium	--	935-8740	3533	--
Manganese	--	50-3030	977.7	--
Mercury	--	<0.2	0	None detectable
Nickel	--	<24.7-42.2	36.5	--
Potassium	--	1,200-24,200	4,932	--
Selenium	--	<1.4	0	None detectable
Silver	--	<9.3	0	None detectable
Sodium	--	4,920-11,600	10,586	--
Thallium	--	<2	0	None detectable
Vanadium	--	<18.3	0	None detectable
Zinc	--	<10.7-111,000	27,808	Only 2 samples >4320
Cyanide	--	<10	0	None detectable
Sulfate	--	4.03-248	60.9	--

TABLE A-3

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-907B

Soil

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
		µg/L		
Aluminum	--	1,200-23,400	9,936	--
Antimony	--	<0.31-3.1	1.24	--
Arsenic	--	0.36-135	8.5	--
Barium	--	7.7-9,900	652	--
Beryllium	--	<0.31-0.71	0.57	--
Cadmium	--	<0.73-34.5	3.3	--
Calcium	--	55.9-7,060	739	--
Chromium	--	<2.2-110	19.9	--
Cobalt	--	<4.1-26.8	12.7	Only 4 samples detectable
Copper	--	<1.3-42.8	9.8	--
Iron	--	1,300-530,000	23,619	--
Lead	--	3.15-6,920	300	--
Magnesium	--	199-1,400	537	--
Manganese	--	16.3-1,920	295	--
Mercury	--	<0.1	0	None detectable
Nickel	--	<3.9-20.5	9.82	--
Potassium	--	10.6-1770	634	--
Selenium	--	<0.23-0.32	0.29	--
Silver	--	<1.5	0	None detectable
Sodium	--	25-625	98	--
Thallium	--	<0.31-0.46	0.37	Only 5 samples detectable
Vanadium	--	<2.8-27.7	12.7	--
Zinc	--	14.6-1,510	201	--
Cyanide	--	<1.1	0	None detectable
Sulfate	--			

TABLE A-4

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-907B

Ground Water

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
			µg/L	
Acetone	2.2	<3-16	6.6	--
Benzene	83	--	0	None detectable
Benzo(a) anthracene	1.38E+6	--	0	None detectable
Benzo(b) fluoranthene	550,000	--	0	None detectable
Benzo(k) fluoranthene	550,000	--	0	None detectable
Bis(2-ethyl hexyl)phthalate	--	--	0	None detectable
2-Butanone	--	--	0	None detectable
Chlorobenzene	330	--	0	None detectable
Chloroform	31	--	0	None detectable
Chrysene	2E+5	--	0	None detectable
1,2-Dichloroethene	59trans 49 cis	--	0	None detectable
2,6-Dinitrotoluene	92	--	0	None detectable
Ethylbenzene	1100	--	0	None detectable
Fluoranthene	38000	--	0	None detectable
2-Hexanone	--	--	0	None detectable
Methylene Chloride	8.8	--	3	1 sample analyte detected (?)
4-Methyl-2-pentanone	--	--	0	None detectable
Napthalene	--	--	0	None detectable
N-Nitrosodiphenyl amine	--	--	0	None detectable
Phenanthrene	14000	--	0	None detectable
Phenol	14.2	--	0	None detectable
Pyrene	38000	--	0	None detectable
Toluene	300	--	0	None detectable
Xylene	--	--	2	Only 1 detectable

TABLE A-5

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-907B

Surface Water

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
		µg/L		
Acetone	2.2	<7-18	12	--
Benzene	83	4	4	Only 1 detectable
Benzo(a) anthracene	1.38E+6	--	0	None detectable
Benzo(b) fluoranthene	550,000	--	0	None detectable
Benzo(k) fluoranthene	550,000	--	0	None detectable
Bis(2-ethyl hexyl)phthalate	--	5	5	Only 1 detectable
2-Butanone	--	--	0	None detectable
Chlorobenzene	330	3	3	Only 1 detectable
Chloroform	31	--	0	None detectable
Chrysene	2E+5	--	0	None detectable
1,2-Dichloroethene	59trans 49 cis	--	0	None detectable
2,6-Dinitrotoluene	92	--	0	None detectable
Ethylbenzene	1100	--	0	None detectable
Fluoranthene	38000	--	0	None detectable
2-Hexanone	--	--	0	None detectable
Methylene Chloride	8.8	3	3	Only 1 detectable
4-Methyl-2-pentanone	--	--	0	None detectable
Naphthalene	--	--	0	None detectable
N-Nitrosodi-phenylamine	--	--	0	None detectable
Phenanthrene	14000	--	0	None detectable
Phenol	14.2	--	0	None detectable
Pyrene	38000	--	0	None detectable
Toluene	300	--	0	None detectable
Xylene	--	--	0	None detectable

TABLE A-6

Scoring for Indicator Chemical Selection
Westinghouse Project No. 4112-88-907B

Soil

Parameter	Koc(ml/g)	Range	Rep. Conc.	Comments
		- - - - µg/L - - - - - -		
Acetone	2.2	<4-42	13	--
Benzene	83	<4-13	8.5	Only 2 samples detectable
Benzo(a) anthracene	1.38E+6	<160-290	225	Only 2 samples detectable
Benzo(b) fluoranthene		220	220	Only 1 sample detectable
Benzo(k) fluoranthene	5.5E+5	160	160	Only 1 sample detectable
Bis(2-ethyl hexyl)phthalate	--	<210-5200	1,013	--
2-Butanone	--	10	10	Only 1 detectable
Chlorobenzene	330	--	0	None detectable
Chloroform	31	<3-6	4.5	Only 2 detectable
Chrysene	2E+5	310	310	Only 1 detectable
1,2-Dichloroethene	59trans 49cis	4	4	Only 1 detectable
2,6-Dinitrotoluene	92	1400	1400	Only 1 detectable
Ethylbenzene	1100	19	19	Only 1 detectable
Fluoranthene	38000	<620-1400	1010	Only 2 detectable
2-Hexanone	--	8	8	Only 1 detectable
Methylene Chloride	8.8	3-24	8.2	--
4-Methyl-2-pentanone	--	<9-86	47.5	Only 2 detectable
Naphthalene	--	220	220	Only 1 detectable
N-Nitrosodi-phenylamine	--	<1200-9100	7975	Only 4 detectable
Phenanthrene	14000	<680-1400	1040	Only 2 detectable
Phenol	14.2	<390-610	500	Only 2 detectable
Pyrene	38000	560	560	Only 2 detectable
Toluene	300	<4-11	8	Only 2 detectable
Xylene	--	<15-100	58	Only 2 detectable

TABLE A-7

Scoring for Indicator Chemicals-Toxicity Information
Westinghouse Project No. 4112-88-907B

Chemical	Tox Class	Rating Val /EPA Cat.	w_T	s_T	a_T
Aluminum	--	--	--	--	--
Antimony	NC	10(oral) 8(inhal)	4.4	2.2E-4	229
Arsenic	PC	A	4.1	2.0E-4	41
	NC	9	18	9.0E-4	180
Barium	NC	10	4.1	2.0E-4	41
Beryllium	NC	8(inhal)	--	--	1.5E+4
	PC	B1	--	--	22.8
Cadmium	NC	10(oral)/ 8(inhal.)	4.5	2.2E-4	359
	PC	B1	--	--	16.5
Calcium	--	--	--	--	--
Chromium VI	NC	8(inhal)	--	--	25
	PC	A	--	--	111
Cobalt	--	--	--	--	--
Copper	NC	5	0.71	3.6E-5	7.1
Iron	NC	--	--	--	--
Lead(inorg-anic)	NC	10	0.89	4.5E-5	8.9
Magnesium	--	--	--	--	--
Manganese	NC	--	--	--	--
Mercury(alkyl)	NC	--	--	--	--
Mercury(inorg)	NC	7(oral)/ 8(inhal.)	18.4	9.2E-4	186
Nickel	PC	A	--	--	2.9
	NC	10	4.3	2.1E-4	157
Potassium	--	--	--	--	--
Selenium	NC	10	105	5.3E-3	1050
Silver	NC	1	20	1.0E-3	200
Sodium	--	--	--	--	--
Thallium	NC	--	--	--	--
Vanadium	NC	1	0.14	7.1E-6	1.4
Zinc	NC	8	0.11	5.3E-6	1.1
Cyanide	NC	--	--	--	--
Sulfate	--	--	--	--	--

TABLE A-8

Scoring for Indicator Chemicals-Toxicity Information
Westinghouse Project No. 4112-88-907B

Chemical	Tox Class	Rating Val /EPA Cat.	w_T	s_T	a_T
Acetone	NC	--	--	--	--
Benzene	NC	5(oral)/ 10(inhal.)	0.12	5.9E-6	118
	PC	A	7.7E-3	3.9E-7	7.7E-2
Benzo(a)	NC	--	--	--	--
anthracene	PC	B2	0.58	2.9E-5	5.8
Benzo(b)	NC	--	--	--	--
fluoranthene	PC	B2	--	--	--
Benzo(k)	NC	--	--	--	--
fluoranthene	PC	D	--	--	--
Bis(2-ethyl	NC	--	--	--	--
hexyl)phthalate	PC	B2	5.7E-4	2.9E-8	5.7E-3
2-Butanone	--	--	--	--	--
Chlorobenzene	NC	4(oral)/ 1(inhal.)	1.4E-1	7.1E-6	0.28
Chloroform	NC	--	--	--	--
	PC	B2	5.6E-2	2.8E-6	0.56
Chrysene	NC	--	--	--	--
	PC	B2	--	--	--
1,2- Dichloroethene	NC	5	5.3E-2	2.7E-6	0.53
2,6-	NC	9	0.60	3.0E-5	6.0
Dinitrotoluene	PC	C	--	--	--
Ethylbenzene	NC	4	1.1E-2	5.5E-7	0.11
Fluoranthene	NC	--	--	--	--
	PC	--	--	--	--
2-Hexanone	--	--	--	--	--
Methylene	NC	10	9.2E-4	4.6E-8	9.2E-3
Chloride	PC	B2	--	--	--
4-Methyl-2- pentanone	--	--	--	--	--
Napthalene	--	--	--	--	--
N-Nitrosodi- phenylamine	PC	B2	--	--	--
Phenanthrene	NC	--	--	--	--
	PC	--	--	--	--
Phenol	NC	3(oral)/ 10(inhal.)	0.10	5.0E-6	2.5
Pyrene	NC/PC	--	--	--	--
Toluene	NC	7	5.2E-3	2.6E-7	5.2E-2
Xylene	NC	--	--	--	--

TABLE A-9

Calculation of CT and IS Values for Carcinogenic Effects
Westinghouse Project No. 4112-88-907B

Chemcial	GW(CT) Max	GW(CT) Rep	SW(CT) Max	SW(CT) Rep	Soil(CT) Max	Soil(CT) Rep	Air(CT) Max	Air(CT) Rep	IS Value Max	IS Value Rep	Rank Max	Rank Rep
Arsenic	0.81	0.81	.056	.056	.027	1.7E-3	--	--	0.84	0.81	1	1
Benzene	0	0	3.1E-5	3.1E-5	5.1E-9	3.3E-9	--	--	3.1E-5	3.1E-5	2	2
Benzo(a) anthracene	0	0	0	0	8.4E-6	6.5E-6	--	--	8.4E-6	6.5E-6	3	3
Benzo(b) fluoranthene	0	0	0	0	--	--	--	--	--	--		
Benzo(k) fluoranthene	0	0	0	0	--	--	--	--	--	--		
Beryllium	--	--	--	--	--	--	--	--	--	--		
Bis(2-ethyl hexyl) phthalate	0	0	2.9E-6	2.9E-6	1.5E-7	2.9E-8	--	--	3.1E-6	--	4	4
Cadmium	--	--	--	--	--	--	--	--	--	2.9E-6		
Chloroform	0	0	0	0	1.7E-8	1.3E-8	--	--	1.7E-8		5	5
Chromium VI	--	--	0	0	--	--	--	--	--	--		
Chrysene	0	0	0	0	--	--	--	--	--	1.3E-8		
2,6-Dinitro toluene	0	0	0	0	--	--	--	--	--	--		
Fluoranthene	0	0	0	0	--	--	--	--	--	--		
Methylene chloride	--	--	--	--	--	--	--	--	--	--		
Nickel	--	--	--	--	--	--	--	--	--	--		
Phenanthrene	0	0	0	0	--	--	--	--	--	--		
Pyrene	0	0	0	0	--	--	--	--	--	--		

TABLE A-10

Calculation of CT and IS Values for NonCarcinogenic Effects
Westinghouse Project No. 4112-88-907B

Chemcial	GW(CT) Max	GW(CT) Rep	SW(CT) Max	SW(CT) Rep	Soil(CT) Max	Soil(CT) Rep	Air(CT) Max	Air(CT) Rep	IS Value Max	IS Value Rep	Rank Max	Rank Rep
Acetone	--	--	--	--	--	--	--	--	--	--	--	--
Antimony	0.29	0.15	.011	.011	6.8E-4	2.7E-4	--	--	0.29	0.15	7	7
Arsenic	3.55	3.55	.25	.25	0.12	7.7E-3	--	--	3.67	3.56	3	2
Barium	142	5.3	23	3	1.98	0.13	--	--	144	5.4	1	1
Benzene	0	0	4.8E-4	4.8E-4	7.7E-8	5.0E-8	--	--	4.8E-4	4.8E-4	12	12
Benzo(a) anthracene	0	0	0	0	--	--	--	--	--	--	--	--
Benzo(b) fluoranthene	0	0	0	0	--	--	--	--	--	--	--	--
Benzo(k) fluoranthene	0	0	0	0	--	--	--	--	--	--	--	--
Beryllium	--	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethyl hexyl) phthalate	0	0	--	--	--	--	--	--	--	--	--	--
Cadmium	.045	.033	.473	.238	7.6E-3	7.3E-4	--	--	.481	.239	6	5
Chlorobenzene	0	0	4.2E-4	4.2E-4	0	0	--	--	4.2E-4	4.2E-4	13	13
Chloroform	0	0	0	0	--	--	--	--	--	--	--	--
Chromium VI	--	--	--	--	--	--	--	--	--	--	--	--
Chrysene	0	0	0	0	--	--	--	--	--	--	--	--
Copper	.041	.014	.019	.018	1.5E-3	3.5E-4	--	--	.043	.014	8	8
1,2-Dichloroethene	0	0	0	0	1.1E-8	1.1E-8	--	--	1.1E-8	1.1E-8	17	17
2,6-Dinitrotoluene	0	0	0	0	4.2E-5	4.2E-5	--	--	4.2E-5	4.2E-5	14	14
Ethylbenzene	0	0	0	0	1.0E-8	1.0E-8	--	--	1.0E-8	1.0E-8	18	18
Fluoranthene	0	0	0	0	--	--	--	--	--	--	--	--
Iron	--	--	--	--	--	--	--	--	--	--	--	--

TABLE A-10 (Continued)

Calculation of CT and IS Values for NonCarcinogenic Effects
Westinghouse Project No. 4112-88-907B

Chemcial	GW(CT) Max	GW(CT) Rep	SW(CT) Max	SW(CT) Rep	Soil(CT) Max	Soil(CT) Rep	Air(CT) Max	Air(CT) Rep	IS Value Max	IS Value Rep	Rank Max	Rank Rep
Lead	1.68	.159	5.16 E-3	2.67 E-3	.311	1.35 E-2	--	--	1.99	.173	4	6
Manganese	--	--	--	--	--	--	--	--	--	--	--	--
Mercury(alkyl)	--	--	--	--	--	--	--	--	--	--	--	--
Mercury(inorg)	1.05 E-2	1.05 E-2	0	0	0	0	--	--	1.05 E-2	1.05 E-2	9	9
Methylene chloride	2.8E -6	2.8E -6	2.8E -6	2.8E -6	1.1E -9	3.8E -10	--	--	2.8E -6	2.8E -6	16	15
Nickel	0.83	0.53	0.18	0.16	4.3E -3	2.1E -3	--	--	0.83	0.53	5	4
Phenanthrene	0	0	0	0	--	--	--	--	--	--	--	--
Phenol	0	0	0	0	3.1E -6	2.5E -6	--	--	3.1E -6	2.5E -6	15	16
Pyrene	0	0	0	0	--	--	--	--	--	--	--	--
Selenium	0	0	0	0	1.7E -3	1.5E -3	--	--	1.7E -3	1.5E -3	11	11
Silver	0	0	0	0	0	0	--	--	0	0	--	--
Thallium	0	0	0	0	--	--	--	--	--	--	--	--
Toluene	0	0	0	0	2.9E -9	2.1E -9	--	--	2.9E -9	2.9E -9	19	19
Vanadium	7.0E -3	7.0E -3	0	0	1.97 E-4	9.0E -5	--	--	7.2E -3	7.1E -3	10	10
Xylene	--	--	0	0	--	--	--	--	--	--	--	--
Zinc	0.42	3.75 E-2	12.2	3.06	8.0E -3	1.07 E-3	--	--	12.2	3.06	2	3
Cyanide	0	0	0	0	0	0	--	--	0	0	--	--

TABLE A-11

Final Chemical Selection
Westinghouse Project No. 4112-88-907B

Chemical	IS Values PC	IS Values NC	Ranking PC	Ranking NC
Barium	--	5.4	--	1
Arsenic	0.81	3.56	1	2
Zinc	--	3.06	--	3
Nickel	--	0.53	--	4
Cadmium	--	0.239	--	5
Lead	--	0.173	--	6
Antimony	--	0.15	--	7
Copper	--	0.014	--	8
Mercury(inorg)	--	1.05E-2	--	9
Vanadium	--	7.1E-3	--	10
Selenium	--	1.5E-3	--	11
Benzene	3.1E-5	4.8E-4	2	12
Chlorobenzene	--	4.2E-4	--	13
2,6-Dinitrotoluene	--	4.2E-5	--	14
Benzo(a) anthracene	6.5E-6	--	3	--
Methylene chloride	--	2.8E-6	--	15
Bis(2-ethyl hexyl) phthalate	2.9E-6	--	4	--
Phenol	--	2.5E-6	--	16
Chloroform	1.3E-8	--	5	--
1,2-Dichloroethene	--	1.1E-8	--	17
Ethylbenzene	--	1.0E-8	--	18
Toluene	--	2.9E-9	--	19

APPENDIX B

Toxicological Profiles of Indicator Chemicals

ANTIMONY

Occurrence

The main ore of antimony is stibnite. Stibnite is found in low-temperature hydrothermal veins, associated with silver, lead, and mercury minerals. It is also found as a chemical deposit from solutions of hot mineral springs (Mottana et al., 1978). Antimony is mined mainly in China, Mexico, and Bolivia. It is also obtained as a by-product from the smelting of lead and silver ores (Stanford Research Institute). More than one hundred minerals contain antimony, but commercial ores are limited to sulfides and complexes of copper, lead, and silver (Clayton and Clayton, 1981).

Chemical and Physical Properties

Antimony is a silver-white, lustrous, brittle metal with a scale-like crystalline structure (Merck Index, 1976). Antimony is insoluble in hot and cold water. It is soluble in hot concentrated sulfuric acid and aqua regia (Weast, 1979). There are two naturally occurring isotopes of antimony. The metal has two valence states, +3 and +5 (Merck Index, 1976). Some general physical properties of antimony are shown in table B-1.

Use

Antimony is produced by three methods (Stanford Research Institute):

- reduction, using coke, of oxides from roasted low-grade antimony sulfide ores;
- direct smelting of high-grade antimony sulfide ores; or
- production as a by-product from smelting of lead and silver ores.

Alloys are the predominant use of antimony metal since its brittleness renders it impractical for direct use (Clayton and Clayton, 1981). It is used in the manufacture of white metal, metal type, bullets, and bearing metal (Merck Index, 1976). Other uses of antimony are listed below:

- fireworks
- thermoelectric piles
- coating metal
- blackening iron
- infrared detectors in semi-conductors
- storage battery grids
- fire retardant compounds
- ceramic and glass additives
- paint pigments
- rubber vulcanization agents

Included below is information describing the consumption pattern for antimony in 1974 (Stanford Research Institute).

o Transportation (mostly storage battery grids)	44%
o Fire retardant compounds	15%
o Rubber vulcanization agents	9%
o Other components	17%
o Alloys for machinery	5%
o Miscellaneous uses	5%

Environmental Fate and Behavior

Extensive data were not available describing the artificial sources or fate of antimony in the environment. Antimony is a common pollutant in urban air (Doull et al., 1980). It has been suggested that there may be compositional changes in the trace element content of shells of mussels and clams with regard to antimony that might be related to man's influence on inshore marine waters (Bertine and Goldberg, 1972). It has been shown that only trace amounts of antimony pass through a filter scrubber following the installation of the devices on antimony smelting plants.

Human Exposure

Acute antimony poisoning can occur from exposure in industrial operations and from contamination of food containers (Venugopal and Luckey, 1978). Hazardous exposures also occur in mining from dust and fumes by those who crush ores, tend or clean extraction chambers, and collect oxide dusts from roasting chambers (Patty, 1963). Dust encountered in antimony mining may contain free silica, and cases of pneumoconiosis, termed "silico-antimoniosis", have been reported among antimony miners. Antimony ore, converted into fine dust during processing, leads to a high atmospheric concentration of fine dust (International Labor Office, 1971). Dust and fumes produced in the manufacture of storage battery grids, pewter, printer's type, lead shot, lead electrodes, and bearing metals are a source of greatest exposure (Browning, 1969).

Toxicity

Antimony has been reported to cause dermatitis, keratitis, gastritis, conjunctivitis, and nasal septal ulceration by contact, fumes, or dust (Merck Index, 1983). Antimony dust in the foundry has been reported as an important cause of gastrointestinal symptoms of exposed workers with symptoms of dyspnea, headache, vomiting, conjunctivitis, and bloody nose discharges (Hamilton and Hardy, 1974). Contractibility and electrical activity of the myocardium has been demonstrated to be lower, and excitability higher, in workers exposed to antimony than in controls (Beskrovnaya, 1972). Under certain conditions, antimony will react with nascent hydrogen to form stibine (SbH_3) which is extremely toxic (Merck Index, 1976).

Carcinogenicity

Antimony is mutagenic in bacteria or phage. It has been shown to induce chromosomal aberrations or abnormal cell division in animal or plant cells. No data were available to suggest carcinogenic effects in humans, however.

Health Criteria

The Immediately Dangerous to Life and Health (IDLH) level for inhalation of antimony is 80 mg per cubic meter (National Institute for Occupational Safety and Health, 1985). The Time-Weighted Average (TWA) for inhalation is 0.5 mg per cubic meter (American Conference of Governmental Industrial Hygienists, 1988). The Clean Water Act (CWA) Water Quality Criteria for protection of human health in water and fish is 0.15 mg per liter, while in fish only, is 45 mg per liter. The CWA values for the protection of aquatic life in freshwater (acute) is 9.0 mg per liter, and in freshwater (chronic), is 1.6 mg per liter.

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ARSENIC

Occurrence

Arsenic is found widely in nature. It occurs most abundantly as arsenophyrite (FeAsS), a sulfide ore (International Labour Office, 1983). The ore is found in high-temperature hydrothermal veins with gold, silver, and nickel, and frequently in metamorphic deposits. Generally, arsenic is found in combination with elements such as oxygen, chlorine, and sulfur to form inorganic species (Life Systems, Inc., 1987). Common inorganic arsenic species include arsenic trioxide, arsenic trichloride, lead arsenate, and calcium arsenate. Common organic arsenic species include arsenilic acid and methylated compounds formed through biomethylation (Goyer, 1986). In general, inorganic arsenic compounds are more toxic than organic forms (Life Systems, Inc., 1987).

Chemical and Physical Properties

Arsenic is a silver-grey, brittle, crystalline substance (International Labour Office, 1983). It is soluble in nitric acid, but insoluble in water (Weast, 1987). The substance is insoluble in caustic and non-oxidizing acids. Some general physical properties of arsenic are shown in table B-1.

Use

Arsenic metal is produced by three methods. One involves roasting the sulfide to form the oxide and then reducing this oxide with carbon. The second method drives off arsenic oxides by heating arsenopyrite in the absence of air. When arsenic-containing ores are smelted, the arsenic becomes gaseous and burns in air to from arsenic trioxide. The arsenic trioxide is trapped as a dust by

electrostatic precipitators. Roasting drives off the arsenic trioxide, which is collected in a purified form in cooling chambers (International Labour Office, 1983). Arsenic is also found in the flue dust of copper and lead smelters from which it is obtained as arsenic trioxide in various states of purity (Sax and Lewis, 1987).

Arsenic is manufactured in three grades; crude (90-95 percent), refined (99 percent), and semiconductor grade (99.99 percent) (Sax and Lewis, 1987). Major uses of arsenic include:

- alloying constituent in metals
- manufacture of certain types of glass
- to make gallium arsenide for electronic devices
- special solders
- medications (arsenical organic compounds)
- as a radioactive tracer in toxicology
- a catalyst in manufacture of ethylene oxide
- used in semiconductor devices
- wood preservatives
- herbicides and desiccants

Environmental Fate and Behavior

Arsenic is found widely in nature and most abundantly in sulfide ore (FeAsS) (International Labour Office, 1983). It is generally found in combination with other elements, such as oxygen, chlorine, and sulfur to form inorganic species. Arsenic can undergo a number of transformations in surface water, including oxidation-reduction reactions, ligand exchange, biotransformation, precipitation, and adsorption (Life Systems, Inc., 1987). Soluble inorganic arsenate (+5 oxidation state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 oxidation state) (U.S. Environmental Protection Agency, 1980). Arsenic as a free element is rarely encountered in natural waters (U.S. Environmental Protection Agency, 1980).

Data have suggested that atmospheric loadings of zinc, lead, cadmium, and arsenic to lakes are similar and are mostly anthropogenic. Atmospheric loadings in industrialized regions were 1.8 to 2.6 times background levels of nonindustrialized areas (Johnson, 1987). Fish generally contain lower arsenic levels than other aquatic organisms. Livers of fish from the great lakes contain 5.6 to 80 ppm arsenic, mainly in the fat fraction (Lundi, 1970). Arsenic levels in fish livers were not consistently higher than in fish muscle (El Nabawi et al., 1987). The arsenic content of the edible muscle of two tuna species caught in Arabian Sea waters was 2.88 and 2.51 μg per gram dry weight, respectively, for the two species. A marked increase in arsenic content was found with increasing weight of the two fish species (Ashraf and Jaffar, 1988).

Animal hair samples from areas polluted by thermal power plants burning coal were taken and compared with hair samples from animals living in relatively nonpolluted control areas. Animal hair samples from areas with higher levels of pollution have shown higher concentration of toxic and essential elements such as As, Co, Cr, Fe, and Se (Obrunsnik and Paukert, 1984).

Colostrum, transitional milk, and mature milk from 15 human mothers were shown to contain comparable levels of arsenic with no demonstrable differences in concentration, which was around 3 μg per liter (range 0.6-6.3 μg/L) (World Health Organization, 1981).

Human Exposure

Arsenic may be absorbed by ingestion, inhalation, and permeation of skin or mucous membranes (National Research Council of Canada, 1978). Heavy metals such as arsenic, used as color pigments in paints, can be ingested from

contaminated hands, fingernails, food, cups, cigarettes and by holding paint brushes in the mouth (Hart, 1987). Overall, the total dietary intake in the United States was approximately 5 μg of elemental arsenic, representing an increase from the preceding years. Whether this increase represents a trend or reflects analytical variation in sampling from year to year is still to be determined (U.S. Environmental Protection Agency, 1984).

The U.S. Environmental Protection Agency estimates that greater than six million people living within 12 miles of major sources of copper, zinc, and lead smelters may be exposed to 10 times the average levels of arsenic in the United States. An estimated 40,000 people living near some copper smelters may be exposed to levels 100 times the national average (Sittig, 1981).

The list of occupational exposures to arsenic is long, including many metal alloy workers, acid-dippers, and semi-conductor makers (National Institute for Occupational Safety and Health, 1975). High exposure to arsenic fumes and dust may occur in the smelting industries, with the highest concentration most likely occurring among roaster workers. Non-worker populations living near point emission sources of arsenic air may have increases in lung cancer (Doull et al., 1986). Individuals at greatest risk are smelter workers, although there is some suggestion that women residing near such operations incur a greater incidence of respiratory cancer (Doull et al., 1980). Urinary arsenic levels of residents in a downwind transect from a smelter have been shown to decrease with distance from the smelter. Levels were 0.3 ppm at a distance of 0.0 miles and 0.02 ppm at a distance of 2.0 to 2.4 miles. It has been suggested that arsenic exposure is not confined to one section of a smelter but extended also to the surrounding community. Thus "non-exposed" smelter workers might also have a degree of arsenic exposure (National Institute for Occupational Safety and Health, 1984).

Normal values of arsenic in urine vary from 0.013 to 0.046 mg per liter to 0.13, and as high as 0.25 mg per liter (American Conference of Governmental Industrial Hygienists, 1986). Physiological factors, personal habits, use of drugs, genetic disorders, acquired diseases, and pathological factors alter biological indicators used to assess industrial worker exposure (Alessio et al., 1987).

Populations at special risk include persons in arsenic work with existing diabetes, cardiovascular diseases, allergic or other skin diseases, or with neurologic, hepatic, or renal lesions (International Labour Office, 1983). Evidence suggests that children may also be at special risk for the effects of inorganic arsenic under the conditions of acute or subacute exposure (U.S. Environmental Protection Agency, 1984).

Toxicity

Exposure to high levels of arsenic results in a number of systemic effects. Typical systemic effects include decreased production of red and white blood cells, abnormal heart and nerve function, and damage to blood vessels, kidneys, and liver (Life Systems, Inc., 1987).

Absorption of arsenic from the gastrointestinal tract depends upon the water solubility of the compound. Studies using laboratory animals indicate that over 90 percent of soluble trivalent and pentavalent arsenic is absorbed (Life Systems, Inc., 1987). In humans, over 95 percent of ingested inorganic arsenic is absorbed. Studies of absorption of arsenic following inhalation indicate that humans absorbed 75 to 85 percent of that deposited in the lungs within four days (Life Systems, Inc., 1987). Not enough information exists concerning dermal absorption of arsenic compounds to evaluate exposure via that route. Acute oral

doses of 50 to 300 mg of inorganic arsenic may result in death to adult humans, while subchronic ingestion of 3 mg per day has been reported to be fatal in infants exposed via contaminated milk (Life Systems, Inc., 1987). Oral LD_{50} values for inorganic arsenic compounds have been reported to range from 10 to 300 mg per kilogram body weight in animals. An oral LD_{50} of 15 mg per kilogram body weight was reported for rats. The results of many studies on the lethality of arsenic compounds suggest that non-human mammals are less sensitive than humans to arsenic (Life Systems, Inc., 1987).

Carcinogenicity

The burden of evidence shows arsenic to be a respiratory and dermal carcinogen. Arsenic induced cancer usually occurs at more than one site and has a long latent period (i.e., from 13 to 50 years after the initial exposure). Studies indicate that a respiratory exposure of 3 μg per cubic meter for less than one year can result in respiratory cancer (National Research Council of Canada, 1978). A great deal of evidence indicates chronic oral exposure to toxic levels of arsenic increases the risk of skin cancer. In one study, residents of a Mexican town exposed to 0.4 mg per liter of arsenic in drinking water had a 3.6-fold increase in basal cell carcinomas compared to residents of a similar town where water contained 0.005 mg per liter (Life Systems, Inc., 1987).

Health Criteria

Maximum Contaminant Levels (MCLs) have been established under the Resource Conservation and Recovery Act (RCRA) and Safe Drinking Water Act (SDWA), for public drinking water systems. MCLs under RCRA and SDWA are 0.05 mg per liter.

The proposed MCL Goal (MCLG) is 0.05 mg per liter. The SDWA MCL for arsenic was established for health concerns and is thus a primary standard.

The Virginia State Water Control Board (SWCB) adopted the SDWA MCL for both surface drinking water supplies and ground water protection. The standard for protection of aquatic life from chronic exposure to arsenic is 190 μg per liter as As (III).

Water Quality Criteria were established under the Clean Water Act (CWA) for arsenic to protect human health. The values are 2.2×10^{-6} mg per liter for water and fish, and 10×10^{-6} mg per liter for fish only. The standards for protection of aquatic life in freshwater (acute) and freshwater (chronic) are 0.8 mg per liter As(V) and 0.3 mg per liter As (III), and 0.048 mg per liter As(V) and 0.1 mg per liter As (III), respectively.

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BARIUM

Occurrence

Barium occurs in nature primarily in two mineral forms, barite ($BaSO_4$) and witherite ($BaCO_3$). Barite is a common mineral in medium and low temperature hydrothermal veins associated with lead, silver, and antimony sulfides and in replacement veins, cavernous limestone formations, and dolomites. Witherite occurs in low temperature hydrothermal veins. It is associated with barite and galena (Mottana et al., 1978).

Chemical and Physical Properties

The mineral forms of barium are relatively insoluble in water, have high melting and boiling points, and very low vapor pressures (U.S. Environmental Protection Agency, 1985). Barium combines readily with oxygen, nitrogen, hydrogen, ammonia, water, halogens, and sulfides. Barium acetate, nitrate, chloride, and hydroxide are very soluble in water. The solubility of barium compounds increases as pH decreases. There is no detectable odor or taste. The compound is soluble in alcohol and insoluble in benzene (Weast, 1988). The major physical characteristics of the compound are listed in table B-1.

Naturally occurring barium is a mixture of seven stable isotopes. Thirteen other radioactive isotopes are known to exist. The substance is somewhat malleable and has a distinctive property of absorbing gases (Clayton and Clayton, 1981).

Use

Barite is the major barium ore. It is used as an additive when drilling for oil, in the paper and rubber industries, in radiography, as a screen for intense radiation when mixed with cement mortar, and to make expensive white pigment. Witherite is mined as a barium ore when available in large enough amounts. It is used in the production of special types of glass.

Barium is extracted by heating barium oxides with aluminum in vacuoles and condensing the barium vapor in the cool end of the tubes. Alloys of lead, barium, and calcium can be prepared (Browning, 1969). Metal containing barium is produced commercially by reduction with a less reactive, non-volatile element such as aluminum (Kirk-Othmer, 1978). Barium can be purified by vacuum distillation. Manufactured forms of the material include solid, powdered, and liquid forms of barium, cadmium, zinc, and phosphorus (Kuney and Mullican, 1988). Additional uses of barium compounds are listed below.

- deoxidizer for copper
- lubricant in x-ray tubes
- spark plug alloys
- ingredients of paints, soap, paper, and rubber
- manufacture of ceramics and glass
- heat stabilizer for plastics
- to improve performance of lead alloy grids of acid batteries
- in the manufacture of steel, copper, and other metals
- a carrier for radium
- emissions used as standards in gamma-spectrometry

The largest end use of barium is as a removal agent in cleaning traces of gases from vacuum and television picture tubes (U.S. Environmental Protection Agency, 1985).

Environmental Fate and Behavior

Barium metal does not occur free in nature (Kirk-Othmer, 1978). It is found as a constituent in zinc or iron ores. Seawater contains about 0.03 ppm barium (Venugopal and Luckey, 1978). No data exist describing the environmental fate, environmental transformations, or transport of the compound. Background levels for soil range from 100 to 3,000 ppm barium (Brown et al., 1983). Barium is emitted into the atmosphere mainly by the industrial processes involved in the mining, refining, and production of barium and barium based chemicals, and as a result of the combustion of coal and oil (Miner, 1969). The average concentration of barium was 5 ng per cubic meter (with a range of 0-1500) in 18 cities in the United States (Friberg et al., 1986).

There are no widespread data available describing environmental concentrations of barium in food, plants, fish, mammals, or milk.

Human Exposure

The probable routes for human exposure include ingestion or inhalation of dust or fume and skin or eye contact. Small numbers of people in Illinois, Kentucky, Pennsylvania, and New Mexico are known to be consuming well waters in which barium concentration ranges are from 1 to 10 times greater than the standard for total barium (1 mg/L is the Interim Primary Drinking Water Standard) (National Research Council, 1977). The average daily intake for the human adult is about 1.3 mg (0.65-1.7 mg) of total barium (Venugopal and Luckey, 1978). Typical barium dietary intake is believed to originate 25 percent from milk, 25 percent from flour, 25 percent from potatoes, and 25 percent from miscellaneous high barium foods consumed in minor quantities, especially nuts (Friberg et al., 1986).

Normal levels of barium in various organs of unexposed persons have been published. About 90 percent of the total body barium occurs in the skeleton. Measurable levels were observed in the eye, lungs, connective tissues, skin, and adipose tissue. In internal organs, barium concentrations were slight (Friberg et al., 1986). Individuals with pulmonary diseases are considered at special risk for barium exposure (ITII, 1982).

Toxicity and Carcinogenicity

Whereas insoluble forms of barium are not toxic by inhalation or ingestion because only minimal amounts are absorbed, soluble barium compounds are highly toxic in humans by either route of exposure. The most important effect of acute barium exposure is a strong, prolonged stimulant action on muscle. Smooth, cardiac, and skeletal muscles are all affected, and a transient increase in blood pressure due to vasoconstriction can occur. Effects on the hematopoietic system and cerebral cortex have also been reported in humans. Accidental ingestion of soluble barium salts has resulted in gastroenteritis, muscle paralysis, and ventricular fibrillation and extra systoles. A benign pneumoconiosis, baritosis, can result from inhaling barium dusts. Doses of barium carbonate and barium chloride of 57 mg per kilogram and 11.4 mg per kilogram, respectively, have been reported to be fatal in humans.

There are no reports of carcinogenicity associated with exposure to barium or its compounds. Effects on gametogenesis and on the reproductive organs have been reported from animal studies after inhalation of barium carbonate.

Health Criteria

The Maximum Contaminant Level (MCL) for barium has been established under the Resource Conservation and Recovery Act (RCRA) to be 1.0 mg per liter. The proposed Safe Drinking Water Act (SDWA) MCL and the proposed MCL Goal (MCLG) are both 5.0 mg per liter. The higher proposed level presumably reflects a lower toxicity for barium than previously anticipated. The SDWA MCL for barium is established for health concerns and is thus a primary standard.

The Virginia State Water Control Board (SWCB) adopted the original SDWA MCL of 1.0 mg per liter for surface drinking water supplies and ground water protection. There is no standard for protection of aquatic life. A water quality criteria of 1.0 mg per liter was set for streams from which humans might ingest water and fish.

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BENZENE

Occurrence

Benzene occurs naturally in volcanos and forest fires. It is a natural constituent of crude oil and plant volatiles (International Agency for Research on Cancer, 1982; Graedel, 1978).

Chemical and Physical Property

Benzene is a clear colorless liquid with an aromatic odor. Its basic physical properties are shown in table B-1 appended to this report. Liquid benzene is miscible with alcohol, chloroform, ether, carbon disulfide, acetone, carbon tetrachloride and glacial acetic acid (Merck Index, 1983). Benzene vapors burn with a smoky flame (International Labour Office, 1983).

Manufacture

The process whereby dehydrocyclization catalysts convert paraffins to aromatics, and dehydrogenation catalysts convert cycloparaffins to naphthenes, using platinum and palladium, is known as catalytic reforming, and is the primary source of aromatics (Kirk-Othmer, 1978). The production of benzene by these processes is associated with the production of toluene and xylene (BTX plants) (Kirk-Othmer, 1978). Benzene is one of the principal components of the light oil recovered from coal carbonization gases (Sittig, 1976).

A miscellaneous source of benzene is recovery from coal tar. The lowest boiling distillate from coal tar is extracted with caustic soda to remove the tar acids. The benzene containing fraction of the oil is purified by hydrodealkylation (Kirk-Othmer, 1978).

The major impurities of benzene are toluene and xylene. Others are phenol, thiophene, carbon disulfide, acetonitrile, and pyridine (National Institute for Occupational Safety and Health, 1974). The following grades of benzene are available (Sax and Lewis, 1987):

- crude
- straw color
- motor
- industrial pure
- thiophene-free
- 99 mole %
- 99.94 mole %
- nanograde

Benzene is a component of gasoline. In Europe the concentration of benzene in gasoline is in the range of 5 to 16 percent. In the United States the concentration ranges from 0.3 to 2.0 percent, averaging around 0.8 percent (National Institute for Occupational Safety and Health, 1974). The production of benzene on a commercial scale was not undertaken until about 1941, although petroleum was known to contain benzene (Kirk-Othmer, 1978). Benzene has been banned as an ingredient in products intended for use in the home (Gosselin et al., 1984). In 1985, it was the 16th highest volume chemical produced in the United States (Sax and Lewis, 1987).

Benzene is used in the manufacture of the following materials:

- medicinal chemicals
- dyes
- organic compounds
- artificial leather
- linoleum
- oil cloth
- rubber
- inks for graphics arts
- polyester resins
- nylon
- insecticides
- varnishes
- lacquers
- solvents for waxes
- resins
- oils
- paints
- adhesives & coatings
- detergents
- polystyrene plastics
- synthetic rubber

It is also used as a thinner for paints, a degreasing agent, a dry cleaning agent, and in lithography (Fishbein, 1977). The tire industry and shoe factories use benzene extensively (Goodman and Gilman, 1985).

In the future, coal will increasingly replace petroleum and natural gas as a source of hydrocarbons for fuel and petrochemicals. Processes such as USA Steel Corporation's Clean Coke process, which yields 38 percent coke and 20 percent chemical by-products, compared to 73 percent coke and 2 percent chemical by-products in conventional coking technology, should soon be used commercially. New coking, liquefaction, and gasification processes for coal are all potential sources of benzene (Kirk-Othmer, 1978).

Environmental Fate and Behavior

Benzene enters the environment from sources such as gasoline, and from emissions associated with the production, storage, and the transport of benzene itself. Environmental sources result from benzene's use as an intermediate in the production of other chemicals, and as a solvent. Other sources result from losses such as spills, including oil spills. A summary of environmental sources include the following:

- production, storage, transport, venting, and combustion of gasoline
- production, storage, and transport of benzene itself
- from use as an intermediate in the production of other chemicals
- from use as a solvent
- spills (including oil spills)
- non-ferrous metal manufacturing
- ore mining
- wood processing
- coal mining
- textile manufacture
- cigarette smoke

In 1976, an estimated 1.3 billion pounds of benzene were released into the atmosphere from 132 million stationary and mobile sources. (Department of Health and Human Services/National Toxicology Program, 1985).

If benzene is released into the atmosphere, it will exist primarily in the vapor phase (Eisenreich, 1981). Gas-phase benzene will be subject to direct photolysis but it will react with photochemically produced hydroxyl radicals. The reaction time in polluted atmospheres which contain nitrogen oxides or sulfur dioxide is accelerated, with the half-life being reported as four to six hours (Korte and Klein, 1982). Products of photooxidation include phenol, nitrophenols, nitrobenzene, formic acid, and peroxyacetyl nitrate. Benzene is removed from the atmosphere in rain due to its fair solubility in water (Kato et al., 1982). Data indicate that the concentration of benzene in the atmosphere is highest near chemical factories where benzene is used, near service stations, and near cigarette smoke (Brodzinsky and Singh, 1982).

If benzene is released to soil, it will be subject to rapid volatilization near the surface. The benzene which does not evaporate will be highly to very highly mobile in soil, and may leach to groundwater (Jury et al., 1984). It may be subject to biodegradation based on the reported biodegradation rate of 24 percent and 47 percent of the initial 20 ppm benzene in a based-rich para-brownish soil in one and ten weeks, respectively (Haider, 1974).

If benzene is released to water, it will be subject to rapid volatilization. Benzene will not be expected to significantly adsorb to sediment, bioconcentrate in aquatic organisms, or hydrolyze. It may be subject to biodegradation under certain conditions (Vaishnau and Babeu, 1987; Wakeham, 1983). Photodegradation could contribute to benzene's removal. In situations of cold water, poor

nutrients, or other conditions less conducive to microbial degradation, photolysis may play an important role in degradation (Wakehum et al., 1983).

Measured Concentrations of Benzene in Surface Water

Area	Number Sampled	Percent Positive	Range (ppb)
Heavily industrialized areas with basins (1975-76)	14	20% (>1 ppb)	1-7
Lake Erie (1975-76)	2	50%	0-1
Lake Michigan (1975-76)	7	71%	0-7
700 random sites in U.S. (1975)	N		5.4 = average
U.S. EPA STORET database	1,271	15%	5.0 = median

(Ewing, Chain, Cook, Evans, Hopke, and Perkins, 1977; Konasewich, 1978; Kraybill, 1977; and Staples, 1985)

Benzene occurs in both ground water and surface public water systems, with higher levels in the ground water supplies. Based upon Federal drinking water surveys, approximately 13 percent of all ground water systems are estimated to contain benzene at levels greater that 0.5 μg per liter (e.g., the highest level was 80 μg/L). Approximately 3 percent of all surface water systems are estimated to be contaminated at levels higher than 0.5 μg per liter. None of the systems are estimated to contain levels higher than 5 μg per liter (U.S. Environmental Protection Agency, 1987).

The following bioconcentration factors (BCF's) have been reported:

Organism	BCF
eels	3.5 (Ogata and Miyake, 1978)
pacific herring	4.4 (Korn et al., 1977)
gold fish	4.3 (Ogata et al., 1984)

Based on a reported log Kow (Octanol-Water Partition Coefficient) of 2.13 (Hansch and Leo, 1985), a BCF of 24 was estimated (Lyman, 1982). Based on the reported and estimated BCF, benzene will not be expected to bioconcentrate in aquatic organisms (Syracuse Research Corporation).

Human Exposure

Human populations are primarily exposed to benzene though inhalation of contaminated ambient air, particularly in areas with heavy traffic and around filling stations. In addition, air close to manufacturing plants which produce or use benzene may contain high concentrations of benzene (International Agency for Research on Cancer, 1982; Graedel, 1978). Another source of exposure from inhalation is from tobacco smoke (International Agency for Research on Cancer, 1982). Although most public drinking water supplies are free of benzene or contain less than 0.3 ppb, exposure can be very high from consumption of water drawn from wells contaminated by leaky gasoline storage tanks, land fills, etc. Benzene has been detected in food, but data is too scant to estimate exposures (Stanford Research Institute, 1977). Benzene has been detected in 8 samples of mothers milk from four U.S. urban areas (Pellizzari et al., 1982).

Toxicity

A large amount of literature is available describing the harmful effects of benzene exposure in humans. Acute exposure to a large amount of benzene by ingestion or inhalation causes a major toxic effect on the central nervous system. Symptoms from mild exposure include dizziness, weakness, euphoria, headache, nausea, vomiting, tightness in chest, and staggering. If the exposure is more severe, symptoms progress to blurred vision, tremors, shallow and rapid

respiration, ventricular irregularities, paralysis, and unconsciousness (Goodman and Gilman, 1985). Many acute deaths, from benzene exposure at high concentrations, have been due to ventricular fibrillation caused by exertion and release of epinephrine. Frequently, an individual who enters a contaminated area to remove an unconscious worker dies during the effort of lifting and carrying the unconscious worker (Thienes and Haley, 1972).

Data from animals and human studies indicate that benzene must undergo biotransformation to exert its toxic effects. Benzene is primarily metabolized in the liver, however the enzymes necessary for metabolism are also present in bone marrow, the reported organ of toxicity. The lymphoid system is another target organ of benzene toxicity (Oak Ridge National Laboratories, 1987).

The hematotoxicity of benzene is expressed primarily as a bone marrow effect leading eventually to complete destruction of myeloid and erythroid marrow components. This effect is manifested as a marked decrease in circulating formed elements (i.e., red blood cells and platelets). The resultant aplastic anemia is a potentially fatal disorder which, in its severe form, has a better than 50 percent mortality rate. In both human and laboratory animals, the extent of bone marrow damage appears proportional to the dose of benzene. Lesser degrees of bone marrow toxicity than aplastic anemia are more common in occupational exposure situations. Classically, the discovery of one individual with significant bone marrow toxicity has led to evaluation of the exposed work force and the finding of a wide variation in the extent of hematotoxicity. This has ranged from decreases in white blood cells, red blood cells, and platelets to a situation in which only one of these is slightly below the normal range (Mehlman, 1983).

In addition to hematotoxicity and immunotoxicity, benzene can cause neurotoxic effects such as drowsiness, dizziness, headache and loss of consciousness. Benzene has not been teratogenic in animal studies, but has caused increased incidences of resorptions, reduced fetal weight, and skeletal variations.

Chronic benzene toxicity is expressed as bone marrow depression resulting in leucopenia, anemia, or thrombocytopenia (leucomogenic action). With continued exposure, the disease progresses to pancytopenia resulting from bone marrow aplasia. Evidence has accumulated implicating benzene in the etiology of leukemias in workers in industries where benzene was heavily used (Snyder et al., 1977).

Carcinogenicity

A major concern is the relationship between chronic exposure to benzene and leukemia. Epidemiological studies have been conducted on workers in the tire industry (McMichael et al., 1975) and in shoe factories (Aksoy, 1974), where benzene is used extensively. Among workers who died from exposure to benzene, death was caused by either leukemia or aplastic anemia in approximately equal proportions (Goodman and Gilman, 1985).

The type of leukemia most commonly associated with benzene is acute myelogeneous leukemia and its variants, including erythroleukemia and acute myelemonocytic leukemia. Acute myelogenous leukemia is the adult form of acute leukemia, and until recent advances in chemotherapy, it was a rapidly fatal disease. The other major form of leukemia, acute lymphocytic leukemia, has been reported to be associated with benzene exposure, but evidence of causal association is weak. There is a somewhat stronger, although still inconclusive,

association in the literature between benzene exposure and the two forms of chronic leukemia, chronic myelogenous leukemia and chronic lymphocytic leukemia. Other hematological disorders possibly associated with benzene exposure include Hodgkin's disease, lymphocytic lymphoma, myelofibrosis and myeloid metaplasia, paroxysmal nocturnal hemoglobinuria, and multiple myeloma (Mehlan, 1983).

A significantly increased frequency of chromatid and isochromatid breaks in the cultured lymphocytes of workers in chemical laboratories and in the printing industry has been reported (Funes-Cravioto et al., 1977). In 52 workers with an eight hour time-weighted average (TWA) exposure of 2 to 3 ppm, in association with short term exposure concentrations of 25 ppm and peak concentration of 50 ppm, chromosomal aberration rates were observed that were two to three times the rates found in controls. These included chromosome breaks, dicentric chromosomes, translocations, and exchange figures (U.S. Environmental Protection Agency, 1980).

Thirty two patients who had recovered from a blood disease (bone marrow impairment) caused by benzene poisoning showed chromosome aberrations present for several years after cessation of the exposure, and after recovery from poisoning (Waldbott, 1973; Pollini and Colombi, 1964). Structural and numeric chromosome aberrations and sister chromatid exchanges have been caused by benzene, although gene mutations have rarely been shown to be caused by benzene exposure (Oak Ridge National Laboratories, 1987).

Health Criteria

A primary drinking water standard of 0.005 mg per liter has been set under the Safe Drinking Water Act (SDWA). The proposed Maximum Contaminant Level Goal

(MCLG) is zero mg per liter. The Virginia State Water Control Board (SWCB) has not set standards for benzene in water.

Water quality criteria have been established under the Clean Water Act (CWA) for protection of human health and freshwater aquatic life. With respect to protection of human health, levels for exposure both from drinking water and from consuming aquatic organisms, and from fish consumption alone, are 6.6×10^{-4} mg per liter and 4×10^{-2} mg per liter, respectively. A value of 5.3 mg per liter, the lowest observed effect level, has been established for protection of aquatic life in freshwater (Acute).

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BIS (2-ETHYLHEXYL) PHTHALATE

Occurrence

Bis(2-ethylhexyl)phthalate (DEHP) is produced commercially by the esterification of phthalic anhydride with 2-ethylhexanol. It may be a natural product in animals and plants (Howard, 1989).

Chemical and Physical Properties

The molecular weight of DEHP is 390 grams per mole. The compound is sparingly soluble in water (0.3 mg/l at 25°C) and has a low vapor pressure (6.45 x 10^{-6} mm Hg at 25°C). The log octanol/water partition coefficient is 5.11. DEHP boils at 230°C (5 mm Hg) and melts at -50°C (Howard, 1989).

Use

The major use of DEHP is as a plasticizer for polyvinyl chloride (PVC) resins. DEHP is also used as an inert ingredient in pesticides, a component of dielectric fluids (replacing PCBs) in electrical capacitors, and in vacuum pump oil (Life Systems, Inc. 1987).

Environmental Fate and Behavior

The environmental fate of DEHP is strongly influenced by its physical and chemical properties. Biodegradation of DEHP occurs under aerobic conditions with a half-life of several weeks to a month. Under anaerobic conditions, DEHP degrades much more slowly. Reaction with the hydroxyl radical is followed by further atmospheric chemical reactions. Deposition and rainout removes DEHP from the atmosphere. DEHP is lipophilic. In the aquatic environment, DEHP

accumulates in sediments, on suspended solid materials, and in the lipid tissues of aquatic biota. In soil, DEHP is strongly held by soil solids and organic material such as fulvic acid, from which it is not readily leached.

Human Exposure

Human exposure to DEHP can occur by a variety of routes. In addition to occupational exposures, humans can be exposed to DEHP leached from plastic products such as water piping and plastic food containers (Verschueren, 1983).

Toxicity

A number of studies have been conducted to investigate the acute toxic effects of DEHP. When administered by the oral, intraperitoneal, intravenous, and inhalation routes, DEHP has a low order of acute toxicity.

The target organs for DEHP appear to be the liver and testes. At relatively high dose levels DEHP has been found to induce morphological and biochemical changes in the liver of exposed rodents. Similar effects have been reported for a number of chemicals which induce hepatic xenobiotic metabolizing capabilities. The testicular effects of DEHP are characterized by a decrease in relative organ weight and damage to the seminiferous tubules. Similar effects have been reported in animals treated with a major metabolite of DEHP.

Studies in rats and mice suggest that DEHP is developmentally toxic. In the rat, a variety of congenital abnormalities have been observed in the offspring of DEHP-treated females. In mice, the developing nervous system appears to be the major target site, producing exencephaly and spina bifida. DEHP is a reproductive toxicant in male and female mice, resulting in reduced fertility including fewer litters and decreased litter size.

A large database exists on the genotoxicity of DEHP. DEHP has been subjected to extensive testing in bacteria and both in vitro and in vivo mammalian assay systems. Evidence suggests that DEHP is not mutagenic in bacterial or mammalian test systems.

Carcinogenicity

In a study by the National Toxicology Program, DEHP caused an increase in the incidence of hepatocellular tumors in both sexes of Fischer 344 rats and B6C3F1 mice. There was a trend toward increasing numbers of tumors with increasing doses. In a draft report, the U.S. Environmental Protection Agency concluded that DEHP is a probable human carcinogen, classified as weight-of-evidence Group B2.

Health Criteria

MCLs have not been established for bis (2-ethylhexyl) phthalate under the SDWA. The only standard set by the SWCB is for protection of aquatic life (3.0 ug/l). Water quality criteria have been established under the CWA both for drinking water and consuming aquatic organisms (15 mg/l) and from fish consumption alone (50 mg/l).

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CADMIUM

Occurrence

Cadmium is a naturally occurring metallic element that occurs widely, usually in association with lead or zinc. Cadmium occurs in greenockite (cadmium sulfide) ore containing zinc sulfide. It also occurs with lead and copper ores which contain zinc. Among sedimentary rock types, the carbonaceous shales, formed under reducing conditions, contain the most cadmium (National Research Council of Canada, 1979). Sphalerite, the main ore for zinc, usually provides cadmium as a by-product (Mottana et al., 1978).

Chemical and Physical Properties

Cadmium is a silver-white, blue-tinged, lustrous metal (Merck Index, 1983). It is odorless (National Institute for Occupational Safety and Health, 1981). Cadmium tarnishes in moist air. The metal is highly corrosion resistant (Sittig, 1981) and malleable (Weast, 1987). Its electrical conductivity is less than silver and copper, but greater than that of iron (Chizhikov, 1966). Cadmium is reported to be insoluble in water (Merck Index, 1983), but soluble in acid, ammonium nitrate, and hot sulfuric acid (Weast, 1987). The hydroxide, oxide, carbonate, and sulfide forms are insoluble in water. The bromide, chloride, fluoride, iodide, nitrate, and sulfate salts of cadmium are relatively soluble compounds (U.S. Environmental Protection Agency, 1985). There are eight stable isotopes of cadmium (National Research Council of Canada, 1979). Other physical properties of cadmium are listed in table B-1.

Use

Cadmium is a by-product of the processing of zinc bearing ores. Zinc concentrates and fine dust are dissolved in sulfuric acid, and the cadmium sponge is precipitated and purified by electrolytic or pyrometallurgical processes. Cadmuim is available in bars, sheets, wire, or granular powder (Merck Index, 1983). It is available in 99.5 to 99.99 percent purity grades (Kuney and Mullican, 1987). Consumption patterns reported indicate that the dominant uses of cadmium are in metal plating and coating, batteries, plastics, stabilizers, pigments, and metal alloys (Bureau of Mines, 1987). Some specific uses of cadmium include:

- alloy with copper, nickel, gold, silver, bismuth and aluminum
- electroplating of automotive, aircraft, electronic parts, marine and industrial machinery
- cadmium selenide mixtures used as pigments
- process engraving
- photometry of UV sun-rays
- electrodes for cadmium vapor lamps
- photoelectric cells (solar-powered batteries)
- in fire protection systems
- baking enamel
- photography and lithography
- plastics stabilizers
- analytical reagent for determination of nitrate
- television phosphors
- neutron absorber in nuclear reactors

Environmental Fate and Behavior

Volcanic action is considered to be the major natural source of cadmium. This is due to the very large quantities of particulate matter emitted, together with the high enrichment of cadmium in volcanic aerosols (Bart-Menard and Arnold, 1978). Coal and other fossil fuels contain cadmium, and their combustion releases the element into the environment (Gilman et al., 1985).

Artificial sources of cadmium include liberation during the following manufacturing processes (National Institute for Occupational Safety and Health, 1981):

- o smelting and refining of ores
- o scrap metal recovery
- o melting and pouring of cadmium metal
- o casting of alloys
- o coating of telephone cables, trolley wires, and other products
- o welding
- o melting of cadmium ingots for paint and pigment manufacture
- o manufacture of cadmium-nickel batteries
- o incineration of plastics
- o kiln use by ceramic artists

Cadmium can enter surface waters from the natural sources and from a variety of manufacturing operations that involve either cadmium itself or zinc that contains a cadmium impurity. Cadmium can enter the water environment from the plating operations when spent plating solutions are discarded. The production of refined cadmium metal is a potential source of cadmium in nearby surface waters (International Agency for Research on Cancer, 1973).

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals, and may be transported in solution as hydrated cations or organic or inorganic complexes. In polluted waters that are high in organic carbon content, complexing with organic matter is the most important factor in determining the aquatic fate and transport of cadmium. Sorption accounts for removal of dissolved cadmium to bed sediments, and is increasingly effective as pH increases. Cadmium is strongly accumulated by organisms at all trophic levels.

Human Exposure

Cadmium is a potent toxicant by both oral and inhalation exposure routes (Merck Index, 1983). Dermal exposure to cadmium compounds is not considered a significant health concern, although absorption routes include cutaneous and transplacental absorption. Cadmium can be inhaled in the form of aerosols or fumes. Humans are exposed through food, water, air, and especially heavy smoking (Gosselin et al., 1984).

Some occupations at risk include alloy makers, battery makers, engravers, textile workers, welders, solder workers, and zinc and lead refiners. Exposure occurs primarily in smelting and refining (International Labour Office, 1971). These occupational exposures represent populations at special risk to cadmium. Other populations at special risk include individuals with the following (Department of Health and Human Services/Agency for Toxic Substances and Disease Registry, 1987):

- renal diseases of other etiology
- genetic differences in the induction of metallothioneic response to cadmium exposure
- dietary deficiencies in metal ions or protein
- neonates or children possibly having higher gastrointestinal absorption rates

Toxicity

Cadmium is a potent toxicant by both oral and inhalation exposure routes. Dermal exposure to cadmium compounds is not considered a significant health concern. Exposure to cadmium is associated with injury to a number of tissues and organs in both animals and humans, including the kidney, liver, cardiovascular system, skeleton, and immune system (Gosselin et al., 1984; and American Conference of Governmental Industrial Hygienists, 1986). Similar

effects are observed for both inhalation and oral exposure, but are not observed following dermal exposure.

Acute exposures to high levels of cadmium compounds are highly irritating to the epithelial cells of the gastrointestinal and respiratory tracts. Other tissues injured by high doses include the liver and testes (Life Systems, Inc., 1987). High levels of exposure associated with injury to these tissues are unlikely. The principal health concern is for chronic low-level exposure to cadmium by either the oral or inhalation route.

Carcinogenicity

Cadmium has been shown to be carcinogenic in humans following exposure by the inhalation route. A retrospective mortality study of 602 white males employed in a cadmium-processing plant showed that the high exposure group had statistically significant excess mortality rates. The excess in lung cancer could not be explained by smoking or previous exposure to arsenic (Life Systems, Inc., 1987).

No data were available on the carcinogenic effects of cadmium in orally exposed humans. Studies conducted to date in animals have not shown cadmium to be carcinogenic by the oral route.

Health Criteria

Primary drinking water standards have been established for cadmium under the Safe Drinking Water Act (SDWA). The Resource Conservation and Recovery Act (RCRA) Maximum Contaminant Level (MCL) is 0.01 mg per liter. The proposed SDWA MCL and MCL Goal (MCLG) are 0.005 mg per liter. The Virginia State Water Control Board (SWCB) adopted the original SDWA MCL of 0.01 mg per liter for surface

water. The Virginia SWCB standard for protection of aquatic life is a function of hardness. The Virginia SWCB ground water standard is 4×10^{-4} mg per liter. The Clean Water Act (CWA) Water Quality Criteria for protection of aquatic life from acute and chronic exposure to cadmium are 0.0039 mg per liter and 0.0011 mg per liter, respectively.

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COPPER

Occurrences

Copper is widely distributed in nature. It is found in its native elemental state and in sulfide, arsenite, chloride, and carbonate ores (Seiler et al., 1988). Several minerals which include copper are chalcopyrite, chalcocite, bornite, tetrahedrite and enargite (Merck Index, 1983). About 80 percent of the world's copper is derived from treatment of chalcopyrite ore. The ore yields the by-products of gold and silver. Native copper rarely occurs in large enough quantities to be worth exploiting commercially (Mottana et al., 1978).

Chemical and Physical Properties

Copper is a reddish metal. It naturally crystallizes in a face centered cubic structure (Merck Index, 1983). Copper is a lustrous and malleable metal. The metal is insoluble in hot and cold water, soluble in nitric acid and hot sulfuric acid, and very slightly soluble in hydrochloric acid and ammonium hydroxide. (Weast, 1987). Copper conducts heat and electricity exceedingly well (International Labour Office, 1983). Copper forms two series of salts with its Cu^{+1} and Cu^{+2} valence types. Both valence types form complex ions that are stable. Other physical properties of copper are listed in table B-1.

Use

Copper is extracted from ores by one of two processes, depending on whether it is in sulfide or oxide ore. Sulfides are crushed, grinded, and concentrated by floatation, while oxide ores are concentrated by leaching with acid. The concentrates are smelted to yield a blister copper which is refined

electrolytically or by fire (Stanford Research Institute). Commercial copper is available in the following six general types (Considine, 1974).

Type	Percent Copper
Electrolytic tough-pitch	99.90
Deoxidized	99.90
Oxygen-free	99.92
Silver-bearing	99.90
Arsenical	99.68
Free-cutting	99.4 - 99.5

Forms of copper available include ingots, sheet, rod, wire, tubing, shot, and single crystals or whiskers (Sax and Lewis, 1987). Listed below are dominant uses of copper (Stanford Research Institute):

	Percent Copper
Electrical & electrical products	54
Building construction	20
Industrial Machinery and equipment	13
Transportation	8
Other	5

Uses of copper are listed below:

- heating, chemical, and pharmaceutical machinery
- metal alloys
- electroplated protective coatings and undercoats for nickel, chromium, and zinc etc.
- cooking utensils
- corrosion resistant piping
- catalysts
- insulation for liquid fuels
- agricultural products (insecticides, fungicides, and herbicides)
- food and drugs
- metallurgy
- nylon
- paper products
- pigment & dyes
- pollution control catalyst
- printing and photo copying
- pyrotechnics
- wood preservatives

Environmental Fate and Behavior

Copper is found in its native state and in association with other elements, such as sulfides, arsenites, chlorides, and carbonates. The concentration of copper in the continental crust is generally estimated around 50 to 70 ppm (Seiler et al., 1988). Copper is present in concentrations averaging about 4 ppm in limestones, 55 ppm in igneous rocks, 50 ppm in sandstones, and 45 ppm in shales.

On a global basis, the atmospheric copper flux from anthropogenic sources is approximately three times higher than its flux from natural sources. Non-ferrous metal production is the largest contribution of atmospheric copper in the United States (U.S. Environmental Protection Agency, 1987). The principal source of elevated copper levels in air is copper dust generated by copper processing operations. Other sources include tobacco smoke and stack emissions of coal burning power plants (U.S. Environmental Protection Agency, 1980). In the vicinity of copper mines or smelting works, water and pastures have been shown to be contaminated with copper (Clarke et al., 1981). The principal sources of copper in the atmosphere are (U.S. Environmental Protection Agency, 1987):

Sources	Percent Copper
o wind blown copper dust	6.5
o iron and steel production	7.4
o coal and oil combustion	4.6
o zinc smelting	3.3
o copper sulfate production	2.7
o municipal incineration	1.9
o others	2.3

Levels of copper in the atmosphere in the United States have been reported to vary from 10 to 570 ng per cubic meter, with the highest values being found

in urban areas. At the South Pole the average copper concentration in air was 0.036 ng per cubic meter (Friberg et al., 1986).

Literature describing the terrestrial fate of copper reflects widespread concern of a copper toxicity effect on soil microorganisms and fungi in association with acid deposition (Hutchinson, 1989; Tyler, 1978; and Seiler et al., 1988). Factors affecting the balance between copper in the parent rock and in the derivative soil include the degree of weathering, the nature and intensity of the soil formation, drainage, pH, oxidation reduction potential, and the amount of organic matter in the soil. Since copper in rocks is likely to be more mobile under acidic than alkaline conditions, the relation of pH to copper in the environment has been of great concern to agriculturalists and biologists. Acid soil conditions promote the solubility of copper, increase the concentration of ionic copper, and thereby change the microorganism and other animal populations, depending on their tolerance for various levels of copper in solution. Due to the variety of conditions which influence the metal's availability, the total copper content of the soils is not an accurate indication of deficiencies or excesses of copper (Seiler et al., 1988). In urbanized areas the effects of land clearing, profile distribution, and increased acid rainfall may increase copper mobilization in these soils (Tyler, 1978).

The average background concentration of copper in surface waters in the United States is less than 20 µg per liter. Some copper complexes may be metabolized, however, there is no information that biotransformation processes have a significant bearing on the aquatic fate of copper (Callahan et al., 1979). Measurements of the copper concentration in drinking water are highly variable. In one study, the mean value of copper in water samples at measurable levels was 60 µg per liter. The mean value for another study was 150 µg per liter (National

Research Council, 1980). Water that is acidic, low in hardness and alkalinity, and consequently corrosive to piping, may leach copper from drinking water pipes (U.S. Environmental Protection Agency, 1985).

There are a number of processes that affect the environmental fate of copper, including complex formation, sorption, and bioaccumulation. Copper can form complexes with humic substances, thus streams high in organic particulates may have a great deal of copper in a bound state. In addition, complexed copper is more easily absorbed by clay and other surfaces than free (hydrated) cations. The concentration of copper in water is also influenced by its affinity for hydrous iron, manganese oxides, clays, and carbonates. The presence of these substances in water will reduce levels of dissolved copper. In polluted natural waters high in organic content, effective control of dissolved copper concentrations will be dependent upon the competition between organic complexing in solution and sorption onto clay and particulate organic matter. Copper is an essential nutrient and is accumulated by all plants and animals; thus biological activity will in part determine the distribution of copper in the ecosystem. Bioconcentration factors range from 12 to 2,400 for algae, 1,000 for plants, and 200 for freshwater fish (Callahan et al., 1979).

Human Exposure

Sources of exposure to copper are from fumes, copper ore smelting and related metallurgic operations, as well as from welding and from dusts of copper metal and copper salts (Clayton and Clayton, 1981). One of the chief industrial exposures to copper from which there are potential health effects is to the fume. Fume exposures occur in copper and brass plants and in welding copper containing metals (American Conference of Governmental Industrial Hygienists, 1986).

Several reports describing copper intoxification indicate that poisoning can result from the use of copper containers for food or drink (Friberg et al., 1986). Gastrointestinal irritation can result following the drinking of carbonated water or citrus fruit juices which have been in contact with copper vessels, pipes, tubing, or valves. Such beverages are acidic enough to dissolve irritant quantities of copper (International Labour Office, 1983). Extra corporeal hemodialysis has been documented as a source of copper poisoning (Gosselin et al., 1984). A partial list of occupations from which copper exposure may occur includes (Sittig, 1981):

- asphalt makers
- battery makers
- electro platers
- fungicide workers
- gem colorers
- lithographers
- pigment makers
- rayon makers
- solderers
- wallpaper makers
- water treaters
- wood preservative workers.

Toxicity

Copper is an essential element at low levels, but has toxic effects at high doses. Toxic effects in laboratory animals and humans, resulting from acute exposure, include gastrointestinal disturbances, hemolytic anemia, kidney and liver damage, and inhibition of the enzyme glucose-6-phosphate dehydrogenase. In general, mammals are protected from the adverse effects of excessive dietary intake of copper due to homeostatic mechanisms.

The data concerning the health effects associated with copper are limited. A Lowest-Observed-Adverse-Effect-Level (LOAEL) of 5.3 mg per day was developed

for humans, based on the lowest oral dose at which gastrointestinal effects were seen.

The literature describing the toxic effects of copper in humans indicates that debate exists surrounding its chronic toxicity effects (Browning, 1969). It has been suggested that copper itself has little or no toxicity (Merck Index, 1983). Many cases of illness formerly attributed to copper are now believed to have been more probably due to a mixture with other metals, especially lead.

Inhalation of dusts and fumes of metallic copper causes congestion of the nasal mucous membranes, ulceration, perforation of the nasal septum, and pharyngeal congestion (Venugopal and Luckey, 1978). Welders exposed to copper fumes complain of sneezing, coughing, digestive disorders, and mucosal irritation in the mouth and eyes. Nasal ulcerations and bleeding occurred in workers inhaling finely divided copper metal dust. Allergic contact dermatitis is less commonly encountered (Clayton and Clayton, 1981). Hemolytic anemia has been reported in men occupationally exposed to low levels of copper and in patients undergoing hemodialysis with excess copper in the dialysis fluid from leaching copper tubing (Finelli, et. al., 1981; and U.S. Environmental Protection Agency, 1985). Exposure to copper dust causes discoloration of the skin (Seiler et al., 1988). It is generally agreed that copper itself is less toxic than its salts.

Carcinogenicity

In general, copper compounds have not resulted in mutations in microbial assays. Subcutaneous injection of copper in mice has resulted in the formation of tumors, though oral administration has not.

Health Criteria

There is no Maximum Contaminant Level (MCL) for copper under the Resource Conservation and Recovery Act (RCRA), however the Safe Drinking Water Act (SDWA) secondary standard for copper is 1 mg per liter, which is based on taste and odor considerations (Callahan et al., 1979). The Virginia State Water Control Board (SWCB) established a standard of 1 mg per liter for surface drinking water supplies and ground water protection. The Virginia SWCB standard for protection of freshwater aquatic life is 2.9 µg per liter. The Clean Water Act (CWA) Water Quality Criteria for protection of freshwater aquatic life from acute and chronic exposure are 0.018 mg per liter and 0.012 mg per liter, respectively.

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LEAD

Occurrence

Lead occurs chiefly as lead sulfide in galena. Other minerals which contain lead compounds include anglesite, cerussite, mimetite, and pyromorphite (Merck Index, 1983). Galena is a typical hydrothermal mineral of medium temperature deposits. The largest lead deposits in the United States are in the Tri-State mining district near Joplin, Missouri. There are sizable deposits of lead-bearing galena near Leadville, Colorado. Silver is a frequent by-product of lead mining (Mottana et al., 1978).

Chemical and Physical Properties

Lead is a silvery gray metal which is lustrous when freshly cut, and has a cubic crystal structure in natural occurrence. It is very soft and malleable, and easily melted, cast, rolled, and extruded. Other basic physical properties are shown in table B-1 (Merck Index, 1983). Lead is insoluble in hot or cold water (Weast, 1987). It is soluble in nitric acid and hot concentrated sulfuric acid (Merck Index, 1983).

Use

The lead mineral in most crude ores is separated from the gangue and other valuable minerals in a staged process. Occasionally the ores are sufficiently rich in lead and low in impurities to be smelted directly. The primary operations of ore processing are crushing, grinding, and concentration. Crushers are first employed to reduce the particle size of the mined ore and liberate the desired mineral from the gangue. Further size reduction is accomplished by wet

grinding in horizontal cylindrical mills containing steel balls, rods, or flint pebbles. Finally, the lead containing material is concentrated by a settling process that depends on differences in the settling rates of materials (Kirk-Othmer, 1981). A floatation technique may also be used to separate lead containing ores.

The series of processes used to produce commercial grade lead from the lead concentrate include blast furnace smelting, drossing, and refining by pyrometallurgical methods. Impurities that may still remain in lead are silver, gold, copper, tellurium, platinum metals, and bismuth. Although the concentrations of impurities may be tolerable for some lead applications, their market value encourages separation and recovery (Kirk-Othmer, 1981).

Lead is available in ingots, sheet, pipe, shot, straps, grids, rod, wire, paste and powder (Sax and Lewis, 1987). Important commercial uses of lead include the following:

- o construction material for tank linings and piping
- o equipment handling of corrosive gases and liquids
- o petroleum refining
- o manufacture of tetraethyl lead
- o radiation protection (e.g., x-ray)
- o bearing metal and alloys
- o building construction
- o manufacture of paint pigments
- o ceramics
- o plastics
- o lead oxide and antimonial lead storage batteries
- o production of ammunition

The most dominant uses of lead in 1979 were for ammunition, bearing metal, brass and bronze, cable covering, caulking lead, storage battery oxides, and anti-knock additives (Kirk-Othmer, 1981).

Environmental Fate and Behavior

The extent of occurrence of lead in the earth's crust is about 15 grams per ton. Lead enters the environment from lead-bearing minerals (Merck Index, 1983). Natural sources contributing to airborne lead are silicate dusts, volcanic emissions, forest fires, sea salt aerosols, meteoric and meteoritic smoke, and lead derived from the decay of radon (World Health Organization, 1977).

Lead is an important commercial metal in the United States. It may enter the environment during its mining, ore processing, smelting, use, recycling, or disposal. The natural sources of lead are minor compared with anthropogenic ones. Metallic lead may be released from smelting and refining plants. If released or deposited on soil, lead will be retained in the upper 2 to 5 cm of soil, especially soils with 75 percent organic matter and a pH of greater than five. While some corrosion may be expected in soil, generally an inert coat of an insoluble salt will form and limit further corrosion. It is expected to convert to more insoluble forms such as $PbSO_4$, $Pb_3(PO_4)_2$, PbS, and PbO. Lead also forms complexes with organic matter and clay minerals that limit its mobility. The amount adsorbed depends on parameters such as the availability of ligands, pH, redox conditions, salinity, iron concentration, composition of dissolved particulate matter, and sediment lead concentration (Syracuse Research Corporation, 1989).

Lead enters water from atmospheric fallout, runoff, or wastewater. Little is transferred from natural ores. Lead is a stable metal, and adherent films of protective insoluble salts form that protect the metal from further corrosion. Lead which dissolves tends to form ligands. Lead is effectively removed from the water column to the sediment by adsorption to organic matter and clay minerals, precipitation as an insoluble salt (the carbonate, or sulfate/sulfide),

and reaction with hydrous iron and manganese oxide. Under most circumstances adsorption predominates (Syracuse Research Corporation, 1989).

Precipitation can be important at relatively high pH. The amount of lead that can remain in solution in water is a function of the pH and the dissolved salt content. Much of the lead carried by river water is in the form of suspended solids. One study, of the distribution of lead between filtrate and solids in stream water from urban and rural areas, reported the ratio of lead in suspended solids to that in filtrate varied from 4 percent in rural areas to 27 percent in urban areas (U.S. Environmental Protection Agency, 1979).

The form of lead that enters the atmosphere is not known. When released to the atmosphere, lead will generally be in the form of dust, or adsorbed to particulate matter, and subject to gravitational settling. It can also be transformed to the oxide and carbonate (Chan, et. al., 1986).

Biodegradation, Abiotic Degradation and Bioconcentration

Lake sediment microorganisms are able to directly methylate certain inorganic lead compounds. Biomethylation of lead by benthic microorganisms can lead to its remobilization into the aqueous environmental compartment (Frances, 1985; Stephenson and Lester, 1987). Tetraethyl and tetramethyl lead have been shown to photodegrade at significant rates (Harrison, 1978; Pierrard, 1969).

Lead does not appear to bioconcentrate significantly in fish, but does in some shellfish such as mussels. One study indicated that lead was absorbed by the fresh water field crab, Bartelphusa guerini, through the gills and distributed to the organs. Lead, bioaccumulated over the course of the study, showed a high degree of organ specificity (Tulasi, 1987). The studies showed

the following intensities of bioconcentration factors (Schulz-Baldes et al., 1983; and Biddinger and Glass, 1984):

Organisms	Log Bioconcentration Factors (Ranges)
freshwater fish	1.38-1.65
freshwater invertebrates species	2.70-3.23
seawater bivalves, mollusks, diatoms, phytoplantation	1.24-3.40

Evidence suggests that lead uptake in fish is localized in the mucous on the epidermis, dermis, and scales, so that its lack of availability in edible portions poses no human health danger (Schulz-Baldes et al., 1983; and Biddinger and Glass, 1984).

Human Exposure

General lead exposure occurs from ambient air, especially in areas with high automotive traffic and sites near industrial sources (International Agency for Research on Cancer, 1977). However, the highest intake is from food and water. Concentrations in food may be elevated due to surface contamination of fresh fruits and vegetables. Food in soldered tin cans may contain particularly high levels of lead. Elevated levels of lead in drinking water usually result from distribution systems containing lead pipe (World Health Organization, 1977).

Several population subgroups are considered at special risk from exposure to lead. The pregnant female and fetus are at special risk for several reasons. Exposure of the fetus to elevated lead concentrations is dangerous because of its great sensitivity during development. The fetus and young children absorb more of the lead that they ingest than adults. Children ingest more lead because, during normal mouthing activity, they are apt to chew painted objects

and ingest soil (Department of Health and Human Services/Agency for Toxic Substances and Disease Registry, 1988). Exposure of a mother to lead results in the transfer of lead to the fetus, and may cause a pre-term birth, reduced birth weight, and decreased intelligence quotient (IQ) in the infant (Department of Health and Human Services/Agency for Toxic Substances and Disease Registry, 1988). Other population subgroups at special risk include:

- o individuals having genetically caused glucose-6-phosphate dehydrogenase deficiency (iron storage diseases may increase lead in the human liver) (Goyer and Mahaffey, 1972);
- o children with sickle cell anemia (Erenberg, Rinsler and Fish, 1974);
- o individuals having hemoglobin S or O thalassemia (Goyer and Mahaffey, 1972);
- o law enforcement officers subjected to toxification while firing weapons in an indoor firing range.

Toxicity

In humans, absorption of lead from the gastrointestinal tract is influenced by age and nutritional factors (Goyer, 1986). In adults, 5 to 15 percent of digested lead is absorbed, and less than 5 percent is retained. In one study, children were found to have an average net absorption of lead of 41.5 percent, and infants were reported to have an average net lead retention of 31.8 percent (Goyer, 1986).

Absorption of lead through the lungs depends on the concentration of lead in the air, volume of air respired per day, size distribution of lead-containing particles, and whether the lead is in vapor or particle form. Of the lead particles in ambient air that are deposited in the lungs, approximately 90 percent are small enough to be retained. Retained lead is efficiently absorbed into the blood through the alveoli (Goyer, 1986).

Most of the retained lead is deposited in the skeleton where it has a half-life in excess of 20 years. The lifetime accumulation of lead is estimated to be 200 mg for non-occupationally exposed workers and 500 mg for those exposed to lead in the work place.

The toxic effects of lead on the central nervous system are considered to be the most serious systemic effect in terms of human health. In children, cognitive or motor neurologic effects are of main concern, while peripheral neuropathy and nephropathy (effects on the kidney) are of concern in adults. Blood levels typically range from 30 to 50 μg per deciliter (Goyer, 1986). Blood levels of 60 to 70 μg per deciliter, and greater than 80 μg per deciliter, are associated with encephalopathy in children and adults, respectively.

In addition to central nervous system dysfunction, high blood levels of lead are associated with hematologic and renal effects. High blood lead levels may result in a shortened life-span of erythrocytes and impaired heme synthesis. Renal effects include reversible renal tubular dysfunction (associated with children following acute exposure to lead) and incurable interstitial nephropathy.

Carcinogenicity

Studies on the carcinogenicity of lead indicate that lead can induce cancer in kidneys of rodents fed high doses, but there is little evidence to indicate that lead is a human carcinogen.

Health Criteria

The following regulations and standards are in effect for lead:

Drinking water (Primary Drinking Water Standard):	0.05 mg/l
Ambient Water Quality Criteria (for protection of aquatic life):	CWA Water Quality Criteria Acute: 0.080 mg/L Chronic: 0.0032 mg/L
OSHA Standard (8-hr TWA):	0.050 mg/m^3 (inorganic lead)
ACGIH Threshold Limit Values:	0.15 mg/m^3 (inorganic lead dusts and fumes)

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MANGANESE

Occurrence

Manganese is one of the most common of the elements and is widely distributed in rocks and soils. The most common forms of manganese in rocks and soils are oxides and hydroxides in which the oxidation state of the element is +2,+3, or +4.

Chemical and Physical Properties

The solubility of manganese will primarily be controlled by the Eh, pH, and amount of dissolved carbon dioxide in the system. For a system in which the activity of dissolved carbon dioxide species is 100 mg per liter of HCO_3^-, the predominant dissolved form of manganese would be the divalent ion Mn^{+2}, at a pH near neutral (Hem 1970).

Environmental Fate and Behavior

Manganese is an essential element in plant metabolism, and it is to be expected that organic circulation of manganese can influence its occurrence in natural water. Terrestrial and aquatic species of plants are noted to be accumulators of manganese. Manganese in plant parts that die back or shed leaves becomes available for solution in runoff and soil moisture. Preliminary studies on the effects of fallen leaves on water quality of a small stream in Virginia showed that plant matter die-back could be an important contribution of manganese at times (Hem 1970).

The rate of oxidation of Mn^{+2} and precipitation of the oxidized form as MnO_2 is greatly increased by increases in pH and goes more rapidly as surface

area increases (Hem 1970). Oxidation of manganese can also be catalyzed by feldspar and other mineral surfaces.

Toxicity

In humans, chronic exposure to manganese causes degenerative changes in the central nervous system in the form of Parkinson-like disease. Modifications to liver functions can also occur. Acute exposure causes manganese pneumonitis.

In humans, manganese dusts and compounds have relatively low oral and dermal toxicity, but they can cause a variety of toxic effects after inhalation exposure. Acute exposure to very high concentrations can cause manganese pneumonitis, increased susceptibility to respiratory disease, and pathologic changes including epithelial necrosis and mononuclear proliferation. Chronic exposure is more common, but generally only occurs among persons occupationally exposed. Degenerative changes in the central nervous system are the major toxic effects. Individuals with an iron deficiency may be more susceptible to chronic poisoning.

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Occurrence

Mercury is found in rocks of all classes. Common host rocks include:

Limestone
Calcareous Shales
Sandstone
Serpentine
Chart Andesite (Soda Lime Fieldspor)
Basalt
Rhyolite

Elemental mercury occurs in some ores, however mercury is recovered almost entirely from cinnabar (mercuric sulfide) (Clayton and Clayton, 1981). Cinnabar occurs in very low temperature hydrothermal veins, or impregnations, and replacement deposits in sedimentary rocks associated with nearby igneous rocks. It is also present as a chemical deposit of hydrothermal, probably alkaline springs (Mottana et al., 1978). The mercury content of some common ores and gangue minerals, as a result of their coexistence in a deposit with cinnabar, meta cinnabar, or other mercury containing materials, is as follows (Jonasson and Boyle, 1972, as cited in National Research Council of Canada, 1979):

Ore or Gangue Material	Percent
Tetrahedrite	17.6 - 21
Grey Copper Ores	14.0
Spallerite	1.0
Wurzite	0.03
Stibnite	1.3
Realgar	2.2
Pyrite	2.0
Argonite	3.7

Mercury is also found in fossil fuels (see section on Environmental Fate and Behavior for more details).

Chemical and Physical Properties

Mercury is a heavy, mobile, liquid metal. It is silver white in color (Merck Index, 1983). The high mobility and tendency to dispersion exhibited by mercury, and the ease to which it forms alloys (amalga) with many laboratory and electrical contact metals, can cause severe corrosion problems in laboratories (Bretherick, 1985). Mercury can attack copper and copper alloy materials (National Institute for Occupational Safety and Health, 1981).

The solubility of mercury in water is 0.28 μmoles per liter at 25°C (Merck Index, 1983). Mercury is soluble in nitric acid. It is insoluble in dilute hydrochloric acid, hydrogen bromide, hydrogen iodide, and cold sulfuric acid (Weast, 1987). Mercury dissolves to some extent in lipids (American Conference of Governmental Industrial Hygienists, 1986). The physical properties of mercury are listed in table B-1.

Use

Mercury is obtained by roasting cinnabar and condensing mercury vapor. Secondary sources of mercury are from batteries, sludges, or water (Stanford Research Institute). Mercury is available in commercial, instrument, redistilled, technical, and triple distilled grades (Environment Canada, 1982). Below are listed some of the uses of mercury (Merck Index, 1983; National Research Council, 1977; Stanford Research Institute; and Farm Chemicals Handbook, 1983):

- barometers, thermometers, hydrometers, and pyrometers
- mercury arc lamps producing UV rays
- switches
- fluorescent lamps
- mirrors
- boilers
- catalyst in the oxidation of organic compounds
- extracting gold and silver from ores
- electric rectifiers
- mercury fulminate

- o cathodic electrolysis
- o anti-fouling paints
- o dry cell batteries

The consumption pattern of mercury indicates the following (Kayser, 1982):

Use	Percent Of Mercury Used
Electrical products such as dry well batteries, fluorescent light bulbs switches and other control equipment	50
Electrolytic preparation of chronic and caustic soda	25
Paint manufacture	12
Dental preparations	3
Industrial catalyst manufacture	1
General Laboratory Use	1
Pharmaceutical	0.1

Environmental Fate and Behavior

Mercury is found in rocks of all classes, including limestone, calcareous shales, sandstone, serpentine, basalt and alkaline feldspar and quartz, (Clayton and Clayton, 1981). Mercury is also found in notable quantities in fossil fuels, as shown below (Jonasson and Boyle, 1972):

Material	Mercury Content (ppb)
Coal	10 - 8,530
Coal in mercuriferious basins	20 - 300,000
Crude oils	20 - 2,000
Petroleum crudes mercuriferous belts	1,900 - 21,000
Bitumens, solid hydrocarbons and asphalts	2,000 - 900,000

One of the major sources of mercury is the natural degassing of the earth's crust (World Health Organization, 1976). Mercury is released into the environment from volcanos and hot springs.

Twenty thousand tons of mercury are released into the environment each year by human activities, such as from the combustion of fossil fuels, as well as from other industrial releases (Friberg et al., 1986). Concentrated local discharges are associated with industrial activities, and diffused discharges of mercury are associated with the combustion of fuels containing mercury impurities (Miller and Buchanan, 1979). Mercury is liberated from the working and smelting of ores of copper, gold, lead, silver, and zinc, which normally contain traces of mercury (Jonasson and Boyle, 1972). In Canada, in 1974, approximately 12 tons of mercury were discharged to the environment as a result of coal combustion. Approximately 90 percent was discharged to the air as vapor, while 9 percent was discharged as fine particulates (National Research Council of Canada, 1979). In general, industrial and domestic products, such as thermometers, batteries, and electrical switches, which account for a significant loss of mercury to the environment, ultimately become solid waste in major urban areas (National Research Council of Canada, 1979). Waterborne mercury pollution originates from sewage, metal refining operations, or most notably from chloralkali plants (National Research Council of Canada, 1979).

Mercury's two characteristics, volatility, and vulnerability to biotransformation, make consideration of its environmental fate unique.

Mercury volatility accounts for high atmospheric concentrations. Twenty to 200 μg per cubic meter are typically found near areas containing high soil levels (10 ppm), compared to a normal atmospheric concentration of 5 μg per cubic meter (Doull et al., 1980). Fifty percent of the volatile form is mercury (Hg) vapor, with a sizeable portion of the remainder being Hg (II) and methylmercury.

Mercury in the environment is deposited and revolatilized many times, with a residence time in the atmosphere of at least a few days. In the volatile phase, it can be transported hundreds of kilometers (Miller and Buchanan, 1979).

The average atmospheric concentration of mercury is estimated to be 2 to 10 μg mercury per cubic meter (U.S. Environmental Protection Agency, 1984b). The concentrations of mercury in the general atmosphere in several locations were estimated as follows (World Health Organization, 1976):

Location	mg Hg per cubic meter
USSR (general atmosphere)	10
Non industrial regions in Japan	0-14
Denver, USA (lowest ions)	2-5
San Francisco, USA	0.5-50
New York City, USA (airborne dust)	1-41
Chicago (particle bound Hg)	3-39

Mercury concentrations in the stack gas of large coal-fired power generating stations in Ontario ranged from 40 to 80 μg per cubic meter (Booth, 1971).

The approximate concentration of all forms of mercury in the earth's crust is 80 ppb (Jonasson, 1970). Mercury is predominantly bound in contaminated waterways (World Health Organization, 1976). Levels of mercury in various soils have been estimated to be the following (Jonasson and Boyle, 1972):

Soil	Mercury (ppb)
Normal soils	20-150
Soil over mercury deposits	0-2000
Normal tills glacial clay and sand	20-100
Soils, tills, the near mercury deposits	0-250
Soil horizons (normal ranges)	60-200 (humic)
	30-140
	25-150
Soil horizons (near mercury deposits - ranges)	200-1860
	140-605
	150-554

Twenty five to fifty percent of mercury in water is organic (Miller and Buchanan, 1979). In aquatic systems, mercury appears to bind to dissolved matter or fine particulates (National Research Council of Canada, 1979). Mercury can be desorbed into the water column, transported by water (bound or chelated to fine particulates or dissolved substances), and redeposited on bed sediments (National Research Council of Canada, 1979). The conversion of inorganic mercury to methyl mercury implies that recycling of mercury from sediment to water, to air, and back, could be a rapid process (Callahan et al., 1979). In the Yatsushion Sea and Minamate Bay in Japan, the croaker (Argyrosomus Argentatus) was a good indication of mercury pollution. Mercury migrated from sediment to the croaker by way of suspended particulate matter and zooplankton. Conversion from inorganic to methyl mercury occurs at the stage of zooplankton (Nishimura and Kumagai, 1983). The following distribution and respective concentrations of mercury in natural waters have been measured as follows (Jonasson and Boyle, 1972):

Water	Mercury Concentration (ppb)
Rainwater, snow	0.01 - 0.48
Normal stream, river, and lake	0.01 - 0.1
Coal mine waters (USSR)	1-10
Stream and river waters near mercury deposits	0.5-100
Oceans Seas	0.005-5.0
Hotsprings and certain mineral waters	0.01-.10
Groundwaters and mine waters near polymettallic sulfide deposits	1-1000
Oil field and other saline waters	0.1-230

The range of mercury levels in drinking water, as estimated by the U.S. Environmental Protection Agency, is 5 to 100 mg mercury per liter (U.S. Environmental Protection Agency, 1984b).

Mercury levels, measured in a number of foods and fish, are shown below:

Mercury Levels Measured in Foods (Organization for Economic Cooperation and Development, 1974)

Food	Mercury Levels in Natural Foods (mg/kg)	Mercury Levels in Abnormal Foods (mg/kg)
Tuna	0.2	10.6
Eggs	0.009	0.029
Cabbage	0.09	0.57

Mercury Levels Measured in Fish & Shellfish (U.S. Environmental Protection Agency 1984b)

Fish/Shellfish	Mercury Levels (ppm)
Tuna (canned)	0.24
Fish sticks	0.21
Shrimp	0.46
Flounder	0.10
Clams	0.05
Crabs/lobsters	0.25
Salmon	0.05
Oyster/scallops	0.04
Trout	0.42
Bass	0.21
Catfish	0.15
Sardines	0.06
Pike	0.61
Snapper	0.45
Whiting	0.05
All others classified	0.21

Concentrations of mercury in fish should not exceed 0.5 ppm (Britt and Hushon, 1976).

Mercury and its compounds occur naturally in trace amounts in plants growing in soils with low mercury concentrations (<500 ppb) (Organization for Economic Cooperation and Development, 1974). The maximum level for mercury in plant tissue is recommended at 0.5 ppm, with a maximum of 0.15 ppm in the soil. These

recommendations reflect human health effects rather than plant responses (Britt and Hushon, 1976). The levels of mercury that have been measured in several plants follow (Jonasson and Boyle, 1972):

Plant Type	Concentration Levels (ppm Hg)
Marine Plants	0.01-37
Terrestrial Plants	0-40
Terrestrial Plants in the vicinity of mercury deposits	200-30,000

Fifty parts per million of mercury in soil impairs the growth of plants. Soils with more than 1,000 ppm must be considered toxic (Environment Canada, 1982).

Mercury bioaccumulates and concentrates in the food chain. The concentrations may be as much as 10,000 times that of water (Environment Canada, 1982). Fish can accumulate mercury to very high levels because uptake is rapid and elimination is slow. Predators achieve higher concentrations than do fish lower in the food chain. In Canadian freshwaters, the highest mercury levels are found in lake trout, pike, and walleye. In the sea, high mercury concentrations are found in sharks, swordfish, tuna, and halibut (National Research Council of Canada, 1979). Bioconcentration factors of 63,000 for freshwater fish, and 10,000 for salt water fish, have been found (Sittig, 1980). Acidification of a body of water might also increase mercury residues in fish, even if no new input of mercury occurs (U.S. Environmental Protection Agency, 1984a).

Human Exposure

The diet greatly exceeds other media, including air and water, as a source of human exposure and absorption of mercury. The dominant source of mercury in

the human diet is fish and fish products (U.S. Environmental Protection Agency, 1984b). Accumulation of mercury in the terrestrial and aquatic food chains results in risks for humans mainly through the consumption of the following (World Health Organization, 1976):

fish from contaminated waters (especially predator species)
tuna fish
swordfish
large oceanic fish
seafood including mussels and crayfish
fish-eating birds and mammals
eggs of fish-eating birds

The major occupational sources of exposure to mercury are in chlor-alkali plants, in the mining and refining of mercury, processing of cinnabar, and in the manufacturing and use of liquid mercury containing materials (Clayton and Clayton, 1981).

Toxicity

Mercury has long been recognized as one of the more toxic metals. The toxicity of mercury depends to some extent on its form, as it can be part of both inorganic and organic compounds. Inorganic mercury is poorly absorbed from the gastrointestinal tract (less than 15 percent), and easily absorbed by inhalation (approximately 80 percent) in humans. Organic mercury is almost completely absorbed from the gut and is assumed to be well absorbed via inhalation (U.S. Environmental Protection Agency, 1984b). Organic mercury is metabolized to inorganic mercury in human tissues. Distribution of organic mercury is primarily to the kidney. Elimination is by biliary excretion and in the urine (U.S. Environmental Protection Agency, 1984b).

Mercury has been shown to have adverse neurological effects in humans. Organic mercury compounds are generally more neurotoxic than inorganic mercury.

In addition, the different forms of mercury can cause somewhat different neurotoxic effects initially, although both will elicit the same effects at higher doses.

Classical symptoms of mercury vapor intoxication are mental disturbances, objective tremors, and gingivitis, which have been observed following chronic occupational exposure to average air concentrations of 0.1 to 0.2 mg mercury per cubic meter (U.S. Environmental Protection Agency, 1984b). The central nervous system appears to be the primary target of organic mercury intoxication. Primary lesions include destruction of cortical cerebral neurons, and damage to the Purkinje cells and granular layer of the cerebellum. Clinical symptoms suggest damage to peripheral nerves, but histopathological documentation is lacking. Clinical symptoms include paresthesia, loss of sensation in the extremities, ataxia, construction of the visual field, and hearing impairment (World Health Organization, 1976). Central nervous system lesions similar to those in humans, and proteinuria and morphological kidney changes, have been reported in animals exposed to mercury (Koller, 1979; and U.S. Environmental Protection Agency, 1984b).

Several investigators have reported embryotoxic and teratogenic effects in experimental animals treated with organic mercury. The most common findings are neurological effects, but an increased incidence of cleft palate in mice has been reported. Brain damage, but not anatomical defects, has been reported in humans exposed prenatally to organic mercury. These epidemiological studies may not have been sensitive enough to detect possible other teratogenic effects of organic mercury in human populations (U.S. Environmental Protection Agency, 1984b).

Health Criteria

A primary drinking water standard of 2×10^{-3} mg per liter has been established under the Safe Drinking Water Act (SDWA) and adopted by the Resources Conservation and Recovery Act (RCRA) program. The proposed Maximum Contaminant Level Goal (MCLG) is 2×10^{-3} mg per liter. The Virginia State Water Control Board (SWCB) adopted the SDWA MCL as a standard for mercury in surface water. The SWCB ground water standard is 5×10^{-5} mg per liter. The Clean Water Act (CWA) Water Quality Criteria have been issued for protection of human health and freshwater aquatic life. With respect to protection of human health from drinking water and fish consumption, the level has been set at 14×10^{-5} mg per liter, and for just from fish consumption, the level is 15×10^{-5} mg per liter. The CWA Water Quality Criteria for protection of freshwater aquatic life from acute and chronic exposure are 24×10^{-4} mg per liter and 12×10^{-6} mg per liter, respectively.

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NICKEL

Occurrence

Nickel is abundant in the earth's crust. It is found in many ores as sulfides, arsenides, antimonides, oxides, and silicates. Primary nickel is recovered from mainly pentlandite $(Fe, Ni)_9S_8$ and pyrrhotite $Fe_{(1-x)}S$ (Mottana et al., 1978). Nickel forms complexes with sulfate, chloride, nitrate, carbonate, oxide, hydroxide, and with organic ligands. Nickel may exist in numerous soluble and insoluble forms depending upon chemical and physical properties of the water.

Chemical and Physical Properties

Nickel is an odorless, silvery metal. It is insoluble in water and ammonia. Physical properties of nickel are listed in table B-1. It is soluble in dilute nitric acid, and slightly soluble in hydrochloric and sulfuric acids (Weast, 1987). Nickel forms compounds with sulfate, chloride, nitrate, carbonate, oxide, hydroxide, and with organic ligands. Under aerobic conditions and below pH 9, the nickel compounds of hydroxide, carbonate, sulfate, and naturally occurring organic ligands are sufficiently soluble to maintain aqueous Ni^{+2} concentrations above 60 μg per liter (Callahan et al., 1979). Hydrolysis of aqueous nickel to the hydroxide is significant only under basic conditions.

Use

Nickel is used primarily in the production of steels and alloys, and in electroplating (Syracuse Research Corporation, 1987). Primary nickel is recovered mainly from pentlandite $(Fe, Ni)_9S_8$ and pyrrhotite $Fe_{(1-x)}S$. More

nickel is recovered from metal scraps than is obtained from both domestic and imported ore combined (Mottana et al., 1978).

There are several methods by which nickel is produced. In one method it is recovered from nickel sulfide ores by crushing and concentrating the material by floatation and magnetic separation. The material is then roasted and smelted to the oxide, or reduced through hydrometallurgy by formation to a salt solution and subsequent reduction to the metal (Stanford Research Institute). High purity nickel can be obtained from nickel oxide using hydrogen gas (Merck Index, 1983).

Nickel is also produced by reaction of the nickel ore with carbon monoxide to form nickel carbonyl gas, which is decomposed by heat to obtain pure finely divided nickel (Stanford Research Institute). Recovery of nickel is also accomplished by ion exchange. Cyanide is often used in this process, and the nickel regenerant can be recovered by precipitation (Patterson, 1985). Nickel is manufactured as pellets, shot, sponge, powder, spray powder, "nickel flour", and as a high density grade compound for electronics (Sax and Lewis, 1987).

Nickel is used extensively for making stainless steel and other corrosion resistant alloys, and in tubing made of copper nickel alloy. It is used in making nickel steel armor plate and burglar proof vaults. Nickel added to glass gives it a green color (Weast, 1987). Nickel is used as a component of the following:

- ferrous and nonferrous alloys
- permanent magnets
- ceramics
- batteries and fuel cells
- surgical and dental prostheses
- silver and coins
- lightning rod tips
- spark plugs
- machinery parts
- nickel chrome resistance wire.

Nickel is also used in the synthesis of acrylic esters, as an anodic inhibitor in cooling towers, and as a catalyst in the hydrogenation of fats and oils.

Environmental Fate and Behavior

The average concentration of nickel in the earth's crust is 60-90 mg per kilogram (National Research Council of Canada, 1981). There is evidence that pure nickel powders are deposited as meteoritic dust from the stratosphere (International Agency for Research on Cancer, 1973). Artificial sources, such as from the increased usage of nickel powder, contribute to increases in the atmospheric concentration of nickel and to increases in the nickel levels already present in foodstuffs. Increases also result from the alloys in food processing equipment, in milling of flour, and in catalytic hydrogenation of fats and oils by nickel catalysts (U.S. Environmental Protection Agency, 1980).

The atmosphere is a major conduit for nickel as particulate matter. Natural sources and anthropogenic activity contribute to the atmospheric load. Natural sources contributing to airborne particles containing nickel include soil, seawater, volcanoes, forest fires, and vegetation. Dry and wet precipitation processes remove particulate material from the atmosphere, transferring it to soils and waters. Soil borne nickel may enter waters by surface run-off or by percolation into ground water. An accumulation of nickel, ranging from 600 to 6,455 mg per kilogram in the organic soil of a farm, has been demonstrated resulting from aerial fallout from a nickel smelter (U.S. Environmental Protection Agency, 1983). Uncontaminated agricultural soils in Canada generally contain less than 30 mg of nickel per kilogram. Soils derived from serpentine rock may contain up to 25,000 mg nickel per kilogram (National Research Council of Canada, 1981).

Once nickel is in surface and ground water systems, physical and chemical interactions occur that will determine its fate and that of its constituents (U.S. Environmental Protection Agency, 1983). Nickel exists in numerous soluble and insoluble forms depending upon chemical and physical properties of the water. The distribution of nickel in the major rivers of the world is estimated as follows: 0.5 percent in solution, 3.1 percent absorbed, 47 percent as a precipitated coating, 14.9 percent in organic solids, and 34.4 percent as crystalline material (Syracuse Research Corporation, 1987). The mobility of nickel in aquatic media is controlled by complexation, precipitation/dissolution, adsorption/desorption, and oxidation/reduction reactions (Syracuse Research Corporation, 1987). Limited data suggest that, in pristine environments, nickel may exist primarily as hexahydrate ions that are subsequently coprecipitated or sorbed by hydrous oxides of iron, silica, and manganese, leading to decreases in mobility and bio-availability. In more organo-rich polluted waters, organic materials will keep nickel solubilized by complexation, and approximately half of the nickel may exist as simple inorganic salts and half as stable organic complexes (e.g., with humic acids). The results of one study indicate that although amorphous oxides of iron and manganese generally control the mobility of nickel in aqueous media, variations in such properties as sulfate concentration, pH, and iron oxide surface areas could affect the mobility of nickel (Syracuse Research Corporation, 1987). No data were found to suggest that nickel is involved in any biological transformation in the aquatic environment.

No information was available describing levels of nickel in plants, fish, animals, or milk.

Human Exposure

The toxicologically important routes of entry for nickel are inhalation, ingestion, and percutaneous exposure. Occupational groups such as nickel workers and others handling nickel comprise the individuals at highest risk. The highest risk of mortality from cancer of the respiratory tract is found among nickel mine workers involved in roasting, smelting, and electrolysis. Other occupational exposures include fabrication of parts and structures by welding alloys, manufacturing of nickel cadmium batteries, constructing nickel molds in glass bottle factories, and spraying nickel containing paints.

Toxicity

Growing evidence suggests that the nickel (III) and nickel (II) redox couple facilitates oxygen free radical reactions, which may represent one of the molecular mechanisms for the genotoxicity and carcinogenicity of nickel compounds (Sunderman, 1987). Nickel, nickel subsulfide, and nickel oxide are generated in relatively large particle sizes during the mining of nickel, and are thus associated with damage to the nasal mucosa (Doull et al., 1986). Nickel metal is also a well known cause of contact dermatitis in sensitized individuals (Grant, 1986). Other potential symptoms of exposure include sensitive dermatitis, allergic asthma, the occurrence of cavities in the nasal passages, and pneumonitis. Nickel dermatitis has been demonstrated in women associated with wearing certain jewelry, wrist watches, and clothing buckles (Schubert, 1987).

Carcinogenicity

Nickel is considered to be carcinogenic to humans and animals (U.S. Environmental Protection Agency, 1980). The occurrence of statistically significant excesses of nasal cavity and lung cancers in nickel refinery workers has been conclusively demonstrated in a number of epidemiologic studies. It is believed that these excess risks are primarily due to inhalation of metallic nickel, nickel subsulfide, nickel oxide, and nickel carbonyl. These conclusions have been supported by observations in experimental animals, and the International Agency for Research on Cancer (IARC) has concluded that it is likely that some forms of nickel are carcinogenic to man by inhalation (International Agency for Research on Cancer, 1982). However, because simultaneous exposure to several nickel compounds usually occurs in the work place, it has been difficult to determine which specific compounds are carcinogenic under these conditions. In addition, there is no evidence that nickel is carcinogenic in humans when ingested, and the U.S. Environmental Protection Agency does not consider nickel to be carcinogenic by ingestion (U.S. Environmental Protection Agency, 1985). However, the National Institute for Occupational Safety and Health has recommended that nickel be treated as a potential human carcinogen.

Health Criteria

Maximum Contaminant Levels (MCLs) have not been established for nickel under the Resource Conservation and Recovery Act (RCRA) or the Safe Drinking Water Act (SDWA). The Virginia State Water Control Board (SWCB) bases their standard for protection of freshwater aquatic life on water hardness. The Clean Water Act (CWA) Water Quality Criteria for the protection of human health have

been established for surface water where fish and water are ingested (1.3×10^{-10} mg per liter) and where fish only are ingested (0.1 mg per liter). The CWA Water Quality Criteria for protection of freshwater aquatic life from acute and chronic exposure are 1.4 mg per liter and 0.16 mg per liter, respectively.

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SELENIUM

Occurrence

Selenium constitutes about 0.09 ppm of the earth's crust. It occurs in nature usually in the sulfide ores of the heavy metals (Merck Index, 1983). There are no true deposits of selenium anywhere, and it cannot economically be recovered from the earth directly (International Labour Office, 1983). Selenium is found in small quantities in pyrite, clausthalite (PbSe), naumannite (Ag Pb)Se), tiemannite (HgSe) and selenosulfur (Merck Index, 1983).

Chemical and Physical Properties

Selenium exists in several allotrophic forms. Three of these forms are generally recognized. Selenium can be prepared in crystalline or amorphous forms. The monoclinic crystalline form is deep red. The hexagonal crystalline form, the most stable variety, is a metallic gray. The amorphous form is red in powder form or black in vitreous form (Weast, 1987).

Selenium is insoluble in water and soluble in sulfuric acid (Weast, 1987). It combines directly with hydrogen and the halogens (except iodine) (Merck Index, 1983). Selenium has the unique properties of conducting electricity and rectifying alternating current (Merck Index, 1983). It exhibits both photovoltaic action where light is converted directly to electricity, and photoconductive action where electrical resistance decreases with increased illumination (Weast, 1987). Other physical properties of selenium are shown in table B-1.

Use

Most of the world's selenium is provided by the anode muds from electrolytic copper refineries. Selenium is recovered by roasting the muds with soda or sulfuric acid, or by smelting them. (Weast, 1987). Selenium is available in commercial, high purity, and ultra high purity grades. Listed below are some uses of selenium:

- o decoloring agent for glass
- o toning baths in photography
- o pigment in the manufacture of ruby pink, orange, or red-colored glass
- o metallic base in making electrodes for arc lights
- o electrical instruments and apparatus
- o rectifier in radio and television sets
- o selenium photocells
- o semiconductor fusion mixtures
- o selenium solar cells.
- o vulcanizing agent in rubber processing
- o catalyst in the determination of nitrogen by the Kjeldahl method
- o dehydrogenation of organic compounds

The relative proportions of the end uses of selenium are described below (Bureau of Mines, 1986):

End uses	Percent
Electronic and photocopier components	35
Glass and manufacturing	30
Chemical and pigments	25
Other	10

Environmental Fate and Behavior

The earth's crust is said to have an average selenium concentration of 0.03 to 0.08 ppm. The following are estimated selenium concentrations in geological materials (Wilber, 1980):

Material	ppm
igneous rock	0.05
shales	0-0.6
sandstones	0.0-0.05
limestones	0.08
soils	0.2

Selenium accompanies sulfur in volcanic effluents. Soils in the neighborhood of volcanos tend to have enriched amounts of selenium (Wilber, 1980). Selenium is the most highly enriched element in coal, being present as an organoselenium compound, a chelated species, or as an adsorbed element (U.S. Department of Interior, 1985). It is present in the major oceans and in inland waters, resulting in the presence of selenium in drinking water.

Various industries discharge small amounts of selenium into air (Wilber, 1980). The atmospheric level of selenium in most urban regions ranges from 0.1. to 10 ng per cubic meter. A greater part of the atmospheric selenium is bound to fly ash and to suspended particles that contain from 1.4 to 11 μg per gram, and from 1 to 110 μg per gram, respectively. Up to 90 percent of the selenium content in ambient air is emitted during the burning of fossil fuels.

The forms of selenium in the soil depend on soil pH and the redox potential. At equilibrium, most soil selenium should be elemental selenium (Parr et al., 1983). In areas of acid or neutral soils, the amount of biologically available selenium should steadily decline. The decline may be accelerated by agricultural or industrial practices. In dry areas with alkaline soils and oxidizing conditions, elemental selenium and selenides, in rocks and volcanic soils, may oxidize sufficiently to maintain the availability of biologically active selenium (U.S. Department of Interior, 1985).

Selenium is a nonmetallic element. Thermodynamic data show that above a pH of 6.6 in aerated water, the stable form of selenium is the anion selenite, SeO_3^{-2}. Under mildly reducing conditions, however, the equilibrium species is elemental selenium, which presumably has a low solubility (Hem, 1970). It is to be expected that selenium concentrations in water in a reducing environment would be very low.

In aerobic waters, selenium is present either in the selenite (H_2SeO_3, $HSeO_3^-$, SeO_3^{-2}) or selenate forms (H_2SeO_4, $HSeO_4^-$, SeO_4^{-2}), with oxidation states of +4 or +6. These chemical species are very soluble, and most of the selenium discharged into the aquatic environment is probably transported in these forms to the oceans. Under reducing conditions, selenium can form metal selenides either by direct reaction with metals or through substitution for sulfur in metal sulfides. Most of the metal selenides, however, have a very low solubility in water.

Selenium has a sorptive affinity for hydrous metal oxides, clays, and organic materials. Sorption by bed sediments or suspended solids can result in enrichment of selenium concentrations in the bed sediments. Sorption or precipitation with hydrous iron oxides is probably the major controlling factor on mobility of selenium in aerobic waters.

Selenium can be methylated by a variety of organisms, including benthic microflora. In a reducing environment, hydrogen selenide (H_2Se) may be formed. Both the methylated forms and hydrogen selenide are volatile and may escape to the atmosphere. Formation of volatile selenium compounds in the sediments can remobilize sorbed selenium.

Human Exposure

Selenium, sodium selenite, sodium selenate, or selenium dioxide can affect the body by inhalation, dermal or eye contact, or by ingestion (National Institute for Occupational Safety and Health, 1981). Selenium dioxide is the primary compound involved with most industrial exposures to the element since the oxide is formed when selenium is heated. The dioxide itself forms selenious

acid with water or sweat, and this acid is an irritant (Clayton and Clayton, 1981).

Exposure to selenium may occur in the following occupations: (Wilber, 1980).

- o arc light electrode manufacturing
- o copper smelter industry
- o electric rectifier production
- o glass production
- o photographic chemical manufacturing
- o plastic production
- o pyrite roaster industry
- o rubber manufacturing
- o semiconductor industry
- o sulfuric acid manufacturing
- o textile manufacturing

Toxicity

Selenium is an essential element in other animals, and probably in humans (U.S. Environmental Protection Agency, 1980; and U.S. Environmental Protection Agency, 1984). However, exposure to amounts only slightly above the required levels can produce acute and chronic toxic effects. Acute toxicities of selenium compounds vary greatly, while the chronic effects of most forms are similar. Acute effects include degeneration of the liver, kidneys, myocardia, hemorrhages in the digestive tract, and brain damage. Eye, nose, and throat irritation may also occur with inhalation exposure. Chronic toxicity in humans appears to occur only in areas where foods containing excessive concentrations of selenium are ingested. Signs of chronic intoxication include depression, nervousness, dermatitis, gastrointestinal disturbances, dental caries and discoloration, lassitude, and partial loss of hair and nails.

Carcinogenicity

There is no evidence that selenium is carcinogenic in humans (U.S. Environmental Protection Agency, 1984). Selenium has been tested by the oral route in experimental animals, but the available data are insufficient to allow unequivocal evaluation of its carcinogenic potential. Several studies have shown that selenium may actually reduce the incidence of tumors under certain conditions. Mutagenicity and reproductive effects have not been adequately investigated (U.S. Environmental Protection Agency, 1984). Various studies on animals have demonstrated teratogenic effects of selenium at high doses (U.S. Environmental Protection Agency, 1985).

Health Criteria

The Resource Conservation and Recovery Act (RCRA) Maximum Contaminant Level (MCL) for selenium is 0.01 mg per liter. The proposed Safe Drinking Water Act (SDWA) MCL and the MCL Goal (MCLG) are 0.05 mg per liter. The Virginia State Water Control Board (SWCB) adopted the original SDWA MCL of 0.01 mg per liter for their surface and ground water standards. The Clean Water Act (CWA) Water Quality Criteria for acute and chronic protection of freshwater aquatic life are 0.26 mg per liter and 0.035 mg per liter, respectively. The CWA Water Quality Criteria for protection of human health from drinking water and consuming fish, and from fish consumption alone, are both 0.01 mg per liter.

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VANADIUM

Occurrence

Vanadium is a naturally occurring metal known to occur in over 65 different minerals, including patronite, vanadinite, roscoelite, and carnotite. It occurs in an oxidized form usually as metal vanadate. It is also found in trace amounts in fossil fuels (National Research Council, 1977).

Chemical and Physical Properties

Vanadium is a light-grey or white, lustrous powder, or it can be found in fused hard lumps. Basic properties are shown in table B-1. It is relatively insoluble in water, hydrochloric acid, and alkali solutions (Merck Index, 1983). However, it is soluble in aqua reagent, sulfuric acid, nitric acid, and hydrogen fluoride (Weast, 1979).

Use

Vanadium is used in the metal industry as an alloying compound for the production of steel, copper, aluminum, and titanium alloys. Vanadium compounds are also used as target material in X-rays, and as catalysts for sulfuric acid and synthetic rubber production.

Vanadium is produced by roasting vanadium-containing ores, and by thermal decomposition of the iodide. It is found in petroleum residues in oil refineries, in slag from the production of ferrovanadium, or in soot from oil burning (Sittig, 1985).

Environmental Fate and Behavior

Vanadium enters the environment from mining operations and refineries, and from the use of petroleum products, as well as from the absorption by plants from naturally occurring sources.

Aqueous species of vanadium that might be stable in water include both anionic and cationic forms of V^{+3}, V^{+4}, and V^{+5}. Many of these possible forms are readily soluble, although V^{+3} and V^{+4} species have a minimum solubility close to 1 μg per liter near neutral pH and under mildly reducing conditions.

Vanadium should be most soluble in surface water or ground water containing a measurable concentration of oxygen. Concentrations as high as 70 μg per liter have been reported in some ground water used for public supplies (Hem, 1970). The concentrations generally observed in surface water seem to be considerably lower.

Biodegradation and Abiotic Degradation

No information is available concerning the biodegradation or abiotic degradation of vanadium.

Human Exposure

Human exposure to vanadium is through either ingestion or respiration. Vanadium is common in many foods. Significant amounts are found in milk, seafood, cereals, and vegetables. Vanadium has a natural affinity for fats and oils; therefore, foods high in fats and oils typically have the highest concentrations of vanadium. Municipal water supplies also contain an average of one to six ppb vanadium (Doull et al., 1980). Urban air generally contains levels of vanadium due to the use of petroleum products from refineries.

Toxicity

When ingested, vanadium is considered nontoxic as a metal (Hawley, 1977). A relatively large amount of vanadium enters the environment from man's activities, but no widespread detrimental effects have been identified. Presumably, humans do not store or accumulate vanadium in toxic amounts (National Research Council, 1977). Most ingested vanadium remains unabsorbed in the gastrointestinal tract and is excreted (National Research Council, 1980). The only reported biological effect of ingested vanadium is where vanadium was thought to have caused an alteration in sulfur metabolism as shown by a reduction in the sh content of the liver and an increased turnover of protein sulfur (Clayton and Clayton, 1981).

Incidents of short-term and long-term occupational exposures to vanadium dust have been reported. Effects consist of respiratory irritation, including coughing, wheezing, breathing difficulties, bronchitis, and chest pains; eye irritation; possible skin irritation; and greenish black discoloration of the tongue (National Institute for Occupational Safety and Health, 1977; and National Academy of Sciences, 1974). In persons who recover from acute vanadium exposure, persistent bronchitis resembling asthma, as well as labored breathing, can occur (National Institute for Occupational Safety and Health, 1977). According to Casarett and Doull (1980), concentrations of vanadium in the lung increase up to age 40. The increases are due to the accumulation of insoluble particles in the lung.

Respiratory changes, similar to those of bronchopneumonia, are the principle toxic effects observed in experimental animals exposed by inhalation of vanadium

dust (Browning, 1969). Other effects include fatty acid degeneration of the liver and kidneys, hemorrhage, and bone marrow changes (Browning, 1969).

Carcinogenicity

The carcinogenicity potential of vanadium was tested by administering vanadyl sulfate at 19.8 mg of vanadium per 100 g body weight for the lifespan of white Swiss mice. No evidence of carcinogenicity was suggested in this study (National Institute for Occupational Safety and Health, 1977).

Health Criteria

The only requirement available for vanadium is a Threshold Limit Value (TLV). The TLV-Time Weighted Average (TWA) is 0.05 mg per cubic meter (for V_2O_5 respirable dust and fume) (American Conference of Governmental Industrial Hygienists, 1988).

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ZINC

Occurrence

The main ore for zinc production is sphalerite, which is zinc iron sulfide. Zinc also occurs in a number of other minerals, including smithsonite, zincite, willemite, and franklinite (Merck Index, 1983). One of the world's main deposits is in the Tri-State mining district of Missouri, Kansas, and Oklahoma (Mottana et al., 1978). The average concentration of zinc in the earths' crust is estimated at 40 mg per kilogram (Friberg et al., 1986).

Chemical and Physical Properties

Zinc is a bluish white lustrous metal (Merck Index, 1983). The metal is insoluble in cold and hot water. It is soluble in acid, (e.g. acetic acid) and alkalines (Weast, 1987). Zinc is a fair conductor of electricity. When heated to 100 to 150°C, zinc becomes malleable; at 210°C, it becomes brittle and pulverizable (Merck Index, 1983). Other physical properties of zinc are listed in table B-1.

The element has an atomic number of 30 and atomic weight of 65.38. Zinc has a valence of +2 in aqueous solution. Compounds of zinc with common surface water ligands are soluble in neutral and acidic solutions; therefore, zinc is readily transported in most natural waters, and is one of the most mobile of the heavy metals. Divalent zinc substitutes for magnesium in the silicate minerals of igneous rocks; thus, weathering of zinc-containing bedrock gives rise to Zn^{+2} in solution (Callahan et al., 1979).

Use

Zinc is manufactured by roasting zinc ore, then leaching it from the roasted material with sulfuric acid to form a zinc sulfate solution. The solution is electrolyzed in cells to deposit zinc on cathodes (Sax and Lewis, 1987). The use of zinc is extensive throughout the metal industry. The following is a list of zinc's uses:

- o used in bronze, glass, gold, magnesium, and aluminum alloys
- o printing plates
- o railroad car linings
- o extracting gold by cyanide process
- o bleaching bone glue
- o purifying fats for soaps
- o negative electrode in alkaline batteries
- o paper defoxing
- o galvanizing coating on steel wires

The most extensive uses of zinc are as zinc-based alloys, for galvanizing, and in brass products (Bureau of Mines, 1989; and Sanford Research Institute).

Environmental Fate and Behavior

During blasting or crushing of zinc ore, moderate losses of zinc to the atmosphere occur. Treatment of the crushed material by means of wet floatation may result in aqueous emissions. During smelting there are often large air emissions. The total zinc emissions to the atmosphere from smelting during 1969 were estimated at 50,000 tons (Friberg et al., 1986). Zinc oxide fumes may also be produced secondary to torch welding and cutting of zinc containing galvanizing material. These processes yield a dispersion into the atmosphere of micron-size zinc oxide particles. However, significant contamination of soil is only seen at point sources (e.g., approximately 50 times higher concentrations of zinc

were found in soil close to smelters compared with the control area) (Friberg et al., 1986).

Most of the zinc introduced into the aquatic environment is partitioned into the sediments by sorption onto hydrous iron and manganese oxides, clay minerals, and organic materials. Precipitation of the sulfide is an important controlling factor on the mobility of zinc in a redox environment. Precipitation of the hydroxide, carbonate, or basic sulfate can occur where zinc is present in high concentrations.

Since zinc is an essential nutrient, it is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,000 μg per liter, and appears to be less toxic in harder water. Chronic toxicity values range from 47 to 852 μg per liter, and appear to be relatively unaffected by water hardness.

Normal levels of zinc range from 10 to 1,000 mg per kilogram in most crops and pastures (Friberg et al., 1986). The average concentration of zinc in prepared food composites of fish is 32.0 ppm (Kirk-Othmer, 1978). The average normal levels of zinc in cattle and levels measured in cattle suffering from zinc poisoning, based on their organ distributions, are listed below (Clark et al., 1981):

	Average Normal Levels (ppm)	Levels in Animals with Zinc Poisoning (ppm)
Liver	135	2,000
Kidneys	80	670
Feces	200	3,740
Serum	0.14	0.515

Human milk contains about 3 mg per liter. A study indicated that the average daily intake of zinc in different areas was in the order of 5 to 22 mg per day (Friberg et al., 1986).

Human Exposure

Exposure to zinc fumes, such as to zinc oxide, is a significant risk in zinc smelting, in the manufacture of zinc oxide powder, in the production of brass, as well as in the melting of galvanized iron (Friberg et al., 1986). Several effects result from industrial exposure to zinc, but that of zinc fume fever is most commonly described and best documented. It has been concluded that attacks of the fever do not result from concentrations below 15 mg per cubic meter. This concentration was recommended as the Threshold Limit Value (TLV) for a number of years (American Conference of Government Industrial Hygienists, 1986). Typically, brass foundry workers exposed to fumes containing zinc experience an unusual metallic taste, which is accompanied by dryness and irritation of the throat, with coughing and labored breathing, weakness, pains in the muscles and joints, and a high fever which is followed by profuse sweating. Recovery occurs within 24 to 28 hours after an attack, provided exposure is discontinued. Gastro-intestinal disturbances and localized dermatitis may also occur after exposure to zinc oxide.

Zinc poisoning occurs accidentally from the intake of pesticides or inadvertent therapeutic use of heavy doses of zinc salts. Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Zinc is an essential mineral in the human diet, and average concentrations of zinc have been established in a number of foods. However, high levels of zinc in the diet have been shown to retard growth and produce defective mineralization.

Adsorption, Distribution, and Excretion

The major part of the total body zinc in humans is found in the muscle (60 percent) and the bone (30 percent), respectively. The highest concentration of zinc is found in the prostate, followed by bone and muscle (U.S. Environmental Protection Agency, 1980). Zinc is mainly excreted via the gastrointestinal tract. Urinary excretion of zinc is relatively small. With extreme heat or exercise, much larger quantities may be excreted in sweat. Zinc is also excreted via hair and milk, and in the female there is placental transfer to the fetus (U.S. Environmental Protection Agency, 1980).

Toxicity

Zinc is an essential element and is present in a number of metalloenzymes. Studies which have been performed on organo-zinc complexes failed to demonstrate teratogenic effects (Stokinger, 1981).

Zinc salts of strong acids are astringent and corrosive. Upon ingestion they act as emetics and cause symptoms of fever, nausea, vomiting, stomach cramps, and diarrhea.

Zinc chloride is caustic and causes severe and occasionally fatal irritation of the epithelial lining of the trachea and bronchi. Acute interstitial fibrosis of the lung occurred in one fatal case following inhalation of $ZnCl_2$ smoke from a smoke generator (Milliken et al., 1963).

Chronic administration of 0.5 to 34.4 mg zinc oxide per day for periods of one month to one year failed to produce signs of toxicity in rats (Stokinger, 1981). In another study, 0.1 percent zinc was tolerated in the diet of rats, but more than 0.5 percent zinc reduced their capacity to reproduce, and diets

of one percent zinc inhibited growth and caused severe anemia and death (Sutton and Nelson, 1937).

Zinc poisoning resulted from drinking water from galvanized pipes or eating from galvanized iron vessels (National Research Council, 1977).

Carcinogenicity

There is no evidence that zinc is carcinogenic.

Health Criteria

A secondary standard of 5.0 mg per liter has been set under the Safe Drinking Water Act (SDWA) and adopted by the Virginia State Water Control Board (SWCB) as a surface water standard. The SWCB standard for protection of aquatic life is 0.047 mg per liter, and the ground water standard is 0.05 mg per liter.

Under the Clean Water Act (CWA), the Water Quality Criteria for protection of aquatic life for freshwater (acute) is 0.13 mg per liter, and 0.11 mg per liter for freshwater (chronic).

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TABLE B-1

Physical Properties of FPRQ Indicator Chemicals
Westinghouse Project No. 4112-88-907B

Compound	Molecular Weight	Melting Point	Boiling Point	Vapor Pressure	Density	Surface Tension
Antimony	121.75	631°	1635°	1 MM Hg @ 886°C	6.69 @ 20°C	ND
Arsenic	74.92	817°C (28 atm)	ND	1 MM Hg @ 372°C	5.73 @ 14°C	ND
Barium	137.33	725°C	1640°C	10 MM Hg 1049°C	3.51 @ 20°C	24 dynes/cm at 720°C in argon
Benzene	78.11	5.5°C	80.1°C	100 MM Hg @ 26.1°C	ND	28.9 dynes/cm @ 20°C
Cadmium	112.41	321°C	765°C	1 MM Hg @ 394°C	8.65 @ 25°C	ND
Copper	63.55	1083°C	2595°C	1 MM Hg @ 1628°C	8.99	ND
Lead	207.20	327°C	1740°C	1.77 MM Hg @ 1000°C	11.34 @ 20°C	ND
Mercury	200.59	-38.87°C	356.72°C	0.0018 MM Hg @ 25°C	13.534 @ 25°C	484 dynes/cm at 25°C
Nickel	58.70	1455°C	2730°C	1 MM Hg @ 1810°C	8.9	ND
Selenium	78.96	170-217°C	690°C	>0.001 MM Hg @ 20°C	4.26-4.81	ND
Vanadium	50.94	1917°C	3380°C	ND	6.11 @ 18.7°C	ND
Zinc	65.38	419.5°C	908°C	1 MM Hg @ 487°C	7.14 @ 25°C	ND

ND = No data available

APPENDIX C

Hazard Risk and Environmental Fate Tables

TABLE C-1

Risk Assessment Data
Westinghouse Project No. 4112-88-907B

Compound	Noncarcinogen Severity Rating	RfD mg/kg/day	NOEL/NOAEL mg/kg/day	LOAEL mg/kg/day	Effect
Antimony	10(oral) 8(Inhal)	4E-4(oral) 3E-3(inhal.)	0.5 mg/m^3	N/A	Myocardial damage
Arsenic	9(oral) 9(inhal)	pending (oral) N/A(inhal.)	N/A	N/A	Pronounced pathologic changes
Barium	10(oral) 9(inhal)	5E-2(oral) N/A(inhal.)	0.51	5.1	Systolic blood pressure
Benzene	5(oral) 10(inhal)	pending (oral & inhal)	2.35 (10 day Health Adv. 10kg child)	N/A	Reversible cellular changes
Cadmium	10(oral) 8(inhal)	5E-4 (oral-water) 1E-3 (inhal.-food) pending (inhal)	0.005 0.01	N/A N/A	Significant proteimuria
Copper	5(oral) 5(inhal)	N/A (oral & inhal)	N/A	N/A	Reversible cellular changes
Lead	10(oral) 10(inhal)	Not Appropriate (oral)	N/A	N/A	Changes in blood enzymes
Mercury (inorg)	7(oral) 8(inhal)	pending (oral & inhal.)	N/A	N/A	Decrement in organ function
Nickel	10(oral) 10(inhal)	2E-2(oral) pending (inhal.)	5	50	Decreased body and organ weights
Selenium	10(oral) 10(inhal)	3E-3 (oral-water) N/A(inhal.)	N/A	0.046	Selenosis
Vanadium	1(oral) 1(inhal)	9E-3	0.89	N/A	Decreased cysteine in hair
Zinc	8(oral) 8(inhal)	3E-4(oral) zinc phosphide	N/A	3.48	Reduced food intake and body weight

TABLE C-1 (Continued)

Risk Assessment Data
Westinghouse Project No. 4112-88-907B

Compound	Carcinogen Classification	Unit Risk oral($\mu g/L$) inhal($\mu g/m^3$)	Slope Factor (PF) mg/kg/day	1/1E+4 Risk (μ/L) $\mu g/m^3$	1/1E+6 Risk (oral) (inhal.)	Effect
Antimony	--					
Arsenic	A(oral)	5E-5(oral)				skin cancer
	A(inhal)	4.3E-3(inhal.)	5.0E+1	2E-2	2E-4	lung cancer
Barium	--					
Benzene	A(oral)	8.3E-7(oral)	2.9E-2	1E+2	1E+0	nonlymphocytic
	A(inhal)	8.3E-6(inhal.)	2.9E-2	1E+1	1E-1	leukemia
Cadmium	B1(inhal)	9.2E-2(inhal.)	3.4E-4	6E-2	6E-4	lung cancer
Copper	--					
Lead	--					
Mercury	--					
Nickel	A(oral)	N/A(oral)				
	A(inhal)	N/A(inhal.)				
Selenium	--					
Vanadium	--					
Zinc	--					

Source - U.S. EPA Integrated Risk Information System (IRIS)

Abbreviations

N/A: Not Available
NOEL: No Observable Effect Level
NOAEL: No-Observed-Adverse-Effect-Level
LOAEL: Lowest-Observed-Adverse-Effect-Level
RfD: Reference Dose
inhal.: Inhalation Route of Exposure
oral: Oral Route of Exposure

TABLE C-2

Environmental Fate and Behavior of the Twelve Indicator Chemicals
Westinghouse Project No. 4112-88-907B

Chemical	Bioaccumulation	Adsorption	Biodegradation
Antimony	Data indicate bioaccumulation in shellfish (especially in shell portion).	No data available	No data available
Arsenic	Data indicate bioaccumulation from analysis of animal hair. Data show high content in fat fraction of fish, though fish generally contain less than other aquatic organisms.	Adsorption likely	No data available
Barium	No data available	Background levels indicate concentrations ranging from 100 to 3,000 ppm. Adsorption likely.	No data available
Benzene	log K_{ow} = 2.12 Low Bioaccumulation potential.	Adsorption processes may not be significant. Rapid volatilization near the surface.	Can be utilized as sole source of carbon by several bioorganisms-biodegradable at slow rate (24% and 47% after one and ten weeks respectively).
Cadmium	Data indicate accumulation by organisms at all trophic levels	Relatively mobile in aquatic environment. Sorption increasingly effective as pH increases. Increased carbon content allows complexation.	No data available
Copper	Bioconcentration factors range from 12 to 2,400 for algae, 1,000 for plants and 200 for freshwater fish.	Can complex with humic substances and then be more easily absorbed by clay then if in free state.	No data available

TABLE C-2 (Continued)

Environmental Fate and Behavior of the Twelve Indicator Chemicals
Westinghouse Project No. 4112-88-907B

Chemical	Bioaccumulation	Adsorption	Biodegradation
Lead	Not significant in fish, however does bioaccumulate in shellfish. Also, lead uptake in fish is limited to the nonedible portions (e.g., epidermis, scales).	High adsorption in soils with high organic matter and pH >5 - lead then retained in upper 2-5 cm of soil. Precipitation occurs to insoluble salt forms. Depends on iron concentration, salinity, and redox conditions.	One of the most stable of metals due to corrosion resistance
Mercury	Bioaccumulation as high as 10,000 times that of water for salt water fish and 63,000 times for fresh-water fish. Accumulation rapid in fish due to fast uptake and slow elimination.	Rapid volatilization. In aquatic systems, appears to bind to dissolved matter or fine particulates. Can be desorbed and transported and redeposited on bed sediment.	Microorganisms covert elemental mercury to methyl mercury salt which then volatilizes
Nickel	No data available	Controlled by complexation, precipitation/dissolution. Primary ion may be hexahydrate which may coprecipitate or be adsorbed by hydrous oxides of iron, silica and manganese.	No data available
Selenium	In acidic or neutral soil, amount of biologically active selenium declines.	Sorption by bed sediments or suspended solids, and precipitation with hydrous iron oxides are major factors on mobility.	Can be methylated by a variety of organisms. The methylated forms are volatile and may escape to the atmosphere.
Vanadium	Ubiquitous element with high affinity for fats and oils. Food oils have high concentrations. Significant amounts are found in milk, seafoods, cereals and vegetables.	Many of the common ions of vanadium are readily soluble in water.	No data available

TABLE C-2 (Continued)

Environmental Fate and Behavior of the Twelve Indicator Chemicals
Westinghouse Project No. 4112-88-907B

Chemical	Bioaccumulation	Adsorption	Biodegradation
Zinc	Essential element which is strongly bioaccumulated, however it does not appear to be biomagnified.	Sorption onto hydrous iron and manganese oxides, clay and organic materials. Precipitation of sulfide is controlling factor on mobility.	No data available

Sources: Chemical Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Clement Associates, Inc., Sept. 1985.

Water-Related Environmental Fate of 129 Priority Pollutants, Vols. I and II. Versar, Inc., Dec. 1979.

APPENDIX D

ARAR Tables

TABLE D-2

Organic Chemical Specific ARARs
Environmental Media - Air
Westinghouse Project No. 4112-88-921B

Parameter	ACGIH TLV (mg/m^3)	OSHA PELs (mg/m^3)
Acetone	1780 2375[a]	1800 2400[a]
Benzene	30[b]	10 ppm[d]
Benzo(a)anthracene	--	--
Benzo(b)fluoranthene	--	--
Benzo(k)fluoranthene	--	--
Bis(2-ethylhexyl)phthalate	--	--
2-Butanone (MEK)	590 885[a]	590 885[a]
Chlorobenzene	350	350
Chloroform	50[b]	2 ppm
Chrysene	b	0.2[e]
1,2-Dichloroethene	790	790
2,6-Dinitrotoluene	1.5[c]	1.5[c]
Ethylbenzene	435 545[a]	435 545[a]
Fluoranthene	--	--
2-Hexanone	20	20
Methylene Chloride	175[b]	500 ppm[g]
4-Methyl-2-pentanone	--	--
Naphthalene	50 75[a]	50 75[a]
N-Nitrosodiphenylamine	--	--
Phenanthrene	0.2[ef]	0.2[e]
Phenol	19[c]	19[c]
Pyrene	0.2[ef]	0.2[e]
Toluene	375 560[a]	375 560[a]
Xylene	435 655[a]	435 655[a]

[a] Listed as an STEL (Short Term Exposure Limit)
[b] Listed as a Suspected Human Carcinogen
[c] Listed with a Skin Notation
[d] With a 25 ppm Acceptable Ceiling Concentration and a 50 ppm Maximum Peak Concentration for 10 minutes above the Acceptable Ceiling Concentration.
[e] Listed as a Coal Tar Pitch Volatile
[f] Listed as a Confirmed Human Carcinogen
[g] With a 1000 ppm Acceptable Ceiling Concentration with a Maximum - Peak of 2000 ppm, for 5 minutes in any 2 hours, above the acceptable ceiling concentration.

TABLE D-1

Organic Chemical - Specific ARARs
Environmental Media - Water
Westinghouse Project No. 4112-88-921B

Parameter	Safe Drinking Water Act/RCRA Levels: RCRA MCL (mg/l)	SDWA MCL (mg/l)	SDWA MCLG (mg/l)	Primary/ Secondary	CWA Water Quality Criteria: Protection of Human Health: Water and Fish (mg/l)	Protection of Human Health: Fish Only (mg/l)	Protection of Aquatic Life: Freshwater Acute mg/l	Protection of Aquatic Life: Freshwater Chronic	Surface Water (mg/l)	VA Water Quality Standards: Aquatic Life (ug/l)	Ground Water (ug/l)
Acetone	--	--	--	--	--	--	--	--	--	--	10 Total Organic C
Benzene	--	0.005	0	Primary	0.00066	0.040	5.3*	--	--	--	1 Petroleum Hydrocarbons
Benzo(a) anthracene	--	--	--	--	--	--	--	--	--	--	--
Benzo(b) fluoranthene	--	--	--	--	--	--	--	--	--	--	--
Benzo(k). fluoranthene	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethyl hexyl)phthalate	--	--	--	--	--	--	0.94*a	0.0030*a	--	3 Phthalate Esters	--
2-Butanone	--	--	--	--	--	--	--	--	--	--	--
Chlorobenzene	--	0.1[c]	0.1[c]	Primary	--	--	--	--	--	--	--
Chloroform	--	--	--	--	0.00019	0.018	28*	1.2*	--	--	--
Chrysene	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethene	--	0.07 cis[c] 0.1trans[c]	0.07cis[c] 0.1trans[c]	Primary Primary	0.000033	0.0019	11*	--	--	--	--
2,6-Dinitrotoluene	--	--	--	--	--	--	--	--	--	--	--
Ethylbenzene	--	0.7[c] 0.03[c]	0.7[c]	Primary Secondary	1.4	3.3	32	--	--	--	--
Fluoranthene	--	--	--	--	0.042	0.054	3.9*	--	--	--	--
2-Hexanone	--	--	--	--	--	--	--	--	--	--	--
Methylene Chloride	--	--	--	--	--	--	--	--	--	--	--
4-Methyl-2-pentanone	--	--	--	--	--	--	--	--	--	--	--
Napthalene	--	--	--	--	--	--	--	--	--	--	--
N-Nitosodiphenyl amine	--	--	--	--	0.0049	0.016	5.8*b	--	--	--	--
Phenanthrene	--	--	--	--	--	--	--	--	--	--	--
Phenol	--	--	--	--	3.5	--	10	2.5	0.001	1.0	--
Pyrene	--	--	--	--	--	--	--	--	--	--	--
Toluene	--	2[c] 0.04[c]	2[c]	Primary Secondary	14	420	17*	--	--	--	--
Xylene	--	10[c]total 0.02[c]	10[c]total	Primary Secondary	--	--	--	--	--	--	--

When 2 or more values conflict, the lower value generally should be used.

* Lowest Observed Effect Level
a Value is for phthalate esters in general
b Value is for nitrosamines in general
c Proposed MCLs and MCLGs from 54 FR 22062

AR302207

TABLE 3

Inorganic Chemical - Specific ARARs
Environmental Media - Water
Westinghouse Project No. 4112-88-921B

Parameter	Safe Drinking Water Act/RCRA Levels: RCRA MCL (mg/l)	SDWA MCL (mg/l)	SDWA MCLG (mg/l)	PRIMARY/ SECONDARY	CWA Water Quality Criteria: Protection of Human Health: Water and Fish (mg/l)	Protection of Human Health: Fish Only (mg/l)	Protection of Aquatic Life: Freshwater Acute mg/l	Protection of Aquatic Life: Freshwater Chronic mg/l	VA Water Quality Standards: Surface Water (mg/l)	Aquatic Life (ug/l)	Ground Water (mg/l)
Aluminum	--	0.05*	--	Secondary	--	--	0.750	0.087	--	--	--
Antimony	--	--	--	--	0.15[a]	45[a]	9.0[a]	1.6[a]	--	--	--
Arsenic	0.05	0.05	--	Primary	0.0000022[b]	0.000010[b]	0.8[xn] 0.3[c]	0.048[xn] 0.1[c]	0.05	190 (AsIII) Total rec.	0.05
Barium	1.00	5*	5*	Primary	1[c]	--	--	--	1.0	--	1.0
Beryllium	--	--	--	--	0.0000068[d]	0.00012[d]	0.1[dx]	0.0053[dx]	--	--	--
Cadmium	0.01	0.005*	0.005*	Primary	0.010e	--	0.0039[ep]	0.0011[ep]	0.01+	A	0.0004
Calcium	--	--	--	--	--	--	--	--	--	--	120 Hardness
Chromium	0.05	0.1*	0.1*	Primary	170[f] 0.050[g]	3433[f]	1.7[fp] 0.016[g]	0.2[fp] 0.011[g]	0.05 Total	11(CrVI) B	0.05
Cobalt	--	--	--	--	--	--	--	--	--	--	--
Copper	--	1	--	Secondary	--	--	0.018[p]	0.012[p]	1.0+	C	1.0
Iron	--	0.3	--	Secondary	--	--	--	--	0.3 soluble	1000	0.3 Piedmont
Lead	0.05	0.05	--	Primary	0.05[h]	--	0.080[h]	0.0032[hp]	0.05	D	0.05
Magnesium	--	--	--	--	--	--	--	--	--	--	--
Manganese	--	0.05	--	Secondary	--	--	--	--	0.05 soluble	E	0.05Piedmont[y]
Mercury	0.002 Total Hg	0.002*	0.002*	Primary	--[i] 0.00014[j]	--[i] 0.00015[j]	0.0024[i] 0.0024[j]	0.000012[i] 0.000012[j]	0.002+	E	0.00005
Nickel	--	--	--	--	0.0134	0.1	1.4[p]	0.16[p]	--	F	--
Potassium	--	--	--	--	--	--	--	--	--	--	--
Selenium	0.01	0.05*	0.05*	Primary	0.010[k]	0.010[k]	0.26[k]	0.035[k]	0.01+	35 total inorganic	0.01
Silver	0.05*	0.09	--	Secondary	0.050[l]	0.050[l]	0.0041[lp]	0.00012[l]	0.05+	G	None
Sodium	--	--	--	--	--	--	--	--	--	--	25 Piedmont[y]
Thallium	--	--	--	--	0.013[m]	0.048[m]	1.4[xm]	0.040[xm]	--	--	--
Vanadium	--	--	--	--	--	--	--	--	--	--	--
Zinc	--	5	--	Secondary	--	--	0.13[q]	0.11[q]	5.0+	47 total recoverable	0.05
Cyanide	--	--	--	--	0.2	--	0.022	0.0052	--	5:2	0.005
Sulfate	--	250	--	Secondary	--	--	--	--	250	--	25 Piedmont[y]

* Proposed MCLs and MCLGs from 54 FR 22062

[a] Antimony and compounds
[b] Arsenic and compounds
[c] Barium and compounds
[d] Beryllium and compounds
[e] Cadmium and compounds
[f] Chromium(III) and compounds
[g] Chromium(VI) and compounds
[h] Lead and compounds(Inorganic)
[i] Mercury and compounds(Alkyl)
[j] Mercury and compounds(Inorganic)
[k] Selenium and compounds
[l] Silver and compounds
[m] Thallium and compounds
[n] Arsenic(V) and compounds
[o] Arsenic(III) and compounds
[p] Hardness dependent criteria (100mg/l used)
[q] Zinc and compounds
[x] Lowest Observed Effect Level
When two or more values conflict, the lower value generally should be used.
[y] For the Piedmont and Blue Ridge Region of Virginia

\+ The numeric standards for the constituents above are designed to protect public water supplies for human consumption. The limits marked with an asterisk may not protect aquatic life.

A) For total recoverable Cadmium $e^{0.7852[\ln(\text{hardness})]-3.490}$
B) For total recoverable trivalent Chromium $e^{0.819[\ln(\text{hardness})]+1.561}$
C) For total recoverable Copper $e^{0.8545[\ln(\text{hardness})]-1.465}$
D) For total recoverable Lead $e^{1.266[\ln(\text{hardness})]-4.661}$
E) No freshwater values
F) For total recoverable Nickel $e^{0.76[\ln(\text{hardness})]+1.06}$
G) For total recoverable Silver $e^{1.72[\ln(\text{hardness})]-6.52} \times 0.01$

AR302208

TABLE D-4

Inorganic Chemical Specific ARARs
Environmental Media - Air
Westinghouse Project No. 4112-88-921B

Parameter	ACGIH TLV (mg/m³)	OSHA PELs (mg/m³)
Aluminum	2[a]	15[a1]
Antimony	0.5[b]	0.5[b]
Arsenic	0.2[c]	0.5[b1]
Barium	0.5[d]	0.5[d]
Beryllium	0.002[e]	0.002[c1]
Cadmium	0.05[f]	0.2[d1]
Calcium	10[g]	15[e1]
Chromium	0.05[h]	0.5[f1]
Cobalt	0.05[i]	0.05[g1]
Copper	1[j]	1[j]
Iron	1[k]	1[k]
Lead	0.15[l]	0.050[h1]
Magnesium	10[m]	10[i1]
Manganese	5[n]	5[j1]
Mercury	0.01[o]	0.01[k1]
Nickel	0.1[p]	0.1[p]
Potassium	--	--
Selenium	0.2[q]	0.2[q]
Silver	0.01[r]	0.01[r]
Sodium	s	s
Thallium	0.1[t]	0.1[t]
Vanadium	0.05[u]	0.05[i1]
Zinc	10[v]	10[m1]
Cyanide	5[w]	5[w]
Sulfate	--	--

a Soluble salts of Aluminum.
b Antimony and compounds as Sb, including Antimony trioxide.
c Arsenic and soluble compounds as Arsenic.
d Soluble compounds as Barium. Barium sulfate TLV is 10 mg/m³ for total dust containing no asbestos and <1% free silica.
e Beryllium and compounds. It is listed as a Suspected Human Carcinogen.
f Dusts and salts as Cadmium. There is a Notice of Intended Change for 1988-89 to 0.01 and listing as a Suspected Human Carcinogen.
g For calcium silicate, calcium carbonate and calcium sulfate as total dust with no asbestos and <1% free silica. For calcium oxide - 2 mg/m³; for calcium hydroxide - 5 mg/m³; for calcium cyanamide - 0.5 mg/m³.
h For chromium (VI) water soluble compounds as Cr and also for certain water insoluble Cr(VI) compounds that are listed as Confirmed Human Carcinogens. For chromium metal, chromium (II) compounds and chromium (III) compounds - 0.5 mg/m³.
i For cobalt metal dust as Co. For cobalt carbonyl and cobalt hydrocarbonyl, as Co - 0.1 mg/m³.
j For dusts and mists as Cu.

TABLE D-4
(Continued)

Inorganic Chemical Specific ARARs
Environmental Media - Air
Westinghouse Project No. 4112-88-921B

k For soluble iron salts as Fe.
l For inorganic lead dusts and fumes.
m For magnesium oxide fumes only.
n For manganese dusts and Mn compounds as Mn.
o For Mercury alkyl compounds. For all forms except alkyl - 0.05 mg/m^3 and for aryl and inorganic compounds - 0.1 mg/m^3. All are listed with a skin notation.
p For soluble compounds as Ni. For nickel metal - 1 mg/m^3.
q For selenium compounds as Se.
r For soluble silver compounds as Ag.
s No values for relevant inorganic sodium compounds, other than for sodium hydroxide - 2 mg/m^3.
t For soluble compounds as Tl. Listed with a skin notation.
u Only for Vanadium pentoxide respirable dusts and fumes as V_2O_5.
v For zinc oxide dusts containing no asbestos and ,1% free silica.
For zinc chromates - 0.01 mg/m^3 and listed as a Confirmed Human Carcinogen.
w For cyanides as Cn. Listed with a skin notation.
al For total dust as Al. For respirable fraction - 5 mg/m^3.
bl For organic compounds as As. Inorganic arsenic compounds are listed as Substances Specifically Regulated by OSHA in 29 CFR 1910.1018 with a PEL of 0.010 mg As/m^3, 8-hr TWA.
cl This is an 8-hr TWA for beryllium and beryllium compounds. The acceptable ceiling concentration is 0.005 mg/m^3, with an acceptable maximum peak of 0.025 mg/m^3 for a maximum duration of 30 minutes.
dl This is an 8-hr TWA for cadmium dust. The acceptable ceiling concentration is 0.6 mg/m^3.
el For total dust of calcium silicate, carbonate and sulfate, with a respirable fraction of 5 mg/m^3. For calcium oxide - 5 mg/m^3.
fl For Cr(II) and Cr(III) compounds as Cr.
gl For cobalt metal, dust and fume as Co.
hl For inorganic lead, this is an 8-hr TWA.
il For magnesium oxide fume-total dust TWA, with a respirable fraction TWA of 5 mg/m^3.
jl Ceiling value for manganese compounds as Mn.
kl For alkyl (organo) mercury compounds (as Hg) as a 8-hr TWA. An STEL concentration of 0.03 mg/m^3 is also given. Listed with a skin notation.
ll For respirable dusts as V_2O_5, with a ceiling limit of 0.5 mg/m^3.
ml For total dust, with a respirable fraction TWA of 5 mg/m^3.